American Association of Equine Practitioners

NASHVILLE

TENNESSEE

PROCEEDINGS

59TH ANNUAL CONVENTION

December 7-11, 2013

2013 ANNUAL CONVENTION & TRADE SHOW

Proceedings of the 59th Annual Convention of the American Association of Equine Practitioners

Nashville, Tennessee
December 7–11, 2013

Program Chair: Jeff A. Blea, DVM

ACKNOWLEDGMENTS
Katherine S. Garrett, DVM, DACVS, Educational Programs Committee Chair
Carey M. Ross, Scientific Publications Coordinator

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Policy Statement

The primary purpose of publishing the Proceedings is to provide documentation of the scientific presentations in abstract form, available at the AAEP annual convention. Its further purpose is to offer easily accessible information that will assist the AAEP membership, and others in the equine industry, in the daily responsibility of providing the best possible care for the horse.

Mission Statement

To improve the health and welfare of the horse, to further the professional development of its members, and to provide resources and leadership for the benefit of the equine industry.

Future AAEP CE Dates

2014	Resort Symposium	Rio Grande, Puerto Rico	February 6–8
2014	Focus on the Sport Horse	Louisville, Kentucky	July 20–22
2014	Focus on the First Year of Life	Phoenix, Arizona	September 11–13
2014	60th Annual Convention	Salt Lake City, Utah	December 6–10
2015	61st Annual Convention	Las Vegas, Nevada	December 5–9
2016	62nd Annual Convention	Orlando, Florida	December 3–7
2017	63rd Annual Convention	San Antonio, Texas	November 17–21
2018	64th Annual Convention	San Diego, California	December 1–5

IVIS
WWW.IVIS.ORG

The AAEP and IVIS (International Veterinary Information Service) have joined their resources to disseminate more widely the scientific content of the AAEP Conventions. The papers in this proceedings book are searchable on the IVIS website at http://www.ivis.org.

Conference Recordings

Video and audio recordings for most talks presented at the 2013 AAEP Annual Convention will be available a couple of weeks after the meeting and will be complimentary to attendees through January 31, 2014. After that, a nominal fee will apply for each recorded session. The content will be hosted at www.prolibraries.com/AAEP.

Electronic Proceedings

All papers published in the 2013 AAEP Annual Convention Proceedings book are available on AAEP's Convention App.

From Your President

American Association of Equine Practitioners
4075 Iron Works Parkway • Lexington, KY 40511
859.233.0147
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www.aaep.org



Dear AAEP members and guests,

Welcome to Nashville! Music City is rolling out the red carpet this week to welcome thousands of equine practitioners, academics, and industry representatives to the Gaylord Opryland Convention Center. The 59th Annual AAEP convention offers something for everyone no matter where you are from or what kind of horses you work with. We're glad you're here!

This meeting came together thanks to the hard work of dozens of volunteers and the staff of the AAEP. A big thank you is due to Dr. Jeff Blea, AAEP 2013 President-elect and program chair, for the two years he has spent tending to the myriad details of the meeting. Dr. Blea has put together nine sessions covering veterinary ethics, lessons from past AAEP leaders, practical "how to" talks, and "hot" topics of equine practice, and has selected a great horseman, Buck Brannaman, as our keynote speaker. I would also like to thank Dr. Racquel Lindroth and Dr. Mike Pownall for their visionary planning of the professional development sessions: an excellent blend of talks and workshops covering the business of equine practice has been assembled.

Overall guidance for AAEP's member education is provided by the wise leadership of Dr. Katie Garrett and Dr. Eric Mueller, chair and co-chair of the Educational Programs Committee, and by the AAEP CE Steering Committee, led by Dr. Ed Kanara. The EPC received over 200 submissions for the Nashville program; eight teams of EPC reviewers scored these papers and made the final cuts for the 78 available slots. Publication details and the program schedule were handled by AAEP Scientific Publications Coordinator, Carey Ross, with her usual finesse and polish.

I know that all of you will enjoy the Opening Session, the Milne lecture, the table topics and the many social events and workgroup meetings that occupy every minute of this crowded week. But don't forget to reserve a few hours to browse the huge trade show that runs Sunday through Tuesday. The show is a great opportunity to see "what's new" in products and services and to meet our Educational Partners, key sponsors of AAEP activity who provide leadership for the entire equine industry; be sure to stop in to their booths to say THANK YOU. I am grateful to Debbie Miles, AAEP meeting coordinator, for making sure the trade show remains the top "shopping" opportunity for our profession.

My welcome message would not be complete without recognizing those that are the future of our profession, our veterinary student attendees. Thank you all for sacrificing precious time at school to discover what the AAEP can offer for your future. Expanded opportunities have been planned for you this year by a task force led by Dr. Emma Read. The special student program will include dry labs and professional development presentations as well as a designated lounge for student networking.

Lastly, I would like to thank the entire membership and AAEP staff for the opportunity I have had to serve as your President this year. I have traveled from Buffalo to Budapest as your representative and have found it an enormous honor and privilege to represent you.

Enjoy all that the Convention and Music City have to offer!

Ann E. Dwyer

Ann E. Dwyer, DVM

Raising the Standard in Horse Health

From Your President-Elect and Program Chair

American Association of Equine Practitioners
4075 Iron Works Parkway • Lexington, KY 40511
859.233.0147
fax: 859.233.1968
www.aaep.org



Dear Fellow AAEP Members and Colleagues:

Welcome to the 2013 AAEP Annual Convention.
As the Program Chair for the 59th annual AAEP Convention in Nashville, it is my honor and privilege to present the 2013 Scientific Program. I want to acknowledge that this program could not have been organized and completed without the dedication and tireless efforts of numerous staff and member volunteers. I would like to personally thank the 46 volunteer members of the Educational Programs Committee, EPC Chair Dr. Katie Garrett, EPC Vice-Chair Dr. Eric Mueller, and Mrs. Carey Ross, for their expertise and commitment in providing an exemplary educational program this year. Additionally, I would like to sincerely thank all those who are participating in this year's meeting.

The foundation of the educational program is based upon our mission statement: "To improve the health and welfare of the horse, to further the professional development of its members, and to provide resources and leadership to the benefit of the equine industry." All invited In Depth and "How To" papers, as well as submitted abstracts, underwent a rigorous review process before inclusion in the program. This year, we received 218 abstracts for 78 available slots. This year's program is quite diverse with an emphasis on practical, take home information for the practitioner.

Some highlights of the meeting are:

◆ ***Keynote Speaker*** – Buck Brannaman, who is a legendary horseman, will remind us of why we are equine practitioners. It is the horse!
◆ ***Kester News Hour*** – Dr. Pat McCue, Dr. Lisa Fortier, and Dr. Carol Clark will provide an informative educational session in a slightly different format this year. Don't miss this.
◆ ***Milne State-of-the-Art Lecture*** – Dr. Sue Dyson will present "Equine Lameness: Clinical Judgment Meets Advanced Diagnostic Imaging" on Monday morning.
◆ ***In Depth Sessions*** – Racing-Related Lameness, Sport Horse Lameness, Geriatric Medicine/Metabolics, Reproductive Endocrinology.
◆ "***How To" Sessions*** – Ophthalmology, Radiograph Technique and Interpretation of Young Performance Horses, Field Anesthesia/Pain Management.
◆ ***Past Presidents Session*** – Several Past Presidents will share some practice tips and techniques they have learned over the years. Additionally, they will discuss several topics that remain controversial in equine practice from a historic perspective.
◆ An ***Ethics Session*** with a dynamic and thought provoking group of presenters.
◆ ***Business Program*** – Jane Shaw will have a communication skills workshop to build client relationships and improve outcome. Kathryn Jeffers will provide a program to help you work more effectively with people. A social media dry-lab is also scheduled. An outstanding program and well worth attending.
◆ ***Table Topics*** will be offered as in previous years with the return of the most popular ones, and some newly added ones.
◆ ***Foundation Celebration*** – This year the "Celebration" will occur at the Wildhorse Saloon in Nashville. Clay Walker, in addition to a very popular house band, will be providing entertainment. This promises to be a spectacular event, with proceeds to benefit the AAEP Foundation.

This meeting provides some outstanding C.E., as well as numerous opportunities for networking, socializing, and catching up with friends and colleagues. I personally want to thank you for attending this meeting and choosing the AAEP as your source for continuing education. Have a great meeting and enjoy your time in Nashville!

Best,

Jeff A. Blea, DVM

Jeff A. Blea, DVM

Raising the Standard in Horse Health

2013 AAEP Board of Directors

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2013 AAEP Awards

Distinguished Life Member – Benjamin Franklin, Jr., DVM
The AAEP Distinguished Life Member designation is awarded in recognition and appreciation of dedication and meritorious service to the veterinary profession and the advancement of equine medicine.

Distinguished Educator Award (Academic) – Frank A. Nickels, DVM
Awarded to an individual educator who by his or her actions and commitment has demonstrated a significant impact on the development and training of equine practitioners.

Distinguished Educator Award (Mentor) – Barrie D. Grant, DVM, MS, DACVS
Awarded to an individual who by his or her actions and commitment has demonstrated a significant impact on the development and training of equine practitioners through mentoring.

Distinguished Service Award – Harry W. Werner, VMD
Awarded to an individual who has provided exemplary service to the AAEP or a similar organization to the benefit of the horse, horse industry, or the profession of equine veterinary medicine.

General Instructions for Authors
60th AAEP Convention
Salt Lake City, Utah
December 6–10, 2014

To submit a paper, go to http://aaep2014.abstractcentral.com

ALL papers must be submitted online by March 17, 2014, 3:00 p.m. ET.

The AAEP Proceedings is protected by copyright and information submitted and accepted becomes the property of AAEP. However, requests for copies or reprints will be honored by AAEP only with the cooperative permission of the presenting author, who by his or her presentation, represents all authors. AAEP reserves the right not to accept any submission without further recourse.

Presentations for the AAEP Convention will be selected directly from the review-ready submissions to the AAEP. Submissions may include case series with follow-up data, or the results of experimental or observational studies as scientific papers, as well as "How to" and review papers. Selection will be made by the Educational Programs Committee. The quality of the submission will determine the selection. Missing data or proposed, but not completed procedures, will exclude the submission from consideration. AAEP invites information dealing with any subject germane to equine practice, but special consideration will be given to submissions by practitioners and material with practical content or new information. At least one author of a report describing diagnosis, treatment, or the interpretation of medical information should be a veterinarian.

All submissions should strictly adhere to the Instructions for Authors. Submissions will be ranked using the AAEP Scoring Criteria and the highest-ranking papers will be selected for the available time.

Authors are expected to acknowledge all sources of funding or support for the work described and to disclose to the Educational Programs Committee any financial interest (including ownership, employment, consultancy arrangements, or service as an officer or board member) they have with companies that manufacture or sell products that figure prominently in the paper or with companies that manufacture or sell competing products. Such an interest will not necessarily influence the decision to accept or reject a submission for the program, but must be included in the Acknowledgments section for the convention proceedings.

Guidelines:

Failure to adhere to the following format will result in nonacceptance. It is the author's responsibility to convince the Educational Programs Committee of the value of the submission, as well as to portray to the reader the contents of the presentation. **Specific instructions for Scientific papers, "How to" papers, Review papers, ≤250 word abstracts, and Business of Practice papers, and can be found in their respective sections.**

Headings should include (but are not limited to) the following:

1. Take Home Message
2. Introduction
3. Materials and Methods
4. Results
5. Discussion
6. References

Title:

The title should be 15 words or fewer, at the top and on the first page.

Example:
Upper Respiratory Dysfunction in Horses During High Speed Exercise

Take Home Message:

This should be a short, concise summarization of the main conclusion and should be no longer than two or three sentences (approximately 50 words). "How to" papers do not require a take-home message.

Example:
Local anesthetic injected into the coffin joint is not selective for only this joint. Such injections will desensitize much of the navicular bone and its suspensory ligaments.

Introduction:

The rationale for the submission should be given briefly and significant published work acknowledged here. The clinical significance should also be included, as well as a clear statement of the objective or purpose of the submission. The statement of objectives is usually found in the last sentence of the Introduction.

Materials and Methods:

This section should describe experimental methodology in the case of a didactic study or, in the case of a clinical study, should include a description of the population from which the animals were selected and how they were selected for inclusion in the report.

Data obtained and how they were obtained must be described. A description of the statistical methods used to summarize data, test hypotheses, and characterize the significance of results should also be included. For weights and measures, metric units should be used. Dosages should be expressed entirely in metric units and with specific time intervals.

Example:
22 mg/kg, q 12 h, IV (not 10mg/lb, BID, IV)

Results:

Actual results with numbers and data must be presented. When possible, quantify findings (mean, median, proportion) and present them with appropriate estimates of measurement error or uncertainty (such as standard deviation (SD), standard error (SE) or confidence interval) in addition to the results of hypothesis testing. If the data can be well represented with a graph or figure, these *are encouraged* if subsequent publication is not anticipated. If numbers and data

are not presented due to concerns regarding publication in a refereed journal, indications of relative differences between groups such as odds ratios, % change, and significant differences must be included in the submission to be considered acceptable. In these instances, the authors should submit the data in the form of means, standard deviations, or other descriptions of comparisons among groups in an appendix, which will not be published and only used for review purposes.

Discussion:

Important findings documented in the results of the study should be stated. Results should be related to other work which has been done and how the results differ or agree with previously published work and why any differences may have occurred should be discussed. The practical take home message for the equine practitioner should be clearly defined and stated in the summarizing final statement. This statement may be longer, but should be similar in content to the take home message at the beginning of the paper.

References:

All submissions must include references. References to published works should be limited to what is relevant and necessary. Number references in the text with superscript numbers consecutively in the order in which they are first cited. Under references, list all authors when there are three or fewer; list only the first three and add "et al." when there are four or more. The author is responsible for the formatting and accuracy of all reference citations. Since readers frequently depend upon the reference citations to guide them in further reading, it is imperative that the citations are correct so that libraries can locate the papers a reader may wish to obtain.

Examples:

Journal article:

Auer JA, Martens RJ, Williams EH. Periosteal deformities in foals. *Am J Vet Res* 1982;181:459–466.

Murphy CJ, Lavoie JP, Groff J, et al. Bilateral eyelid swelling attributable to lymphosarcoma in a horse. *J Am Vet Med Assoc* 1989;194:939–942.

Some common journal abbreviations include: *Acta Vet Scand, Am J Vet Res, Can J Vet Res, Can Vet J, Cornell Vet, Compend Contin Educ Pract, Equine Vet J, Equine Vet J Suppl, J Am Vet Med Assoc, J Vet Diagn Invest, J Vet Intern Med, Prev Vet Med, Vet Clin North Am Equine Pract, Vet Radiol, Vet Rec, Vet Surg.* Other journal names should be abbreviated in accordance with the National Library of Medicine and *Index Medicus*.

Book:

Turner AS, McIlwraith CW. *Techniques in large animal surgery*. Philadelphia: Lea and Febiger, 1982;186–191.

Banks P, Bartley W, Birt LM. *The biochemistry of the tissues 2nd ed.* London: John Wiley & Sons, 1968;24.

Devlin TM, ed. *Textbook of biochemistry with clinical correlations*. New York: John Wiley & Sons, 1982;14–36.

Chapter in a book:

Axelrod B. Glycolysis. In: Greenberg DM, ed. *Metabolic pathways, vol 1. 3rd ed.* New York: Academic Press, 1967; 112–145.

Kainer RA. Functional anatomy of equine locomotor organs. In: Stashak TS, ed. *Adams' lameness in horses 4th ed.* Philadelphia: Lea and Febiger, 1987;12–18.

Proceedings:

Divers TJ. Acute renal failure in horses and cattle, in *Proceedings*. 3rd Am Coll Vet Int Med Forum 1985;93–95.

Lamb CR, Koblik PD, O'Callaghan MW, et al. Comparison of bone scintigraphy and radiography as aids in the evaluation of equine lameness: Retrospective analysis of 275 cases, in *Proceedings*. Am Assoc Equine Pract 1989;35: 359–368.

Footnotes:

References to dissertations, theses, abstracts, personal communications and papers submitted but not yet accepted for publication should be footnoted:

Jones CD. The selective advantage of the ABO blood groups [thesis]. Ithaca, NY: Cornell University; 1990.

Bramlage LR. Lexington, KY. (personal communication) 1996.

Smith AB. Unpublished data. January 1990.

Evans LH. Entrapment of the epiglottis. Am Assoc Equine Pract. In Press 1981.

Products and equipment should be identified by chemical or generic names or descriptions.
All products should be footnoted, along with the manufacturer's full address. A trade name may be included in a lettered footnote along with the name and location (full mailing address including zip code) of the manufacturer when the product or equipment was essential to the outcome of the experiment or treatment.

Example:
All horses were sedated with a combination of detomidine HCL[a] (10–20 mg/kg IV) and butorphanol tartrate[b] (0.01–0.02 mg/kg IV).
a Dormosedan® Orion Corporation, Espoo, Finland.
b Torbugesic®, Fort Dodge Animal Health, Fort Dodge, IA 50501.

Figures:

- The resolution should be at least 300 dpi.
- Figures should be cited in the text in parentheses (Fig. 1) consecutively in the order of which they are first mentioned.
- The figure itself should also be numbered to correspond to the citation in the text.
- Figures must include captions, 40 words or fewer.

Tables:

Tables should be self explanatory and should supplement the text. Provide a concise, descriptive title for each table.

Acknowledgments:

Acknowledgments should include financial and material support for research (e.g. Grayson Jockey Club Research Foundation, AQHA Foundation) and technical support for work performed. Authors are expected to disclose the nature of any financial interests (including ownership, employment, consultancy arrangements, or service as an officer or board member) they have with companies that manufacture or sell products that figure prominently in the submission or with companies that manufacture or sell competing products.

Permissions:

If you wish to use previously published material, including text, photographs, or drawings, you must acknowledge the original source and submit written permission from the copyright holders (author and publisher) to reproduce the material. Provide this permission when you submit your original manuscript.

Conflicts of Interest:

Summary

1. Any and all authors listed on the paper must disclose any actual or potential conflicts of interest
2. Any and all authors listed on the paper must disclose if no conflict exists
3. The nature of the conflict (actual or potential) needs to be described

At the point of submission, the American Association of Equine Practitioner (AAEP)'s policy requires that authors must disclose and describe the nature of any actual or potential financial and/or personal relationships they have with companies that manufacture or sell products that figure prominently in the submission or with companies that manufacture or sell competing products. (This includes ownership, employment, consultancy arrangements, or service as an officer or board member.) When considering whether a conflicting interest or connection should be declared, the author is asked to answer the following: Is there any arrangement that would embarrass you or any of your co-authors if it was to emerge after publication and you had not declared it?

As an integral part of the online submission process, Submitting authors are required to confirm whether they or their co-authors have any actual or potential conflicts of interest to declare, and to provide details of these. It is the Submitting author's responsibility to ensure that all authors adhere to this policy.

If the paper is published, the conflict of interest information will be communicated in a statement in the proceedings.

If the authors have no conflict of interest to declare, they must also state this at submission.

Conflict of Interest in Industry Sponsored Research:

Authors whose papers are submitted for publication must declare all relevant sources of funding in support of the preparation of a paper. The AAEP requires full disclosure of financial support as to whether it is from the industry, the pharmaceutical or any other industry, government agencies, or any other source. This information should be included in the Conflict of Interest section of the published paper.

Authors are required to specify sources of funding for the study and to indicate whether or not the text was reviewed by the sponsor prior to submission, i.e., whether the study was written with full investigator access to all relevant data and whether the sponsor exerted editorial influence over the written text. In addition to disclosure of direct financial support to the authors or their laboratory and prior sponsor-review of the paper, submitting authors are asked to disclose all relevant consultancies prior to submission. This information should be included in a Conflict of Interest section at the end of paper.

All authors are requested to disclose any actual or potential conflict of interest including any financial, personal or other relationships with other people or organizations within three years of beginning the submitted work that could inappropriately influence, or be perceived to influence, their work.

Example of COI Statement

Conflict of Interest: Dr. John Doe has no conflict of interest. Dr. Jane Doe has served as a paid technology analyst for the venture capitalists that initiated the formation of Company ABC and served as a member of the Board of Directors of Company ABC from its inception until 2008. Company ABC is currently commercializing the use of Product XYZ. Dr. Jane Does has also served as a paid consultant and continues to serve on the Company ABC Advisory Board.

Ethical Considerations:

1. Authors are expected to disclose the nature of any financial interests they have with companies that manufacture or sell products that figure prominently in the submission or with companies that manufacture or sell competing products. (This includes ownership, employment, consultancy arrangements, or service as an officer or board member.)
2. If your paper or presentation references the use of a compounded pharmaceutical, please be certain that you are familiar with the FDA guidelines on the use of compounded pharmaceuticals and that the product you reference is in compliance.
3. All submissions should cite levels of evidence-based medicine.

You should plan to include any ethical considerations as part of your oral presentation if your paper is accepted.

IACUC Approval:

AAEP is dedicated to the humane use of animals in scientific research in accordance with the Institutional Animal Care and Use Committee (IACUC). AAEP recognizes the difficulty practitioners may have when attempting to obtain IACUC approval therefore the Educational Programs Committee has compiled a list of liaisons that practitioners can use as a resource. This list can be found on the AAEP website (www.aaep.org).

Standard of Care:

The AAEP is sensitized to having people use the term "Standard of Care" from the podium. If you plan to do this please include this in your abstract or written submitted material so the EPC can confirm its agreement with your statement.

1. A diagnostic and treatment process that a clinician should follow for a certain type of patient, illness, or clinical circumstance. Adjuvant chemotherapy for lung cancer is "a new standard of care, but not necessarily the only standard of care." (New England Journal of Medicine, 2004).
2. In legal terms, the level at which the average, prudent provider in a given community would practice. It is how similarly qualified practitioners would have managed the patient's care under the same or similar circumstances. The medical malpractice plaintiff must establish the ap-

propriate standard of care and demonstrate that the standard of care has been breached.

Deadline:

ALL papers must be submitted online by March 17, 2014, 3:00 p.m. E.T.; under no circumstances will submissions received after the deadline be considered or reviewed. ALL deadlines must be adhered to in order to have the published Proceedings available at the meeting.

Review Process:

To respect the integrity of the Annual Convention program and ensure the fairness of the review process, AAEP has adopted blind reviewing in which the identity of the authors and reviewers are not known to each other. Papers will be reviewed, scored, and selected by the Educational Programs Committee.

Scoring Criteria

One goal of the Educational Programs Committee (EPC) in choosing submissions for the AAEP annual meeting is to combine the best available clinical research with clinical experience and expertise to meet the needs of our patients. The AAEP Scoring Criteria can be found at www.aaep.org or request a copy from cross@aaep.org

Pre-Press Approval:

Authors will have final approval at the page proof stage. Changes/updates in numbers, dosages or inappropriate grammar may be made within one week of receiving page proofs. Final grammatical changes will be the decision of the editors. Substantial changes or removal of any data will result in forfeiture of complimentary registration and travel, and exclusion from the program.

Reimbursement:

Presenting authors will receive one complimentary registration and a reimbursement of $550 to help support travel.

Mentors for Authors:

Paper submissions by private practitioners and first time authors are highly encouraged. The AAEP website (www.aaep.org) features a list of members in various areas of expertise that have agreed to volunteer their time to mentor an author who needs guidance.

Scientific Papers: Guidelines for Authors 60th AAEP Convention Salt Lake City, Utah December 6–10 2014

To submit a paper, go to
http://aaep2014.abstractcentral.com

ALL papers must be submitted online by March 17, 2014, 3:00 p.m. ET.

Authors who do ***not*** intend to publish in a refereed journal are welcome to submit a scientific paper.

Scientific Paper selection will be made by the Educational Programs Committee. The quality of the Scientific Paper will determine the selection. Missing data or proposed, but not completed procedures, will exclude the Scientific Paper or other paper from consideration. AAEP invites information dealing with any subject germane to equine practice, but special consideration will be given to presentations by practitioners and material with practical content or new information. At least one author of a report describing diagnosis, treatment, or the interpretation of medical information should be a veterinarian.

Scientific papers should be formatted as described in the General Instructions for Authors. Scientific papers should be no fewer than 600 words. No upper word limit.

The "How to" Paper: Guidelines for Authors 60th AAEP Convention Salt Lake City, Utah December 6–10, 2014

To submit a paper, go to
http://aaep2014.abstractcentral.com

ALL papers must be submitted online by March 17, 2014, 3:00 p.m. ET.

"How to" papers are presented to describe and explain a technique or procedure used in equine veterinary medicine or the equine industry. The technique should be relatively new or not widely understood or used in practice. The goal of the "How to" paper is to give the equine veterinarians the information they need to critically evaluate the pros and cons of the technique and implement it in their practice if they choose.

"How to" papers can be patterned after a modification of the style for a Scientific Paper supporting a scientific presentation. Refer to General Instructions for Authors as you prepare your submission.

The title should begin with "How to . . ." and clearly identify the technique or procedure that will be presented. A "Take Home Message" is not required for "How to" papers. The Introduction should include why you use the technique. If there is a problem with the traditional methods or the currently used method can be improved, this should be explained.

The Materials and Methods section should explain exactly how the technique is performed so that another veterinarian familiar with the subject area could follow your example. You may use a step-by-step method for the paper and the presentation. All medications, supplies, and equipment used should be described using generic names. Trade names and addresses of commercial products critical to the technique can be included in footnotes.

The Results section should include a summary of what happens when you use this technique. The number of horses treated in this manner and an assessment of the outcome should be included. You may use personal assertions or data to assert its value, but you must explain how you determined that the technique works.

In the Discussion section you can give your personal views as to why you think the technique works. Discuss the pros and cons of your approach. Explain how the technique has helped you in your practice and why this should be important to your colleagues. The end of the discussion should contain a summary of the technique and its advantages in the take home message. Case selection, case study number, and case follow-up should all be included.

Review Paper: Guidelines for Authors 60th AAEP Convention Salt Lake City, Utah December 6–10, 2014

To submit a paper, go to http://aaep2014.abstractcentral.com

ALL papers must be submitted online by March 17, 2014, 3:00 p.m. ET.

Review papers are presented for the purpose of updating the membership on a new subject or for gathering information that may be conflicting. The aim of the paper is to help the membership put the information in perspective, and to make judgments on conflicting information. A review paper will not principally present original data. The goal is to clarify existing knowledge on a subject and help the membership better use the information in their day to day practice.

Review papers should generally be formatted as described in the "Instructions for Authors of Manuscripts" except where otherwise noted here. The paper should be titled "Review of Some Subject." The content of review articles should be organized with headings and subheadings that provide a logical flow to the material presented. A "Take Home Message" is required for a Review Paper. The Introduction should define the subject matter and put it in context, explaining why the review is necessary. The purpose of the review paper should be clearly stated in the Introduction.

Agreement and disagreement within the subject matter should be identified along with the strengths and limitations of the information sources. Reference should be made to the authors who generally support the opinions stated. The author's perspective, including his/her own interpretation of the information if it is different from previously published opinions, should be included. The end of the discussion should contain a summary and the conclusion that the author has drawn for the audience, based upon the reviewed data. As with a Scientific Paper, a "Take Home Message" should be provided by the author that summarizes the practical application of the information for the practitioner.

An appropriately complete reference list should be included. The format for references is the same as that described in "General Instructions for Authors."

Illustrations should be provided in the format described in "General Instructions for Authors". If previously published material is submitted, including text, photographs or drawings, the author must acknowledge the original source and submit written permission from the copyright holders (author and publisher) to reproduce the material. This permission must accompany the original manuscript at the time of submission.

Abstracts ≤250 Words: Guidelines for Authors

For those who intend to publish in a refereed journal

60th AAEP Convention Salt Lake City, Utah December 6–10, 2014

To submit a paper, go to http://aaep2014.abstractcentral.com

ALL papers must be submitted online by March 17, 2014, 3:00 p.m. ET.

In order to encourage submission of the newest scientific information for inclusion in the AAEP Annual Convention program and simultaneously not jeopardize future publication of this material in a refereed journal, the following criteria have been developed for these submissions of Scientific Papers that will be published in the AAEP Proceedings. In such instances, the published abstract can be ≤250 words. However, these "abbreviated abstracts" should follow a structured format with the same subheadings (Take Home Message, Introduction, Materials & Methods, Results and Discussion) as the full-length scientific paper. Please be aware that the Take Home Message is included in the total word count. The abbreviated abstract does not need references but appropriate acknowledgments should be included. Note that this abbreviated abstract format does not apply to Review, How To, or In-Depth Papers. **A full paper** conforming to the General Instructions for Authors must also be submitted to allow the reviewers to assess the experimental design, materials and methods, statistical analyses, results (with graphs, tables, charts, etc.) and a discussion of the results as it pertains to interpretation and conclusions (**see specific guidelines below for full papers**). The submitting author must include a statement that only the short abstract can be published in the AAEP Convention Proceedings. It remains the authors' responsibility to preserve their right to publish in a refereed journal by contacting the respective journal to discuss their prior-publication criteria, so that an accepted abbreviated abstract will not jeopardize publication in the refereed journal. These submitted abbreviated abstracts should be identified with the words "RESEARCH ABSTRACT" at the end of the title.

Guidelines for Full Papers

- No more than 4 single-spaced pages. This does not include tables, figures, and references
- 12 point font
- 1″ margins
- When submitting online, please put both papers in one document; the 250 word abstract should be first, followed by the full-length scientific paper.

Business of Practice Papers: Guidelines for Authors 60th AAEP Convention Salt Lake City, Utah December 6–10, 2014

To submit a paper, go to http://aaep2014.abstractcentral.com

ALL papers must be submitted online by March 17, 2014, 3:00 p.m. ET.

Business of Practice papers must follow the same guidelines as detailed in the "General Instructions for Authors." The theme for 2014 will be ***Collaboration by Choice*** with the intent of bringing forward the idea of building collaborative partnerships/teams. Areas to consider for presentation include, though are not limited to:

- Creating partnerships with our clinents—e.g. fostering client loyalty/focus on client relations; e.g. preventative health care plans
- Building organizational health within the business: e.g. creating a practice culture; e.g. hospital management considerations–conflict resolution
- Collaborating with colleagues: e.g. examples of co-operative efforts regarding job-sharing; shared facilities and equipment; e.g. business mergers
- Collaborating with other professional: e.g. partnering with our pharmaceutical companies/representatives; e.g. establishing good working relations with affiliates in the equine industry (farriers; horse trainers etc.); e.g. creating support teams with CPA/Lawyer/Insurance

These suggestions are purely intended to spark ideas. We welcome paper submissions on any topic pertaining to the Business of Practice.

Want to know how your AAEP Annual Convention program came together?

The Educational Programs Committee (EPC) is charged with creating and reviewing educational content to produce high-quality CE for the AAEP. The committee is composed of AAEP member volunteers from both small and large private practices as well as academia and industry. Members include both general practitioners and specialists.

The Nashville scientific program includes invited papers for the "In-depth" and "How to" sessions as well as sessions comprised of papers that independent authors submitted for consideration. Topic choices for the invited "In Depth" and "How To" sessions are based on member feedback from the 2010 AAEP CE Needs Analysis survey. Topic session leaders are selected by the Program Chair, and then these session leaders invite a slate of speakers to prepare the papers that become an "In Depth" overview or a series of related "How To" talks. Although invited, these papers undergo a rigorous peer review process by the EPC.

Papers submitted by independent authors are each assigned 3 reviewers from the EPC. The reviewers do not know the names of the authors. Content is scored using the criteria of Study Design, Study Quality, Innovation and Impact, Practicality, and Manuscript Quality. Once papers are scored they are discussed by the section facilitators and reviewers. The highest ranking papers are included on the program to accommodate the number of slots available. This year 218 papers were submitted for the 78 available slots on the program.

Non-scientific sessions addressing business, welfare, ethical and industry concerns are also planned as the scientific program materializes. Speakers who are invited to participate in these sessions prepare papers that are also reviewed by members of the EPC for inclusion in the Proceedings.

The peer review process for the AAEP Proceedings is rigorous. It requires an enormous effort by every one of the 46 members of the EPC member to create the best possible program for the AAEP membership. Several thousand volunteer hours were spent putting together the Nashville program, so please thank them for all their hard work creating this program for you.

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Ethics and Equine Medicine: Curse, Pipedream, or Opportunity?

David W. Ramey, DVM

Equine veterinarians exercise special knowledge and skill. The use of this special knowledge is governed by society, through laws, as a service to the public. Generally, those governing are not veterinarians. Ethics is a moral endeavor, and even though practical applications of ethics may not always be black and white, a strong code of professional ethics, practiced both individually and organizationally, helps to keep the public's trust in equine veterinarians and encourages the horse-owning public to continue seeking their advice and services. Author's address: PO Box 16883, Encino, CA 91416; e-mail: ponydoc@pacbell.net. © 2013 AAEP.

1. Introduction

Ethics is a branch of philosophy that involves systematizing, defending, and recommending concepts of right and wrong conduct. Etymologically, the term ethics originates with horses, coming from the Greek word *ethos*, meaning "accustomed place" (as in ἤθεα ἵππων "the habitat of horses").[1] Late Latin borrowed the Greek word as *ethicus*, the feminine of which (*ethica*, for ἠθική φιλοσοφία "moral philosophy") is the origin of the modern English word "ethics." One of the earliest examples of a code professional ethics is the *Hippocratic Oath.*[a]

Equine veterinarians make judgments and informed decisions and possess and apply skills that, in general, the public cannot make and does not have. When members of the public lack training and expertise in any area, they can be easily exploited. Thus, to prevent exploitation of the public and to preserve their own integrity, most professions have internally enforced ethical codes of practice to which members of the profession are expected to adhere. These codes allow a profession to define a standard of conduct. Ethical standards also help maintain the public's trust in a profession, encouraging the public to continue seeking their services.

2. What Behaviors Characterize Ethical Professional Conduct?

Professional organizations may break down their ethical approach into discrete components.[2] These components include:

- Honesty: Truthfulness and straightforwardness along with the absence of lying, cheating, or theft. This would include honesty about the potential effectiveness of treatments being offered.
- Integrity: An adherence to moral principles.
- Transparency: Operating in such a way that it is easy for others to see what actions are being performed.
- Accountability: Answerability, blameworthiness, liability, and the expectation of assumption of responsibility for one's actions.
- Confidentiality: The expectation that the vet-

NOTES

erinarian will hold secret all information relating to a patient, unless there is consent permitting disclosure from the client.
- Objectivity: Fairness, having no personal stake, factuality, and nonpartisanship (that is, not acting in one's own self-interest).
- Respectfulness: Courteous regard for people's feelings.
- Obedience to the law.

Ethical standards and behavior provide confidence to the public about the reliability and actions they can expect when using the services of a professional. They build trust. In maintaining a strong veterinarian-client relationship, trust is essential. Trust can be hard to both gain and maintain, but it can be lost very easily. Still, if veterinarians should act ethically, and if ethical practice improves the public's perceptions of professions, one might reasonably wonder why it is necessary to have discussions on ethics at all in professional organizations and why ethical practice may sometimes seem to be such an elusive goal. To answer these questions, it is necessary to look at the players in the ethical engagement and to examine the obstacles to ethical practice.

People Have Their Own Ethics

Ethics is a philosophical discipline, and it has been argued that ethics is objective, perhaps most eloquently by the Russian-American philosopher Ayn Rand, in books such as *The Fountainhead* (1943) and *Atlas Shrugged* (1957). However, one individual's moral and ethical judgments may be clearly different from another. As such, individuals may disagree about a certain moral or ethical situation, even if they share many of the same morals and ethics.

Nevertheless, it can be argued that some ethics are better than others. If that is the case, it should be possible to ascertain which ethics are better through a critical examination of the reasons underlying one's practices and beliefs. So, for example, practices that are fairer, more honest, kinder, and more considerate are likely to be considered more ethical than practices that are unfair, dishonest, cruel, or thoughtless, and particularly when such practices are judged by society at large.

Ethical Responsibilities to Oneself

Ethical concerns are more than simply how veterinarians treat animals. Whatever else veterinary medicine may be, it is also a business by which veterinarians support themselves and their families. Although ethical conduct is important to how professionals relate to others, it is also important to consider the ethical responsibilities to oneself.

Unfortunately, the "ethical responsibility to myself," argument may be used as a justification for putting the veterinarian's needs above obligations to the horse, the horse owner, or society. Ethics that are overly self-oriented tend to foster irresponsible and dishonest conduct. Under such circumstances, actions become Darwinian, in which the ultimate goal becomes survival ("If I don't do it, someone else will"). Such Darwinian behavior was captured by Joseph Heller, in his 1961 novel, *Catch-22.*

"From now on I'm thinking only of me."

Major Danby replied indulgently with a superior smile: "But, Yossarian, suppose everyone felt that way."

"Then," said Yossarian, "I'd certainly be a damned fool to feel any other way, wouldn't I?"

Ultimately, this type of conduct—even if it is practiced by only one individual—undermines the integrity of both the individual professional as well as of the profession itself.

It can be difficult to be ethical and consider more than oneself, especially if one practices in a world full of competing desires (eg, "The horse first" versus "If we win this class we qualify for the championships!") and pressures to compromise. If it were easy to be ethical, there would be no need for discussions on ethics. While doing the right thing and acting fairly and ethically is at the foundation of building a professional reputation, there are also times when telling the truth or refusing to perform a procedure may result in the loss of the client and may not be in a veterinarian's immediate self-interest. There are times when acting ethically carries a cost. Ultimately, the acid test of one's character and commitment to ethics is whether he or she will do the right thing even if it is not necessarily in his or her immediate best interest to do so.

Ethical Responsibilities to Society (Social Responsibility)

The idea that an individual or an organization has a responsibility to society is called social responsibility. Being socially responsible means that an individual or organization has responsibilities that lie beyond their own immediate self-interest. Whereas the goal of a business may be thought to maximize profits, social responsibilities imply that business activities should also benefit society at large and not be solely devoted to the bottom line. As it concerns equine medicine, this would mean that the success of a particular veterinary endeavor should not only be determined by how much money it makes, but also by how many horses and people it helps. "Equitarian" endeavors, or providing volunteer assistance at rescue facilities, pony clubs, or other organizations, for example, would be good examples of veterinarians fulfilling their ethical responsibilities to society. Such actions benefit the veterinary profession in general by generating good will and positive press as well as the individuals who participate in such endeavors.

Professional organizations such as the American Association of Equine Practitioners (AAEP) also maintain ethical codes to inform society of its ethical principles. If individual veterinary practitioners fail to meet the ethical standards of the organiza-

tion, their membership may be revoked by the AAEP. Such disciplinary action is intended to protect those professionals who act with conscience to practice from being undermined commercially by those who have fewer ethical qualms. Unfortunately, in cases in which professional bodies regulate their own ethics, there are opportunities for such bodies to become self-serving and to fail to follow their own ethical code when dealing with renegade members and in responses to societal pressures. If professional bodies fail to regulate their own ethics, it can cast an entire profession into disrepute and lead to onerous external regulation. Thus, it is as important for professional organizations to act as ethically and responsibly as it is for its individual members.

Ethical Responsibilities to the Client

Veterinary medicine differs from most of human medicine in that the client and the patient are not the same individual (or species). As such, the veterinarian's role with the client and his or her equine patient is most closely akin to the role of the pediatrician, whose patient is the child, but who has to deal with the parent concerning matters of treatment as well as payment for services rendered.

Although many clients want only what is best for their animals, others—particularly in performance horse disciplines—may have other, competing interests. For example, the goal of getting to the next show, the next race, or the next event may be more important to a client than the health of the horse. Some clients may even be willing to risk injury to the animal in the pursuit of short-term gain. Further, clients can sometimes stand in the way of effective treatment for their horses, however much they "love" them. It is not the ethical responsibility of the veterinarian to simply accede to a client's demands. The veterinarian's ethical responsibility to self, as well as the responsibility to the horse, supersedes such demands. Veterinarians who act at the whim of a client also put themselves in a vulnerable position, should a procedure go wrong; the defense, "I was just doing what the client wanted," is not tenable, should an adverse reaction occur, and the procedure is not justifiable medically. The law generally assumes that the veterinarian's primary duty is to the animal and not to the client.[3]

Still, in the eyes of the law, owners also have virtually complete control over their animals, with the exception of the laws barring overt cruelty and neglect. Owners may choose not to treat sick animals, may choose to euthanize a sick animal, or, on the other hand, may demand some therapy to "improve performance" that may not be in accordance with industry standards, or even law. This state of affairs can create a major problem for veterinarians who, embracing the pediatrician model, want to act only in the interest of the horse. The veterinary clinician does not have the power of law behind him or her to force the owner to take (or prevent) action. Thus, although some veterinarians may see their role as analogous to pediatricians, society (ie, the legal system) has not yet caught up with the ethics underlying that view, even though many members of society would probably agree with it.

Aesculapian authority, the traditional "godlike" place of honor given to physicians in society and referring to Aesculapius, the Greek and Roman god of medicine and healing, is probably the veterinarian's most powerful tool for getting clients to act in the best interest of the animal.[4] However, this authority can easily fail if a veterinarian fails to fulfill his ethical responsibilities. For example, a veterinarian could advocate for an unproven therapy without disclosing the fact that the therapy was unproven, causing the client to waste time and money, with no benefit for the animal. This action would be clearly unethical, lacking both honesty and transparency. If such action were discovered, the discovery probably would reflect poorly on the veterinarian as well as the veterinary profession in general.

Societal Obligations to Horses and Owners

Whereas veterinarians may have ethical obligations to society, society has essentially no ethical obligations to veterinarians, or to horse owners. Societal concerns—undoubtedly expressed by the majority of the population that does not own horses—are simply for the perceived welfare of horses. In general, societal concern for animal welfare is increasing.

For example, society regulates how owners can keep horses, with laws on the books pertaining to horse welfare and abuse. As such, horse owners cannot care for their horses in any manner that they choose, even if those owners proclaim themselves to be horse care "experts." Owners who fail to care for their horses in accordance with societal standards can have their horses taken away from them; individuals who treat their animals in a particular fashion can be fined or jailed.

There are numerous examples of society's interest in the ethical treatment of horses. The failure of the Tennessee Walking Horse industry to protect horse welfare has led to Federal legislation banning the practice of "soring."[5] Even though those with "expertise" in that industry may feel that they should be able to use whatever means necessary to get their horses to move in a certain fashion, clearly, society feels differently, and has regulated accordingly. Numerous other examples of societal regulations in the horse world—made with or in spite of advice from the veterinary community—exist, including:

(1) Federal legislation banning horse slaughter has been proposed and introduced in the United States Congress several times (*The Equine Prevention of Cruelty Act*[6]). Although the act has not been passed, these proposed laws were introduced over the ob-

jections of both the AAEP and the American Veterinary Medical Association (AVMA). As irresponsible as such laws might be (at least in the eyes of some), it is inarguable that they were introduced because of societal concerns.

(2) The Pregnant Mare's Urine (PMU) Industry. The PMU industry was the subject of intense societal scrutiny in the late 20th century. As a result of that pressure, improved regulations were put in place concerning care of the mares and foals. Because of concerns about slaughter of the foals necessary for the perpetuation of the industry, PMU farms now also use horse breeds that will produce more marketable foals.[7] Published ethical standards and regulations might have helped prevent some of the furor that was directed at that industry.

(3) The Carriage Horse Industry. Although many feel that horse-drawn carriage rides are picturesque and romantic ways to see major metropolitan areas, others feel that it is a form of equine abuse.[8] Ultimately, the fate of the carriage horse industry will result from a tug of war between those interests. Those interested in maintaining a viable carriage horse industry would do well to ensure that their horses are treated ethically and humanely, and do so loudly, for example, by publishing guidelines and schedules for examinations and treatment, to allay concerns from those who feel that Carriage Horses should be banned from city streets.

Societal Obligations to Equine Veterinarians

Society ultimately regulates the practice of equine veterinary medicine on the basis of two things: (1) perceptions that veterinary medicine is a profession with "special knowledge" and (2) perceptions of how veterinary medicine is fulfilling its obligations (including ethical ones) to the horse, horse owners, and society.

Society does not necessarily grant veterinarians a free pass pertaining to special knowledge. Indeed, laws pertaining to the regulation of veterinarians are mutable and have been amended to allow the practice of various modalities by non-veterinarians (eg, "lay dentists" in several states; acupuncturists in Maryland). It should be obvious from the recent changes to laws in various states that the mere holding of a veterinary license does not guarantee that the practice of veterinary medicine will retain its protected status. To help retain that status, veterinarians should consider it their ethical responsibility to society to advance knowledge of the practice of veterinary medicine and should use that knowledge to come up with better approaches for diagnosis and treatment, to confirm the utility of existing treatments, and to discard ineffective treatments.

Perceptions of how veterinary medicine is fulfilling its obligations to society are also important. It has been said that, "Perception is reality"; the way others perceive veterinarians is "reality" for the veterinarian. For example, if veterinarians are perceived as, "In it for the money" or as acting in ways deemed merely to protect their own turf—as opposed to acting in the best interest of the horse—society may choose to ignore the veterinarian's voice when discussing its concerns about horses. Among the most powerful influences on the perception of professions is their ethical conduct.

In November 2012, Gallup polling service released their Honesty/Ethics in Professions survey, rating the honesty and ethical standards of people in different fields.[9] At the top of the list were nurses (85%), people who generally do not make primary care decisions but devote their careers to providing care. On the other hand, professions at the bottom of the list, such as car salespeople and members of the United States Congress, suffer from the perception that their incentives often are not aligned with the interests of their customers.[b] The Angus Reid Three Country Public Opinion Survey found veterinarians to be very highly thought of, with 92%, 89%, and 86% of respondents characterizing veterinarians as deserving of a "great deal" of respect in Canada, the United States, and Great Britain, respectively.[10] In general, individuals whose work requires them to respond to the needs of others—not only medical professionals but others, such as firefighters, military personnel, and teachers—tend to rate very highly in public opinion surveys, whereas those that are seen as being in it for their own benefit—for example, lawyers, politicians, and used car salesmen—tend not to fare well.[11] Ultimately, it will be up to the veterinary profession to decide where on such lists it wants to fall.

Client Obligations to the Veterinarian

Clients have few ethical responsibilities to their veterinarians. One could reasonably assert that clients have an ethical responsibility to pay their bills and to tell the veterinarian the truth about their horse(s), as well as about their concerns and expectations. However, as anyone who has practiced knows, they may not always fulfill those responsibilities. Furthermore, clients have no ethical responsibilities to even use veterinarians; societal concerns, as reflected by law, are that horses are cared for but do not necessarily mandate who provides care. The point is that insofar as a veterinarian-client relationship goes, ethical obligations can be something of a one-way street. In their own interest, veterinarians would be well served to seek out clients who act ethically and who have the best interest of their horses in mind; clients and their horses will be best served by veterinarians with an ethical bent.

Ethical Responsibilities to the Horse

Ideally, in regard to the care and treatment of horses, the ethical responsibilities of horse ownership, societal interests in horse welfare, and veterinary medical treatment should mirror each other, all working together for the best interest of the horse. Ethically speaking, the veterinarian's moral duty is to the patient; the veterinarian is obliged by the nature of the veterinary profession to act in the best interest of the patient; consequently, veterinarians must avoid orders or requests from the third party that are not in the best interest of the patient.

Unfortunately, client demands—for example, for "better" performance—may not be in the best long-term health interest of the horse. This fact sometimes makes the veterinarian's goal of ethical practice in the best interest of the horse's health difficult. In addition, societal concerns—even if they are well-meaning—may not be expressed in ways that are beneficial for the horse. For example, whereas the goal of allowing horses to run free on the range in the American West may be laudable, it may not be in the best interest of the horses if there is not sufficient range to sustain or there is a lack of sufficient predators to control their herds.[12] Nevertheless, by virtue of their special knowledge about horses, ethical veterinarians are in the best position to argue for the welfare of the horse, and it is to the ultimate advantage of both the horse and the veterinary profession that they do so.

3. Responding to Ethical Challenges

Whereas veterinarians have a special authority[c] that helps buttress their legal authority to practice medicine on animals, there are a number of challenges that can confront even the most ethical of veterinarians.[13] The way in which veterinarians handle such challenges will ultimately determine the long-term health of the veterinary profession. Such challenges include the following.

(1) The ability of clients to readily obtain their own pharmaceutical products from on-line pharmacies, feed stores, and so forth. Owners can obtain virtually any substance they believe a horse needs.

Veterinarians have typically made a living by selling two things: products and services. The human medical field has traditionally chosen to outsource its products to pharmacies; this has avoided the ethical conflict inherent in selling a product that an individual prescribes.[d]

The only thing that veterinarians have that is unique is their special knowledge. Individual products do not vary among vendors; the only thing that may differ is price. Veterinarians simply cannot compete with large companies offering drugs at discount prices and would probably be wise not to try to do so. Indeed, some states require veterinarians to write prescriptions for clients to have filled elsewhere if requested by the client for an animal being treated by the veterinarian (veterinarian's do not have to, and should not, respond to requests for blank prescriptions). The AVMA's *Principles of Veterinary Medical Ethics*[14] recommends that veterinarians comply with their client's wishes and provide written prescriptions if the client prefers having the prescription filled elsewhere, assuming, of course, the existence of a veterinary-client-patient relationship.

The easy availability of prescription medications to clients does not mean that veterinarians cannot act ethically, nor does it mean that veterinarians cannot sell products (although prescribing products that one sells does carry its own inherent conflict of interest). The AVMA notes several advantages when clients purchase their medications from veterinarians, all of which are consistent with good medicine and ethical practice.[15]

- Veterinarians have medications in stock and can provide medications immediately.
- Veterinarians can answer questions, provide instructions for use, and demonstrate how to give medications.
- Medications controlled by veterinarians have the assurance of having been handled properly.

Clearly, there are many types of businesses that show little concern for any of the ethical issues associated with selling prescription medications. However, "fighting fire with fire" by offering drugs on demand is unethical; according to the AVMA *Principles of Veterinary Medical Ethics,* it is unethical, and in most states, unlawful, for a veterinarian to write a prescription or dispense a prescription drug outside a veterinarian-client-patient relationship. Rather than compete directly against unethical selling of drugs by engaging in that practice, ethical veterinarians might consider other business models, for example, practicing more along the model of "concierge medicine" in human medical practice, focusing on providing care, as opposed to selling products.[16,17]

(2) Competing non-veterinarian "experts" (in a variety of areas), many of whom state that they provide services about which veterinarians either do not know or do not care (eg, care of the horse's teeth), and a seemingly endless array of products and services.

Veterinarians are in the business of providing care; horse owners are generally interested in providing the care that their horses need. However, a variety of factors—including advertising, lobbying, differing ideologies, competitive pressures, and economic interests—have led some people to believe that a variety of treatments, supplements, or diagnoses are important for the welfare of their horses.

Veterinarians are uniquely qualified to help clients determine which products and services are good for their horses. By virtue of their special

knowledge about horses and their ability to conduct scientific research, they can help determine whether such products and services are not in the best interest of the horse or of their client's pocketbook.

It may not be advisable to try to compete against other "experts" on their own turf. For example, equine veterinary "dentists" do not offer an obvious advantage to lay "tooth floaters" in the eyes of clients. If, by virtue of the ethics and knowledge, veterinarians only perform services that are necessary and supported by good science, it will build client trust. Any other attempts to maintain professional hegemony over the horse's mouth are likely to be derided as "turf protection."

In that same vein, veterinarians are probably at a disadvantage when competing with chiropractors (who actually have chiropractic degrees), as well as many other non-veterinarian service providers. Similarly, veterinarians who provide special products or supplements may have a difficult time in distinguishing themselves from other purveyors of such products. Veterinarians who engage in such competition risk being viewed as, "In it for the money," by their peers, and when and if such products are shown to be a waste of time and money, by the public, to the detriment of both the individual veterinarian(s) and the veterinary profession.

The rationale for such products and services is often presented in terms of client satisfaction, for example, "what the customer wants." Although providing what the customer wants may be thought of as desirable, in human medicine, studies indicate that it may not be desirable. In 2012, a study of more than 50,000 US adults found that patients who grade their care the highest are more likely to have worse health outcomes and accrue more medical expenses than the least-satisfied patients, even after adjusting for factors such as age, income, illness severity, and insurance coverage.[18] Ethical veterinarians should focus on what is best for the horse and not on competing in areas in which they have no special knowledge or training. They will be rewarded in terms of client loyalty when owners recognize that their veterinarian is interested in the welfare of their horse(s), first and foremost.

(3) "If I don't do it, somebody else will."

This is a famous and time-honored rationalization known as the "futility illusion." The rationale is clear enough. An action, for example, administering a forbidden substance to a horse, is objectionable. The concern is that if the veterinarian who is present does not undertake the action, someone else will, and the substance will be administered regardless. Consequently, the conclusion goes, there is no point in not giving the substance, because it will not prevent the action, and business will be lost. The fear is that ethical refusal to do the right thing will only hurt the ethical person without preventing the unethical action. Of course, the rationale is faulty, self-serving, and absurd. If a group of young people were to come across a solitary old person, and one of the group decided that the old person should be robbed and could not be dissuaded, the rest of the group would not be blameless if they decided to participate in the robbery because it was going to happen anyway.

In that same vein, it may also be argued that it is more humane for an unethical procedure (ie, tail blocking, or cutting a tail) to be performed by a veterinarian, with the use of aseptic technique, adequate sedation, and post-procedure analgesia, than it is to subject the animal to the risk of such procedures performed by an untrained individual. When considered in a vacuum, that is undoubtedly the case; invasive procedures should be performed by trained individuals with the use of adequate postoperative analgesia. Whereas the "futility illusion" would still apply under such circumstances, the fact is that the procedure being performed would still be unethical, and its performance by veterinarians would only serve to perpetuate the unethical practice and thus could not be condoned.

The futility illusion is invoked when someone has something to lose (ie, business) by not doing the dubious action, or something to gain (ie, money) by doing it. In fact, there are times when someone else will not do it. In 1989, months before the Berlin Wall fell, Hungarian border guard Bella Arpad refused to obey orders to shoot East Germans crossing to freedom. No one else shot, either, and history changed.[19] Furthermore, the impact of a refusal to act unethically can also lead to a good result, even when the action happens. The individual's determination to do right is always desirable in itself. Under any circumstances, the futility illusion is merely an excuse for not acting in the best interest of the horse.

(4) Providing treatments.

When horses need treatment for disease or injury, it is in their best interest that they receive the treatments that are most likely to be effective. Of course, some conditions—for example, tendon injuries or osteoarthritis—lack universally effective treatments. Thus, new treatment opportunities are being constantly advanced to veterinarians. However, because of any number of factors, including lack of funding, lack of sufficient numbers, and lack of incentive, many such treatments are advanced with little or no evidence of efficacy.

There is certainly nothing wrong with providing new therapies, per se. However, the ethics of informed consent mandate that the person selecting a therapy for his or her horse fully understand:

- the nature of the decision/procedure
- reasonable alternatives to the proposed intervention
- the relevant risks, benefits, and uncertainties related to each treatment[20]

The failure to provide necessary information so that the client fully understands what is known about the potential risks and benefits of a treatment is an ethical failure on the part of the person providing treatment. Furthermore, therapies should not be promoted on the basis of criteria that are not relevant to the likelihood of a successful outcome for the horse ("new," "natural," etc).

Other difficult ethical dilemmas may occur when a client's ideology differs from that of the veterinarian. To take a simple example, consider a horse suffering from chronic laminitis, in which there are no realistic options except euthanasia. Despite many veterinarians' opinions that euthanasia decisions should be left up to the client, there are cases in which the client refuses to let go, and the horse suffers. Although the owner may choose to try myriad unproven approaches, in such a case, a veterinarian should consider doing whatever it takes to end the animal's suffering and pull out all stops to persuade the owner to euthanize, if, in the veterinarian's judgment, the animal has no positive quality of life left. Therapies that lack a good foundation can lead to animal suffering as well as a waste of client resources if they do not work.

If a client demands an essentially harmless but probably worthless therapy, perhaps the best a veterinarian can do is articulate his or her reasons for rejecting the therapy in question but not relinquish the client (and the animal) totally to the treatment. Ethical veterinarians should continue to work with their clients, in part to help ensure that unscrupulous practitioners or the cost of new treatments do not financially bleed them and in part to keep the client focused on objective milestones that signify efficacy or lack thereof.

(5) Self-Policing.

Self-policing is a process in which individuals or groups provide their own discipline and enforce it without outside help. Self-policing allows a profession to maintain control over the standards to which they are held. Stringent self-policing serves as a way to handle internal problems within a profession and is an alternative to public relations campaigns to repair the damage that can occur when such internal problems are exposed.

Unfortunately, self-policing attempts often fail because of the inherent conflicts of interest that occur. For example, whereas the legal profession has a model ethical code and more than 400,000 members, its members are not held in high esteem; in fact, the legal profession is routinely the brunt of jokes about its lack of ethics. An organization may be loath to criticize unethical practice of its members because of fears of bad publicity or loss of membership. However, if the public becomes aware of the failure to self-police, an external, independent organization may be given the duty of policing them. For example, in October 2012, New York Governor Andrew Cuomo completed a takeover of horse racing in the state, hoping to end ". . . decades of scandal and mismanagement in an industry important to the state."[21]

It would follow that individuals should also be active in self-policing their own to maintain a high standard of practice. However, individuals may also be reluctant to criticize colleagues because of fear of being ostracized. Even the vernacular used to describe individuals who point out unethical practice is couched in pejorative terms (eg, "ratting out," "snitching," "squealing"). Regardless, outside agencies are more than happy to make sure that veterinarians practice ethically and responsibly; veterinary malpractice is arguably the most rapidly evolving area of the law in the United States.[22,23]

4. Ethics: Curse, Pipedream, or Opportunity?

Ethics may be perceived as something of a curse, particularly by those who feel that ethical concerns constrain a practitioner's ability to do what he or she wants. However, the fact is that veterinary professionals cannot simply do whatever they want —even if they are not constrained by ethics, as the law and the legal system stand ready to provide a reminder. If other individuals—veterinarian or non-veterinarian—act free of ethical concerns, any advantages provided by such freedom are short-lived and illusory.

It is probably a pipedream[e] to think that every veterinarian who practices on horses will behave ethically or with the horse's best interests in mind. However, if ethical practice is perceived as the standard by which veterinarians should operate, it should be possible for ethical veterinarians and the organizations that represent them to lead the fight against such practices. Ultimately, societal concerns about unethical practices result in laws regulating them and open the door to the legal profession to redress complaints through the legal system.

Although ethical practice can be a challenge for equine veterinarians, strict attention to ethical practice provides an opportunity for veterinarians to separate themselves from other providers of products and services to horse owners. Ethics is a powerful driver of clients' perceptions of trust, honesty, and professionalism. If veterinarians are perceived as practicing for the benefit of the horse, their opinions and expertise are likely to be solicited; on the other hand, if they are perceived as being in it for themselves, they may be derided.[24] Professional organizations representing veterinarians should advocate strongly for the ethical treatment of horses and especially before public pressure requires that they do so. Leaders can strongly influence unethical behavior in the workplace.[25] Vocal advocacy for the welfare of the horse will only serve to benefit veterinarians as well as the veterinary profession.

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[a]The term *Primum Non Nocere* ("above all, do no harm") is not stated in the *Hippocratic Oath.* Searches of writings back to the Middle Ages have uncovered the appearance of the axiom as expressed in English, coupled with its unique Latin, in 1860, with attribution to the English physician, Thomas Sydenham. Smith, CP. Origin and Uses of *Primum Non Nocere*—Above All, Do No Harm! *J Clin Pharmacol* 2005;45:371–377.

[b]Veterinarians were not included in the 2012 poll; in the last Gallup survey looking specifically at veterinarians, conducted in 2007, veterinarians scored "very high," with 71% of those polled viewing veterinarians as having high standards of honesty and integrity.

[c]Veterinary authority is derived from a combination of traits—"sapiential" (ie, special wisdom and knowledge); "moral" (deriving from the overwhelming moral imperative to heal, relieve suffering and retard death); and charismatic (derived from the fact that medicine is still related to magic in the eyes of the scientifically and medically naive; ie, most people). This combination has led to state laws regulating the practice of veterinary medicine.

[d]In human medicine, the practice of "doctor dispensing" is under increasing fire, and some states, including California and Oklahoma, have clamped down on the practice. Available at: http://www.nytimes.com/2012/07/12/business/some-physicians-making-millions-selling-drugs.html?pagewanted=all&_r=0.

[e]1870; the sort of improbable fantasy one has while smoking opium; from pipe (n.1) + dream (n.). Old English "pipdream" meant "piping." From the *On-Line Etymology Dictionary.* Available at: http://www.etymonline.com/index.php?term=pipe+dream. Accessed February 1, 2013.

Equine Welfare as a Mainstream Phenomenon

Bernard E. Rollin

The 20th century has witnessed a bewildering array of ethical revolutions, from civil rights to environmentalism to feminism. Often ignored is the rise of massive societal concern across the world regarding animal treatment. Regulation of animal research exists in virtually all Western countries, and reform of "factory farming" is regnant in Europe and rapidly emerging in the United States. In 2012, a series of articles in *The New York Times* focused welfare attention squarely on the horse industry. Opponents of concern for animals often dismiss the phenomenon as rooted in emotion and extremist lack of appreciation of how unrestricted animal use has improved human life. Such a view ignores the rational ethical basis for elevating legal protection for animals. Author's address: Department of Philosophy, Colorado State University, Fort Collins, CO 80523; e-mail: Bernard.rollin@colostate.edu. © 2013 AAEP.

1. Introduction

Businesses and professions must stay in accord with social ethics or risk losing their autonomy. A major social ethical issue that has emerged in the past four decades is the treatment of animals in various areas of human use. Society's moral concern has outgrown the traditional ethic of animal cruelty that began in biblical times and is encoded in the laws of all civilized societies. There are five major reasons for this new social concern, most importantly, the replacement of husbandry-based agriculture with industrial agriculture. Other concerns include demographic changes in society, ethics changes in recent history, rise of scholarly literature on ethics and animals, and media interest. The loss of husbandry to industry has threatened the traditional fair contract between humans and animals and resulted in significant amounts of animal suffering arising on four different fronts. Because such suffering is not occasioned by cruelty, a new ethic for animals was required to express social concerns. Because ethics proceeds from preexisting ethics rather than being created *ex nihilo* (out of nothing), society has looked to its ethic for humans, appropriately modified, to find moral categories applicable to animals. This concept of legally encoded rights for animals has emerged as a plausible vehicle for reform.

2. Background

Although society has always had an articulated ethic regarding animal treatment, that ethic has been very minimalistic, leaving most of the issue of animal treatment to people's personal ethic rather than to the social ethic. Since Biblical times, that limited social ethic has forbidden deliberate, willful, sadistic, deviant, purposeless, unnecessary infliction of pain and suffering on animals, or outrageous neglect, such as not feeding or watering. Beginning in the early 19th century, this set of prohibitions was articulated in the anti-cruelty statutes of the laws in all civilized societies.[1] Even in Biblical and medieval times, however, the social ethic inveighed against cruelty. The *Old Testament* injunctions

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against yoking an ox and an ass together to a plow, or muzzling the ox when it is being used to mill grain, or seething a calf in its mother's milk, all reflect concern with and abhorrence for what the Rabbinical tradition called *tsaar baalei chaiim*; the suffering of living things. In the Middle Ages, St. Thomas Aquinas, while affirming that, lacking a soul, animals enjoyed no moral status, nonetheless strictly forbade cruelty on the grounds that permitting such behavior toward animals would encourage its spreading to human beings, an insight buttressed by more than two decades of recent research.[2] For the overwhelming majority of human history, until some four decades ago, the anti-cruelty ethic served as the only socially articulated moral principle for animal treatment.

The past 50 years have witnessed a dazzling array of social ethical revolutions in Western society. Moral movements such as feminism, civil rights, environmentalism, affirmative action, consumer advocacy, children's rights, the student movement, anti-war activism, and public rejection of biotechnology have forever changed the way governments and public institutions comport themselves. This is equally true for private enterprise: to be successful, businesses must be seen as operating solidly in harmony with changing and emerging social ethics. For example, it is arguable that morally based boycotting of South African business was instrumental in bringing about the end of apartheid, and similar boycotting of some farm products in the United States led to significant improvements in the living situations of farm workers.

Not only is success tied to accord with social ethics but, even more fundamentally, freedom and autonomy are as well. Every profession—be it medicine, law, or agriculture—is given freedom by the social ethic to pursue its aims. In return, society basically says to professions it does not understand well enough to regulate, "you regulate yourselves the way we would regulate yourself if we understood what you do, which we don't. But we will know if you don't self-regulate properly and then we will regulate you, despite our lack of understanding." For example, some years ago, Congress became concerned about excessive use of antibiotics in animal feeds and concluded that veterinarians were a major source of the problem. As a result, Congress was about to ban extra-label drug use by veterinarians, a move that would have killed veterinary medicine as we know it. However, through extensive efforts to educate legislators, such legislation did not proceed to law.

One major social ethical concern that has developed over the past four decades is a significant emphasis on the treatment of animals used by society for various purposes. It is easy to demonstrate the degree to which these concerns have seized the public imagination. According to members of both the US National Cattlemen's Beef Association and the National Institutes of Health (the latter being the source of funding for the majority of biomedical research in the United States)—both groups not inclined to exaggerate the influence of animal ethics—by the early 1990s, Congress had been consistently receiving more letters, phone calls, faxes, e-mails, and personal contacts on animal-related issues than on any other topic.[a,b] Whereas 30 years ago one would have found no bills pending in the US Congress relating to animal welfare, recent years have witnessed dozens of such bills annually, with even more proliferating at the state level. The Federal bills have ranged from attempts to prevent duplication in animal research, to saving marine mammals from becoming victims of tuna fishermen, to preventing importation of ivory, to curtailing the parrot trade. Ethical concerns about the welfare of horses has resulted in passage of the *Federal Horse Protection Act,* banning "soring" of Tennessee Walking Horses, and Federal legislation banning horse slaughter has been proposed and introduced in the US Congress several times.

State laws passed in large numbers have increasingly prevented the use of live or dead shelter animals for biomedical research and training and have focused on myriad other areas of animal welfare. Eight states have abolished the steel-jawed leg-hold trap, as have some 90 countries.[3] When Colorado's politically appointed Wildlife Commission failed to act on a recommendation from the Division of Wildlife to abolish the spring bear hunt (because hunters were liable to shoot lactating mothers, leaving their orphaned cubs to die of starvation), the general public ended the hunt through a popular referendum. Seventy percent of Colorado's population voted for this as a *Constitutional Amendment*.[4] In Ontario, the environmental minister stopped a similar hunt by executive fiat in response to social ethical concern.[5] California abolished the hunting of mountain lions, and state fishery management agencies have been taking a hard look at catch-and-release programs on humane grounds.[6]

According to the director of the American Quarter Horse Association, the number of state bills related to horse welfare filled a telephone-book–sized volume in 1998.[7] Public sentiment for equine welfare in California carried a bill through the State Legislature, making the slaughter of horses or shipping of horses for slaughter a felony in that state. Municipalities have passed ordinances ranging from the abolition of rodeos, circuses, and zoos to the protection of prairie dogs, and, in the case of Cambridge, Massachusetts (a biomedical Mecca), the strictest laws in the world regulating research.

Even more dramatic, perhaps, is the worldwide proliferation of laws to protect laboratory animals. In the United States, in 1985, for example, two major pieces of legislation regulating and constraining the use and treatment of animals in research (which I helped draft and defend) were passed by the US Congress in 1985 despite vigorous opposition from the powerful biomedical research and medical lob-

bies. This opposition included well-financed, highly visible advertisements and media promotions indicating that human health and medical progress would be harmed by implementation of such legislation. For example, there was a less-than-subtle film titled "Will I Be All Right, Doctor?" The query came from a sick child. The response came from a pediatrician who affirmed, in essence, "*You* will be [a sick child] if '*they*' don't leave us alone to do as we wish with animals." Social concern for laboratory animals was unmitigated by such threats, and research animal protection laws moved easily through Congress and were implemented at considerable cost to taxpayers. When I testified before Congress on behalf of this law in 1982, a literature search in the Library of Congress turned up no papers in the scientific literature on laboratory animal analgesia and only two on animal analgesia, one of which said "there ought to be papers." Now there are more than 11,000. In 1986, Britain superseded its pioneering act of 1876 with new laws aimed at strengthening public confidence in the welfare of experimental animals.[8] Many other countries have moved or are moving in a similar direction, despite the fact that some 90% of laboratory animals are rats and mice.

Inevitably, agriculture has felt the force of social concern with animal treatment—indeed, it is arguable that contemporary concern in society with the treatment of farm animals in modern production systems blazed the trail leading to a new ethic for animals. As early as 1965, British society took notice of what the public saw as an alarming tendency to industrialize animal agriculture by chartering the Brambell Commission, a group of scientists under the leadership of Sir Rogers Brambell, who affirmed that any agricultural system failing to meet the needs and natures of animals was morally unacceptable.[9] In 1988, the Swedish Parliament passed, virtually unopposed, what *The New York Times* calls a "Bill of Rights" for farm animals, abolishing in Sweden, in a series of timed steps, the confinement systems currently dominating North American agriculture.[10] European Union legislation banning sow stalls in all newly built, rebuilt, or newly commissioned buildings since 2003 has been passed, beginning in 2013.[11]

Although the United States has been a latecomer to agricultural issues, things have moved rapidly, with referenda pressed by the Humane Society of the United States abolishing sow stalls, battery cages, and veal crates across the United States.

In 2008, the Pew Commission (better known as the National Commission on Industrial Farm Animal Production) called for the end of high confinement animal agriculture within 10 years, for reasons of animal welfare, environmental despoliation, human and animal health, and social justice.[11] Most dramatically, an agreement between the Humane Society of the United States and the Colorado Livestock Association passed jointly sponsored farm animal welfare law in Colorado in 2008, abolishing sow stalls and veal crates. The agriculture community in the United States has been far behind societal concern, and it is apparent from articles appearing in *The New York Times* in 2012 that the equine community has lagged as well.

Science Versus Ethics

There is one monumental conceptual error that is omnipresent in the scientific, agricultural, and equine industries' discussions of animal welfare, an error of such magnitude that it trivializes the industry's responses to ever-increasing societal concerns about the treatment of agricultural animals. When one discusses farm animal welfare with industry groups or with the American Veterinary Medical Association, one finds the same response; animal welfare is a matter of "sound science." For example, one representative of the Pork Producers, testifying before the Pew Commission, answered that whereas people in her industry were quite "nervous" about the Commission, their anxiety would be allayed were we to base all of our conclusions and recommendations on "sound science." Hoping to rectify the error in that comment as well as educate the numerous industry representatives present, I responded to her as follows: "Madame, if we on the Commission were asking the question of how to raise swine in confinement, science could certainly answer that question for us. But that is not the question the Commission, or society, is asking. What we are asking is, ought we raise swine in confinement? And to this question, science is not relevant." Similarly, discussions about whether the incidence of exercise-induced pulmonary hemorrhage is reduced by pharmaceuticals such as furosemide[12] generate the question of whether horses that bleed from the lungs after exercise should be running.

Questions of animal welfare are at least partly "ought" questions, questions of ethical obligation. The concept of animal welfare is an ethical concept to which, once understood, science brings relevant data. When we ask about an animal's welfare, or about a person's welfare, we are asking about what we owe the animal and to what extent. A document called the Council for Agricultural Science and Technology (CAST) report, first published by US Agricultural scientists in the early 1980s, discussed animal welfare and affirmed that the necessary and sufficient conditions for attributing positive welfare to an animal were represented by the animals' productivity. A productive animal enjoyed positive welfare; a non-productive animal enjoyed poor welfare.[13] Presumably, for horses, analogous measures under such a concept would be race times, numbers of blue ribbons, or high scores on a dressage test.

However, the notion that welfare is somehow tied to productivity is fraught with many difficulties. Productivity is an economic notion predicated of a

whole operation; welfare is predicated of individual animals. An operation such as caged laying hens may be quite profitable if the cages are severely overcrowded, yet the individual hens do not enjoy good welfare. An equine racing stable may be quite profitable, but individual animals may be severely injured in the course of racing or training. Equating productivity and welfare is, to some significant extent, legitimate under husbandry conditions, in which the producer does well if and only if the animals do well. However, if animals do not naturally fit in the niche or environment in which they are kept, or, as in the case of many performance horses, if they are indiscriminately given performance-enhancing drugs, pain relievers, and so forth so that they can run faster or jump higher, concerns about welfare become paramount.

Ethics and Welfare

Equine welfare is an ethical concept. If the underlying rationale for caring for horse is, "What we owe horses, and to what extent, is simply what it takes to get them to create profit for people," this implies that the horses are well-off if they have only food, water, and shelter. Increasingly, society is taking an issue with such a view.

In Great Britain, during the 1970s, the Farm Animal Welfare Council (FAWC) affirmed that "The welfare of an animal includes its physical and mental state and we consider that good animal welfare implies both fitness and a sense of well-being. Any animal kept by man must at least be protected from unnecessary suffering. We believe that an animal's welfare, whether on farm, in transit, at market, or at a place of slaughter, should be considered in terms of 'Five Freedoms':[14]

(1) Freedom from Hunger and Thirst—by ready access to fresh water and a diet to maintain full health and vigor.
(2) Freedom from Discomfort—by providing an appropriate environment including shelter and a comfortable resting area.
(3) Freedom from Pain, Injury, or Disease—by prevention or rapid diagnosis and treatment.
(4) Freedom to Express Normal Behavior—by providing sufficient space, proper facilities, and company of the animal's own kind.
(5) Freedom from Fear and Distress—by ensuring conditions and treatment that avoid mental suffering."

The "correct" notion of man's moral obligation to horses cannot be decided by gathering facts or doing experiments—indeed, which ethical framework one adopts will in fact determine the shape of science studying animal welfare. For example, if one holds the view that a horse is well-off when it is winning races, the role of welfare science in this case will be to study what feed, bedding, temperature, medication, and so forth are most efficient at getting horses to run the fastest, or which techniques are the best at repairing injuries: much what veterinary science does today. On the other hand, if one takes the FAWC view of welfare, efficiency will be constrained by the need to acknowledge the animal's natural behavior and mental state and to ensure that there is minimal pain, fear, distress, and discomfort. Thus, in a real sense, sound science does not determine concepts of welfare; rather, concepts of welfare determine what counts as sound science!

The failure to recognize the inescapable ethical component in the concept of animal welfare leads inexorably to those holding different ethical views talking past each other. Thus, for example, in the 20th century, producers of pregnant mare's urine (PMU) largely ignored questions of animal pain, fear, distress, confinement, truncated mobility, bad air quality, social isolation, and impoverished environment. Animal advocates, on the other hand, give such factors primacy and were totally unimpressed with how efficient or productive the system may have been and flooded the Internet and newspapers with articles accusing the PMU industry of causing great suffering in horses. The PMU industry ultimately responded to such concerns by breeding foals who were marketable and by self-policing.[15]

The Changing Social Ethic Toward Animals

If the notion of equine welfare is inseparable from ethical components and peoples' ethical stances on obligations to horses differ markedly across a highly diverse spectrum, an important question for the equine industry is, "Whose ethic is to predominate and define, in law or regulation, what counts as equine welfare?" The answer, of course, is society, because society makes the laws.

It is of interest to consider the things that have occurred in society during the last half-century that led to social disaffection with the venerable ethic of anti-cruelty and to strengthening of the anti-cruelty laws, which now make cruelty a felony in over 40 states. In a study commissioned by United States Department of Agriculture to answer this question, I distinguished a variety of social and conceptual reasons[16]:

(1) Changing demographics and consequent changes in the paradigm for animals. Whereas at the turn of the century, more than half the population was engaged in producing food for the rest, today only some 1.5% of the US public is engaged in production agriculture.[17] One hundred years ago, if one were to ask a person in the street, urban or rural, to state the words that come into their mind when one says "animal," the answer would doubtless have been "horse," "cow," "food," "work," and so forth. Today, however, for the majority of the population, the answer is "dog," "cat," "pet." Repeated

studies show that almost 100% of the pet-owning population views their animals as "members of the family"[17]; many horse owners would agree. Virtually no one in society views horses merely as an income source.

(2) We have lived through a long period of ethical soul-searching. For almost 50 years, society has turned its "ethical searchlight" on humans traditionally ignored or even oppressed by the consensus ethic. The same ethical imperative has focused attention on our treatment of the non-human world: the environment and animals. Many leaders of the activist animal movement in fact have roots in earlier movements. As a result, the society's views on the use of animals for entertainment have changed drastically. Previously acceptable activities such as dog- and cock-fighting have been outlawed; others, such as bull-fighting, rodeo, show jumping, and horse racing, are under scrutiny.

(3) The media has discovered that "animals sell papers." One cannot channel-surf across normal television service without being bombarded with animal stories, real and fictional. (A *New York Times* reporter recently told me that more time on cable TV in New York City is devoted to animals than to any other subject.) *The New York Times* reports on the racing and show horse industries in 2012 are a poignant example.

(4) Strong and visible arguments have been advanced in favor of raising the status of animals by philosophers, scientists, and celebrities.[18–23]

(5) Changes in the nature of animal use demanded new moral categories beyond the traditional concept of "cruelty."

Even though all of the reasons listed above are relevant, they are nowhere as important as the precipitous and dramatic changes in animal use that occurred after World War II. These changes were (1) huge conceptual changes in the nature of agriculture and (2) the rise of significant amounts of animal research and testing.

For virtually all of human history, animal agriculture was based foursquare in animal husbandry. Husbandry, derived c. 1300, "management of a household," meant taking great pains to put one's animals into the best possible environment one could find to meet their physical and psychological natures and then augmenting their ability to survive and thrive by providing them with food during famine, protection from predation, water during drought, medical attention, help in birthing, and so on. Thus, traditional animal use was roughly a fair contract between humans and animals, with both sides being better off in virtue of the relationship. So powerful is the notion of husbandry that when the Psalmist seeks a metaphor for God's ideal relationship to humans, he seizes on the shepherd in the *23rd Psalm*. In husbandry, a producer did well if and only if the animals did well, so productivity was tied to welfare. No social ethic was thus needed to ensure proper animal treatment; only the anti-cruelty laws designed to deal with sadists and psychopaths was needed to augment husbandry. Self-interest virtually ensured good treatment.

After World War II, this beautiful contract was broken by humans. Symbolically, at universities, Departments of Animal Husbandry became Departments of Animal Science, defined not as care, but as "the application of industrial methods to the production of animals" to increase efficiency and productivity. If a 19th-century agriculturalist had tried to put 100,000 egg-laying hens in cages in a building, they all would have died of disease in a month; today such systems dominate. The new approach to animal agriculture was not the result of cruelty, bad character, or even insensitivity. It developed rather out of perfectly decent, prima facie plausible motives that were a product of dramatic significant historical and social upheavals that occurred after World War II.

After World War II, agricultural scientists and government officials became extremely concerned about supplying the public with cheap and plentiful food for a variety of reasons. As a result of the Dust Bowl and the Great Depression, many people in the United States had soured on farming, and the American consumer was, for the first time in history, fearful of an insufficient food supply. Urban and suburban encroachment on agricultural land was resulting in a diminution of land for food production. In addition, many farmers had been sent to both foreign and domestic urban centers during the war, thereby creating a reluctance to return to rural areas that lacked excitement; recall the song of the early 20th century, "How're You Gonna Keep 'Em Down on the Farm (After They've Seen Paree)?"[24] When the above considerations of loss of land and diminution of agricultural labor was coupled with the rapid development of a variety of technological modalities relevant to agriculture during and after World War II and with the burgeoning belief in technologically based economics of scale, it was probably inevitable that animal agriculture would become subject to industrialization. This was a major departure from traditional agriculture and a fundamental change in agricultural core values—industrial values of efficiency and productivity replaced and eclipsed the traditional values of "way of life" and husbandry. In addition, in the mid-20th century there arose large-scale use of animals in research and testing for toxicity. This too was an unprecedented large-scale use of animals, lacking the fairness of husbandry agriculture.

These new developments represent a radically different playing field of animal use from the one that characterized most of human history; in the modern world of agriculture and animal research,

the traditional anti-cruelty ethic became increasingly less applicable: the suffering that animals experience at human hands today is not generally of the sort condemned by the anticruelty ethic and laws. Nevertheless, people realize that biomedical and other scientific research, toxicological safety testing, uses of animals in teaching, pharmaceutical product extraction from animals, racing horses to earn enough money to justify their cost of care, medicating horses so that they can win ribbons or belt buckles, and so on all produce far more suffering than does overt cruelty. In horses, this suffering comes from such things facilitating the spread of equine disease in crowded stables, trauma from performance activities that overstress the equine musculoskeletal system, and the like; producing pain, fear, learned helplessness, aggression, and other states for research; poisoning animals to study toxicity; and performing surgery on animals to develop new operative procedures. Indeed, the discomfort and suffering that animals used in research experience by virtue of being housed under conditions that are convenient for us but inimical to their biological natures—for example, keeping rodents, which are nocturnal, burrowing creatures, in polycarbonate crates under artificial, full-time light—arguably equal or exceed the suffering produced by invasive research protocols.

Unintentional Suffering

Farmers, researchers, and horse trainers are often not intentionally cruel. In fact, they are often motivated by plausible and decent intentions: to cure disease, advance knowledge, ensure product safety, provide cheap and plentiful food, attempt to allow riders to ride more safely (by tranquilizing performance horses), and so forth. Nonetheless, they may inflict great amounts of suffering on the animals they use. However, because the traditional ethic of anti-cruelty and the laws expressing it had no vocabulary for describing unintentional suffering, a new set of concepts beyond cruelty and kindness was needed.

Society eventually became aware that new kinds of suffering were engendered by modern animal use. Although overseers of animal use (including horses) could not be categorized as cruel, they were responsible for new types of animal suffering on at least four fronts:

(1) Production diseases arising from the ways the animals are housed. For example, liver abscesses in cattle are a function of certain animals' responses to the high-concentrate, low-roughage diet that characterizes feedlot production; equine gastric ulcer syndrome (EGUS) and colic have been associated with feeding high levels of grain concentrates in the feed[25,26] (these, of course, are not the only cause of liver abscesses, EGUS, or colic). Even the stress of stabling/confinement is considered to play a factor in such diseases. Although a certain percentage of the animals get sick and sometimes die, the overall economic efficiency of feedlots, or the ability to get calories into horses that expend large amounts of energy, is maximized by the provision of such diets. The ideas of a method of care creating diseases that were "acceptable" would be anathema to a husbandry agriculturalist.

(2) The huge scale of industrialized agricultural operations and the small profit margin per animal militate against the sort of individual attention that typified much of traditional agriculture. In traditional dairies 50 years ago, one could make a living with a herd of 50 cows. Today, one needs literally thousands. In the United States, dairies may have 10,000 cows.

(3) Another new source of suffering in industrialized agriculture results from physical and psychological deprivation for animals in confinement: lack of space, lack of companionship for social animals (such as horses), inability to move freely, boredom, austerity of environments, and so on. Because the animals evolved for adaptation to extensive environments but are now placed in truncated environments, such deprivation is inevitable. This was not a problem in traditional, pastoral agriculture or in horses kept in pastures.

(4) In confinement systems, workers may not be "animal smart"; the "intelligence," such as it is, is in the mechanized system. Instead of husbandmen, workers in swine factories are minimum-wage, often animal-ignorant labor. Therefore there is often no empathy with, or concern for, the animals. These sources of suffering, like the ones in research, are again not captured by the vocabulary of cruelty, nor are they proscribed or even acknowledged by the laws based on the anticruelty ethic. Furthermore, they typically do not arise under traditional agriculture and its ethic of husbandry.

As a result of its awareness of new kinds of suffering, society was faced with the need for new moral categories and laws that reflect those categories. Society became concerned with limiting animal suffering in science, in agriculture, and in entertainment events (including many equestrian activities). Western society has extended its moral categories for humans to people who previously were morally ignored or invisible, for example, women, minorities, the handicapped, children, and citizens of the Third World. New and viable ethics do not emerge *ex nihilo*, thus, a plausible and obvious move is for society to continue in its tendency and attempt to extend the moral machinery it has developed for

dealing with people, appropriately modified to animals. This is precisely what has occurred. Society has taken elements of the moral categories it uses for assessing the treatment of people and is in the process of modifying these concepts to make them appropriate (or, in the eyes of some, inappropriate) for dealing with new issues in the treatment of animals, especially their use in science, entertainment, and confinement agriculture.

Extending Ethics to Animals

What aspect of our ethic for people is being so extended? One—quite directly applicable to animal use—is the fundamental problem of weighing the interests of the individual against those of the general welfare. Different societies have provided different answers to this problem. For example, totalitarian societies opt to devote little concern to the individual, favoring instead the state or whatever their version of the general welfare may be. At the other extreme, anarchical groups such as communes give primacy to the individual and very little concern to the group; hence they tend to enjoy only transient existence. In Western society, however, there are usually attempts to strike a balance. Although most of society's decisions are made to the benefit of the general welfare, fences are built around individuals to protect their fundamental interests from being sacrificed to the majority. Thus, we protect individuals from being silenced even if the majority disapproves of what they say; we protect individuals from having their property seized without recompense even if such seizure benefits the general welfare; we protect individuals from torture even if they have planted a bomb in an elementary school and refuse to divulge its location. We protect those interests of the individual that we consider essential to being human, to human nature, from being submerged, even by the common good. Those moral/legal fences that so protect the individual human are called *rights* and are based on plausible assumptions regarding what is essential to being human.

It is this notion to which society in general is looking to generate the new moral notions necessary to talk about the treatment of animals in today's world, in which cruelty is not the major problem but where such laudable, general human welfare goals as efficiency, productivity, knowledge, medical progress, and product safety are responsible for the vast majority of animal suffering. People in society are seeking to "build fences" around animals to protect the animals and their interests and natures from being totally submerged for the sake of the general welfare, and are trying to accomplish this goal by going to the Legislature. In husbandry, this occurred automatically; in industrialized agriculture, in which it is no longer automatic, people wish to see it legislated.

It is necessary to stress certain things that this ethic, in its mainstream version, is not and does not attempt to be. As a mainstream movement, it does not try to give human rights to animals. Because animals do not have the same natures and interests flowing from these natures as humans do, human rights do not fit animals. Animals do not have basic natures that demand speech, religion, or property; thus, according them, these rights would be absurd. On the other hand, animals have natures of their own and interests that flow from these natures, and the thwarting of these interests matters to animals as much as the thwarting of speech matters to humans. The agenda is not, for mainstream society, giving animals the same rights as those of people. It is rather preserving the common-sense insight that, "Fish got to swim, birds got to fly,"[27] and suffer if they don't. This new ethic is conservative, not radical, harking back to the animal use that necessitated and thus entailed respect for the animals' natures. It is based on the insight that what we do to animals matters to them, just as what we do to humans matters to them, and that consequently we should respect that mattering in our treatment of use of animals as we do in our treatment and use of humans.

Importantly, because respect for animal nature is no longer automatic as it was in traditional husbandry agriculture, society is demanding that it be encoded in law. In 2004, no fewer than 2,100 bills pertaining to animal welfare were proposed in US state legislatures. More than 90 law schools now teach animal law. With regard to animal agriculture, the pastoral images of animals grazing on pasture and moving freely are iconic. As the *23rd Psalm* indicates, people who consume animals wish to see the animals live decent lives, not lives of pain, distress, and frustration.

It is in part to try to avoid society's gaze that industrial agriculture, as well as certain equestrian industries, attempt to conceal the reality of its practices from a naïve public; witness Perdue's advertisements about raising "happy chickens," or the California "happy cow" ads. As ordinary people discover the truth about the conditions under which animals are raised or trained, they may be shocked. When I served on the Pew Commission and other commissioners had their first view of sow stalls, many were in tears and all were outraged; an ABC News video on the abuse meted out to Tennessee Walking horses sparked a national outcry.[28]

3. Conclusions

Just as our use of people is constrained by respect for the basic elements of human nature, people wish to see a similar notion applied to animals. Animals, too, have natures, what I call *telos*, following Aristotle—the "pigness of the pig," the "cowness of a cow." Pigs are "designed" to move about on soft loam, not to be in gestation crates. If this no longer occurs naturally, as it did in husbandry, people wish to see it legislated. This is the mainstream sense of "animal rights."

As property, strictly speaking, animals cannot have legal rights. But a functional equivalent to rights can be achieved by limiting human property rights. When I and others drafted the US Federal laws for laboratory animals, we did not deny that research animals were the property of researchers. We merely placed limits on their use of their property. I may own my car, but that does not mean I can drive it on the sidewalk or at any speed I choose. Similarly, our law states that if one hurts an animal in research, one must control pain and distress. Thus, research animals can be said to have the right to have their pain controlled. In the case of farm animals, people wish to see their basic needs and nature, *teloi*, respected in the systems that they are raised. Because this no longer occurs naturally as it did in husbandry, it must be imposed by legislation or regulation.

A Gallup poll conducted in 2003 shows that 75% of the public wants legislated guarantees of farm animal welfare.[29] This is what I call "animal rights as a mainstream phenomenon." Legal codification of rules of animal care respecting animal *telos* is thus the form animal welfare takes when husbandry has been abandoned. Thus, in today's world, the ethical component of animal welfare prescribes that the way we raise and use animals must embody respect and provision for their psychological needs and natures. It is therefore essential that those systems that cause animal suffering by violating animals' natures be phased out and replaced with systems that respect their natures.

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The Ghost of Veterinary Medicine Yet to Come: Lessons Learned From Medical Business Ethics

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Medical ethics is based on the notion of fiduciary obligation, which means that physicians are expected to act in the best interests of their patients, placing their patients' interests even above their own, should they conflict. In recent years, financial conflicts of interest in medicine have threatened this fiduciary relationship. Whereas the repercussions for patients are largely unknown, the repercussions for physicians are painfully obvious: increased paperwork and oversight, restricted relationships, and a climate of suspicion. Just as Ebenezer Scrooge learned from the Ghosts of Christmas Past, Present, and Yet to Come, veterinary medicine can learn from the past and present in the field of human medicine. Authors' address: Bander Center for Medical Business Ethics, Saint Louis University, 3545 Lafayette Avenue, St Louis, MO 63104; e-mail: duboisjm@slu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

A colleague and I recently offered a research integrity training program for medical researchers. Because we offered CMEs (Continuing Medical Education) credits, my co-instructor and I needed to disclose our conflicts of interest. I had none to disclose. However, I was surprised when the CME office at our university contacted me with the requirement that I, as the training program director, develop a management plan for my colleague's conflict of interest. What was his conflict? His wife was on the speakers bureau for a pharmaceutical company. To be clear, neither my colleague nor I have prescribing privileges, and our program was not meant to address medical practice. Yet, I had to fill out a second form for this program, explaining how our program would not be distorted by my colleague's wife's speaking activities (it was probably my 30th conflict of interest form of the year—every publication and talk I give in medicine requires one). I am afraid that most physicians in academic medicine have many similar stories to share—stories of oversight requirement that increase the workload with little obvious benefits to patients or research participants.

In this report, I describe the "original ethic" in medicine, examine how and why this ethic has increasingly been replaced by onerous oversight systems, and suggest that contemporary medicine is the Ghost of Veterinary Medicine Yet to Come.

The Original Ethic in Medicine

In a recent publication, colleagues and I have traced a shift from a virtue ethic in medicine to an ethic of mistrust and oversight.[1] Virtue ethics had a nice,

Editor's Note: The first-person voice is that of James DuBois, who will deliver this presentation. Elena Kraus is a contributing author, who provided extensive literature review.

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long run in medicine. We see it articulated clearly in the writings of Sir William Osler at the end of the 19th century. We readily think of self-interest and competition as 21st-century phenomena, yet Osler wrote: "In these days of aggressive self-assertion, when the stress of competition is so keen and the desire to make the most of oneself so universal, it may seem a little old-fashioned to preach the necessity of virtue but I insist of its sake. . . ."[2] In the 20th century, the prominent Harvard physician Henry K. Beecher argued in the pages of the *New England Journal of Medicine* that "an intelligent, conscientious, compassionate, responsible investigator" provided the best protection for participants.[3] Beecher considered these qualities more important even than informed consent; new regulations were not even considered as contenders. In the late 20th century, Edmund Pellegrino, who later chaired President George W. Bush's President's Council on Bioethics, systematically adapted Aristotelian virtue theory to the practice of medicine. In essence, the virtues of medicine are the characteristics of physicians that help them to achieve the goal—the *telos*—of their practice: healing, prevention, and palliation.[4]

Note that these goals are all patient-centered. Although it may be legitimate to earn a living or to educate the next generation of physicians, the primary goals of medicine are healing, prevention, and palliation. Thus, one of the specific virtues that Pellegrino identified is *fidelity to trust*: faithfully protecting the patients' trust that physicians will act in patients' best interests.[5] This is what the courts describe as acting within a fiduciary relationship.

Given this longstanding focus on virtues in medicine, the Institute of Medicine issued a report in 2009 on "Conflicts of Interest in Medical Research, Education, and Practice." It offered a series of recommendations that would limit the kinds of relationships physicians have and increase oversight of medical practice. However, it omitted entirely any reference to the virtue or integrity of physicians. How did this happen?

Cases and Data That Led to the Ethic of Oversight

The shift to increased oversight of human medicine appears to be due to two major factors: high profile cases of physician wrongdoing that involved financial conflicts of interest and growing awareness of a body of social science literature on conflicts of interest.

Cases

Following are two cases of physician conflicts of interest that hit the press during the past decade.

Case 1

Dr. Gleason was charged with promoting a drug for purposes not approved by the federal government. Gleason promoted Xyrem as a drug for depression and pain relief at hundreds of speeches and seminars. (The Food and Drug Administration had approved Xyrem only for the treatment of narcolepsy.) Jazz Pharmaceuticals paid Dr. Gleason generously for these services—more than $100,000 in 1 year. Gleason told audiences that "table salt is more dangerous" than Xyrem. However, the active ingredient in Xyrem is gamma-hydroxybutyric acid (GHB), a fast-acting central nervous system depressant that can suppress breathing and cause coma or death during an overdose. It was designated as a Schedule 1 controlled substance after highly publicized cases in 2000 in which women died or were raped after GHB was slipped into their drinks.[6]

Case 2

Dr. Chan, a neurosurgeon, earned $200,000 a month and amassed $10 million performing spinal fusions. He was charged by the Federal Bureau of Investigation with demanding and receiving thousands of dollars in kickbacks from medical-device manufacturers. A Medtronic hardware representative accused Dr. Chan of switching to a different supplier after the new company agreed to offer him cash kickbacks and alleged that Dr. Chan did many unnecessary surgeries simply for profit. One of several malpractice cases against Dr. Chan involved fusion surgery on a man in his 80s who had only weeks to live with terminal cancer and who would not have benefited from the procedures.[7,8]

These cases were part of a string of highly publicized cases involving unnecessary procedures, fraud, abuse,[9–12] and medical research biased by significant financing from for-profit companies.[13] Spurred by these cases, some people suspected that they were the tip of the iceberg and began studying systematically physicians' relationships to industry.

Social Science Data

Several review articles have found that financial relationships between physicians and the drug and device industries are ubiquitous[14,15] and have measurable effects on physician diagnosis and treatment patterns[16] as well as the design, results, and publication of biomedical research.[17–19] Additionally, when physicians own the companies that provide services, they use these services more often than do other physicians.[20]

At the same time, most physicians insist that their practice of medicine is not affected by such financial relationships, even though they believe that their peers' behavior is negatively affected.[21] How is this possible? Perhaps the answer lies in the social science research that was reviewed by the Association of American Medical Colleges (AAMC) and the Institute of Medicine (IOM).[22,23] An appendix to the IOM report entitled "How psychological research can inform policies dealing with conflicts of interest in medicine" concluded the following:

> Research shows that when individuals stand to gain by reaching a particular conclusion, they

> tend to unconsciously and unintentionally weigh evidence in a biased fashion that favors that conclusion. Furthermore, the process of weighing evidence can occur beneath the individual's level of awareness, such that a biased individual will sincerely claim objectivity.[24]

The last point is particularly important. It helps to explain how physicians may think they are prioritizing their patients' well-being even as financial factors bias their decisions.

Life in an Era of Oversight and Suspicion

Despite calls for professional self-regulation, physicians have been unable to preempt outside restrictions.[25] Regulation in the areas of physician self-referrals and referrals for goods and services has come in the form of federal anti-kickback and Stark laws (see Table 1). Senator Charles Grassley (R-Iowa) became a watchdog for the US government in cleaning up federally funded research. His call for investigations and audits at medical practices, hospitals, and academic institutions kept the issue at the forefront of the news and prompted the codification of the Physician Payment Sunshine Act as well as a revamping of the National Institutes of Health (NIH) conflict of interest guidelines.[26–29]

To inspire confidence and show that physicians could in fact self-regulate, numerous medical institutions,[30] medical journals,[31] and medical organizations, including the AAMC, the IOM, and even the Pharmaceutical Manufacturers of America, released various reports and guidelines on the interaction of physicians with for-profit industries.[24,32–36] Unfortunately, the imposed regulations are often vague, arbitrary, and burdensome, provoking physician discontent and the perception of slowing the progress of science.[37,38]

Who's Complaining?

Ironically, patients are not the ones who have complained and insisted on tighter regulations in medicine. When confronted with conflict-of-interest scenarios, most patients believe that physicians in general would be biased by financial conflicts of interest—but not their own physician.[39] At the same time that medicine has grown more distrusted in regulatory circles, the public has expressed growing trust: In 1976, medical doctors saw 56% of Gallup poll respondents rate their honesty and ethical standards as high or very high; but this percentage has steadily climbed over the past two decades, reaching 70% in 2012.[40] (As an aside, the 2012 rating of physicians' ethics is 7 times higher than the ratings received by the members of Congress, who increasingly oversee the practice of medicine.)

2. Conclusions

Veterinary medicine is not immune to problems with conflicts of interest. I am not an expert in this arena, but, after conducting the briefest of literature reviews, my co-author and I identified several stories addressing business practices in veterinary medicine. For example, veterinarians are becoming targets in the controversy surrounding the use of prescription drugs in racehorses and the clear financial incentives to keep unhealthy horses racing.[41–43] Politicians such as Governor Cuomo of New York,[43] groups such as the Jockey Club,[44] and individual veterinary professionals[45] are calling for investigations and regulations to decrease apparent conflicts of interest and prescription violations by equine veterinarians at America's racetracks.[44,46,47]

Although we could be mistaken, we have the impression that veterinary medicine is not as heavily regulated as human medicine. The American Association of Equine Practitioners' "Ethical and Professional Guidelines" provide excellent guidance to equine veterinarians, just as the American Medical Association's "Code of Medical Ethics" has since 1847. In this regard, veterinary medicine seems to be in a position roughly analogous to the position of medicine in the 1990s and early 2000s.

We speak from outside the field of equine veterinary medicine and thus are not in a good position to assess working conditions or to make predictions about the field. Nevertheless, we will offer a few tentative conclusions from these brief reflections.

(1) Returning to the leitmotif adapted from Dickens, the *Ghost of Medicine Present* may be the *Ghost of Veterinary Medicine Yet to Come*: If this is true—if veterinary practice is currently less regulated than medicine and coming under increasing scrutiny—then we would warn: Provide ethical leadership and self-regulate or else others will do it for you (and probably very badly).

(2) One's clients are not a good gauge of whether external oversight is forthcoming. As noted above, patient trust appeared to be strong and growing at the very time when Congress and regulatory bodies increased oversight.

(3) Because individuals are generally unaware of how self-serving biases affect their judgments and behaviors, "self-regulation" cannot occur only at the individual level; professionals must police professionals. It is not enough for individuals to try very hard to behave well. To avoid the fate of medicine, professional associations of veterinarians may need to create and enforce reasonable rules.

(4) Good business ethics often build on good clinical practice. In a fee-for-service system, practitioners generally earn more by providing more procedures. Meeting inappropriate patient demands for treatment—or client demands in veterinary medicine—can be profitable. Yet, many scandals in medical business ethics have arisen from providing unnecessary or inappropriate treatments—

Table 1. Sample of Laws Regulating Business Practices and Relationships in Medicine

Rule	Applies to	Defined as	Penalties for Noncompliance
NIH Conflict of Interest Rules, 42 Code of Federal Regulations (CFR)[49]	All applicants for or recipients of Public Health Service (PHS) funding	Responsibilities of institutions regarding investigator financial conflicts of interest. Information is collected and managed by the institution and provided to the public only when requested. Reports must be given to the PHS.	Depends on the situation. Remedies could include fines and suspension of funding, which can end a researcher's career.
Physician Payments Sunshine provisions of the Patient Protection Affordable Care Act[29]	All US drug and medical device manufacturers covered under Medicare, Medicaid, or State Children's Health Insurance Program (SCHIP)	A federal database of all payments and transfers of value made to physicians and teaching hospitals. The Department of Health and Human Services will post information received on a publicly available, searchable on-line database starting September 30, 2013. Databases will be updated on June 30 annually thereafter.	Fines of up to $10,000 will be charged for each failure to report, not exceeding $150,000 annually.
Anti-kickback laws[50]	All physicians practicing in the United States	Prohibits paying, soliciting, or receiving any remuneration (kickback, bribe rebate) in return for referring an individual to a person for the furnishing or arranging for the furnishing of any item or service for which payment may be made under Medicare or a State healthcare program.	Felony conviction results in a fine of not more than $25,000 and imprisonment for not more than 5 years, or both.
Stark Laws[51]	Federal law	Govern physician self-referral for Medicare and Medicaid patients. Prohibits physicians from referring Medicare patients for certain designated health services to an entity with which the physician or a member of the physician's immediate family has a financial relationship, unless an exception applies.	Denial of payment or refund of monies received, payment of civil penalties of up to $15,000 for each service provided in violation of the law, and 3 times the amount of improper payment received from the Medicare program, exclusion from the Medicare program and other State programs, and payment of civil penalties for attempting to circumvent the law of up to $100,000 for each circumvention scheme.
False Claims Act[52]	Federal law pertaining to those who bill for medical services	Any person who knowingly submits false claims to the government, causes another to submit a false claim to the government, or knowingly makes a false record or statement to get a false claim paid by the government is liable for damages. Includes a qui tam provision that allows people who are not affiliated with the government to file actions on behalf of the government (whistleblowing).	One who is liable must pay a civil penalty of between $5500 and $11,000 for each false claim and 3 times the amount of the government's damages.

that is, treatments that are neither the standard of care nor based on evidence. As a general rule, the right of patients and parents to decline treatment has few exceptions; however, patients generally have no right to demand treatments that a physician considers contrary to good clinical practice.[48] For example, a competent patient may choose to decline antibiotics to treat strep throat, but a physician has no obligation to prescribe antibiotics to treat a viral infection. I imagine that similar rules of thumb are useful in guiding clinical practice in veterinary medicine.

(5) The first step to sensible rule-making is to clarify to whom your primary fiduciary obligation exists. Veterinarians are in a tough position, analogous to the position of pediatricians. The person communicating with you and paying bills is your client but not your patient. Your patients do not sue; your clients can. Your patients will not go to another veterinarian; your clients can. These are facts that can shift allegiances away from your patients.

In discussions of ethics, there are rarely easy answers, but I do believe that deep and realistic consideration of the tensions between the interests of the provider, client, and patient is the starting point for any sound business ethic within veterinary medicine.

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How We Select, Prepare, and Maintain a Stimulus/Mount Mare

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A stimulus/mount mare that is an easy keeper, sound, and reliably attractive and receptive to most stallions, as well as comfortable with her work and easy to handle, is invaluable to a breeding facility. Organized and systematic behavior evaluation specific to your clinic's protocols before selection will reveal undesirable behaviors that may prove unsafe in the semen collection environment or may complicate or prevent successful semen collection. In the case of intact mares, bilateral ovariectomy performed through colpotomy or a hand-assisted laparoscopic approach with colpotomy allows for a short convalescent period compared with other approaches. Additionally, flank sensitivity can be avoided by colpotomy. In the absence of functional ovaries, frequent, low-dose estrogen supplementation can usually maintain estrus adequate for a stimulus/mount mare. Authors' address: School of Veterinary Medicine, University of Pennsylvania, New Bolton Center, 382 West Street Road, Kennett Square, PA 19348; e-mail: kmwhite@vet.upenn.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Although some stallions, initially or after training, respond adequately for semen collection in the absence of a live mare, most semen collection facilities do require a live mare to accommodate stallions that do not respond. A cycling mare in natural estrus close to ovulation is typically the most stimulating to stallions. However, to ensure availability of a suitable mare in estrus on any given day throughout the year, a fairly substantial herd of monitored/cycle manipulated mares from which to choose is typically required. It has long been known that in the absence of ovarian steroid hormones, whether during anestrus, in certain karyotipic abnormal states (XO), in senescence, or after ovariectomy, mares can show estrus sufficient to stimulate response of most stallions for semen collection as well as tolerate mounting for semen collection or schooling for natural cover. Accordingly, a commonly used alternative to a cycling mare in estrus is a mare without functional ovaries that is administered exogenous estrogen as needed to maintain estrus at a level sufficient for the needs of the facility.

In a quantitative study of the behavior of 10 ovariectomized and 10 seasonally anestrus mares, Asa et al[1] reported that 17 of the 20 mares showed some degree of receptivity (tolerance of the stallion's approach, teasing, mounting, insertion) on most test days. The probability of any particular mare exhibiting estrus on any test day in their study was reported to be 50%. The intensity of estrus observed was comparable to that of mares transitioning between estrus and diestrus phases, which is less intense than is typical of the day before and the day of ovulation. This study also quantitatively confirmed the long-time anecdotal observations of wide

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variation in the degree of proceptivity (solicitousness), receptivity (tolerance of mounting), and attractivity (stallion sexual response) of mares without ovarian steroid influence, both among mares and from day to day within-mare. Only one of the 20 mares was observed to exhibit estrus on all 15 observation days of the study. In a follow-up study of steroid hormone treatment of ovariectomized mares, it was demonstrated that estrogen treatment increased the intensity and decreased variation in estrus as well as correspondingly increased the intensity and decreased the variation in stallion response.[2] Estrogen-treated mares showed strong estrus for 80% of the tests and weak estrus for an additional 3%. Untreated mares showed strong estrus in only 23% of tests and weak estrus for an additional 33%.

This report presents our experience with selection of candidates for stimulus/mount mares as well as preparation and estrogen treatment of ovariectomized mares for comfortable, effective, and safe use in an equine reproduction and behavior teaching clinical and research facility with a variety of unknown and known stallions of various breeds. These circumstances necessitate year-round availability of reliable stimulus mares that are also compliant with mounting for semen collection, novice stallion schooling, and a variety of other reproduction teaching exercises involving rotating staff and students of various levels of handling ability and horse breeding experience.

2. Candidate Selection

Initial Considerations

When considering particular mares as candidates for preparation to be a stimulus/mount mare, we first try to identify candidates with (1) a behavioral history of excellent temperament and interaction with humans and other horses, both for general ground handling as well as when interacting with a stallion for estrus detection or breeding, and (2) generally good health, soundness, and ease of maintaining good body and particularly foot condition. We also consider the age of the mare because there is considerable effort and expense to preparation, and we prefer to expect as many years of service as possible. Although there are no data to confirm the concepts, we consider the tendency for (1) demonstrative estrus for several days each cycle and (2) less frequent and demonstrative aggressive responses to stallions during diestrus to be predictive of better performance and comfort as a stimulus/mount mare. If this history is known, we certainly take it into consideration. Also, because of our experience with apparent color, breed, and size preferences and aversions among stallions, we aim to maintain a variety of these characteristics among our available stimulus/mount mares.

Systematic Behavior Evaluation

If a mare meets those initial behavior and soundness selection criteria, we then systematically evaluate her behavioral comfort and compliance with a battery of anticipated breeding shed protocols for semen collection, breeding, and schooling of stallions specific to the environment in which she will be used. Winter anestrus presents an ideal opportunity for this evaluation because it most closely approximates the ovariectomized condition. However, for mares that are not anestrus, we of course evaluate the specific breeding shed tasks when she is in estrus.

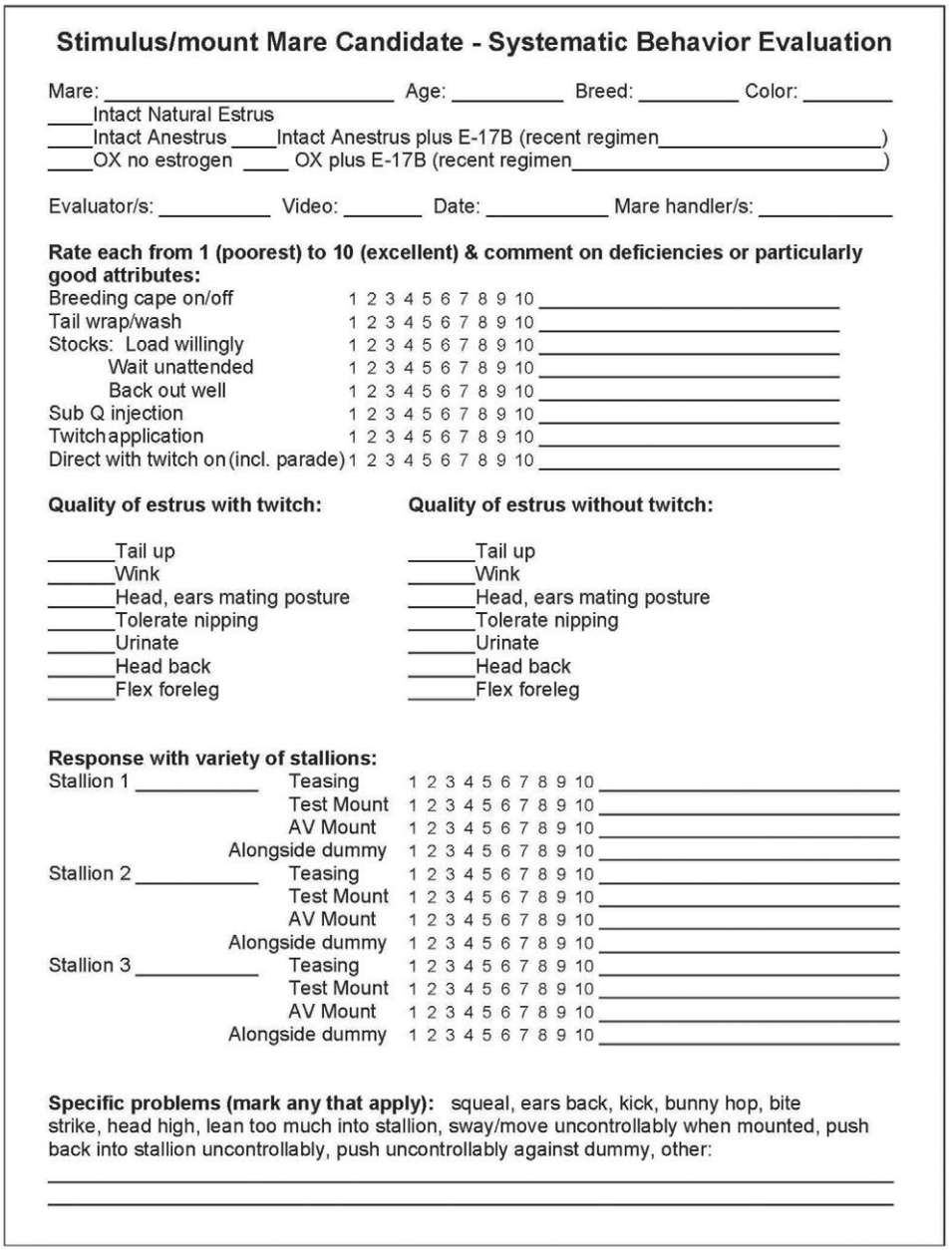
Comfort and compliance with tasks specific to the stimulus/mount mare occupation in our teaching and clinical environment include tolerance and comfort with application of a twitch, breeding cape, hobbles, breeding boots, and tail wrap; expression of proceptive and receptive responses as well as attractivity to stallions both with and without a twitch; easy "steerability" with and without a twitch; ease and comfort with loading, standing for long periods, and unloading in stocks or adjacent the dummy mount; absence of tendency for non-receptive or discomfort responses (striking, squealing, biting, kicking, striking, "bunny hopping," rearing, slapping or swishing tail, moving away, pinning ears, tensing up, cowering) even with prolonged teasing; tolerance of mounting by a test stallion wearing an "apron" (for teaching purposes). We aim to evaluate the stallion and mare response with stallions of various ages, experience, breeds, and breeding temperaments. Because we are a teaching facility with day-to-day variation in the handling team, we also try to assess the candidate's cooperation and comfort with handlers of various skill levels and handling styles. A particular concern is to identify behavior that would pose safety threats for less experienced student handlers in a teaching environment. Fig. 1 illustrates an example check sheet that we use to record test session findings. Fig. 2 includes a series of images from a candidate evaluation session.

3. Preparation

The goal of a dedicated stimulus mare is to remove the limitations of cyclicity and seasonality encountered when selecting mares to aid in semen collection. This can be achieved through ovariectomy and subsequent estrogen replacement or estrogen replacement alone for mares with non-functional ovaries.

Ovariectomy

Various approaches for ovariectomy in the mare include colpotomy, flank laparotomy, paramedian celiotomy (oblique or caudal), ventral midline celiotomy, and various laparoscopic techniques, either conventional or hand-assisted as summarized by Loesch and Rodgerson.[3] A complete discussion of surgical technique is beyond the scope of this report;



Stimulus/mount Mare Candidate - Systematic Behavior Evaluation

Mare: _____ Age: _____ Breed: _____ Color: _____
____Intact Natural Estrus
____Intact Anestrus ____Intact Anestrus plus E-17B (recent regimen_____)
____OX no estrogen ____ OX plus E-17B (recent regimen_____)

Evaluator/s: _____ Video: _____ Date: _____ Mare handler/s: _____

Rate each from 1 (poorest) to 10 (excellent) & comment on deficiencies or particularly good attributes:

Breeding cape on/off	1 2 3 4 5 6 7 8 9 10	_____
Tail wrap/wash	1 2 3 4 5 6 7 8 9 10	_____
Stocks: Load willingly	1 2 3 4 5 6 7 8 9 10	_____
Wait unattended	1 2 3 4 5 6 7 8 9 10	_____
Back out well	1 2 3 4 5 6 7 8 9 10	_____
Sub Q injection	1 2 3 4 5 6 7 8 9 10	_____
Twitch application	1 2 3 4 5 6 7 8 9 10	_____
Direct with twitch on (incl. parade)	1 2 3 4 5 6 7 8 9 10	_____

Quality of estrus with twitch:

_____Tail up
_____Wink
_____Head, ears mating posture
_____Tolerate nipping
_____Urinate
_____Head back
_____Flex foreleg

Quality of estrus without twitch:

_____Tail up
_____Wink
_____Head, ears mating posture
_____Tolerate nipping
_____Urinate
_____Head back
_____Flex foreleg

Response with variety of stallions:

Stallion 1 _____	Teasing	1 2 3 4 5 6 7 8 9 10	_____
	Test Mount	1 2 3 4 5 6 7 8 9 10	_____
	AV Mount	1 2 3 4 5 6 7 8 9 10	_____
	Alongside dummy	1 2 3 4 5 6 7 8 9 10	_____
Stallion 2 _____	Teasing	1 2 3 4 5 6 7 8 9 10	_____
	Test Mount	1 2 3 4 5 6 7 8 9 10	_____
	AV Mount	1 2 3 4 5 6 7 8 9 10	_____
	Alongside dummy	1 2 3 4 5 6 7 8 9 10	_____
Stallion 3 _____	Teasing	1 2 3 4 5 6 7 8 9 10	_____
	Test Mount	1 2 3 4 5 6 7 8 9 10	_____
	AV Mount	1 2 3 4 5 6 7 8 9 10	_____
	Alongside dummy	1 2 3 4 5 6 7 8 9 10	_____

Specific problems (mark any that apply): squeal, ears back, kick, bunny hop, bite strike, head high, lean too much into stallion, sway/move uncontrollably when mounted, push back into stallion uncontrollably, push uncontrollably against dummy, other:

_____
_____

Fig. 1. Example check sheet for systematic behavior evaluation of stimulus/mount mare candidates.

however, consideration of the implications of the various approaches for intended use as a stimulus/mount mare is warranted. Flank incisions not only require an extended convalescent period (4 to 6 weeks), but, in our experience, flank incisions often result in an aversion to flank contact that not only complicates mounting by a stallion for semen collection through the use of the artificial vagina or as an intermediate training step but also seems to predispose some mares to be less tolerant of vigorous precopulatory interaction. In contrast, for colpotomy, the convalescent period is only 2 weeks (or in our experience less if necessary), and there is no external scarring.[4] Although a conventional laparoscopic approach carries the advantage of improved visualization of the ovary and mesovarium over the flank approach, extension of portal incisions to facilitate extraction of the ovary is still necessary. Therefore, postoperative healing time, incisional complications, and persistent flank sensitivity are concerns for a stimulus/mount mare. Recently, hand-assisted techniques have been developed to combine the laparoscopic visualization with ovary extraction by means of colpotomy to minimize flank incisional length and consequently, convalescent time. For such techniques, a chain ecraseur or vessel sealing/tissue dividing instrument[a] has been used for transection and hemostasis of the mesovar-



Fig. 2. Systematic evaluation of a stimulus/mount mare candidate: The mare demonstrated difficulty accepting twitch application, repeatedly escaping the twitch even with two skilled handlers (A); initially appeared fearful of the breeding cape but later acclimated (B); loaded easily into stocks (C); tolerated subcutaneous injection (D), tail wrap (E), and perineal washing (F); appeared comfortable standing unattended in stocks for approximately 5 minutes (G), after which she became fidgety and appeared anxious; showed estrus to a stallion initially (H); however, when the stallion was allowed more vigorous close contact, she became less tolerant (I), after which she appeared to panic and attempted to escape.

ium.[4–6] The use of this combined approach removes inherent risk of a traditionally blind colpotomy approach while minimizing concerns associated with flank incisions. However, other potential complications from colpotomy, including peritonitis, eventration, intra-abdominal adhesions to the vagina, and damage to the cervix, bladder, or bowel, are not circumvented.

Estrogen Treatment

Anecdotally, there seems to be wide variation in estrogen treatment protocols used to maintain adequate estrus in stimulus/mount mares. Ginther[7] discussed heightened sensitivity of anestrus mares to low doses of estrogens, citing early work done by Nishikawa demonstrating a minimum effective dose of estrone for induction of estrus to be 50 mg. Estrus was seen as early as 4 hours and no later than 10 hours after administration, and the duration of estrus ranged from 3 to 10 days. Ginther also discussed estrogen supplementation in ovariectomized mares, citing work done by Hillman administering 0.5 mg or 5 mg of estradiol to mares that resulted in interest to a stallion within 3 hours and maximal estrus response in 9 hours. Mares subsequently returned to pretreatment behavior in 48 hours for the 0.5-mg dose and 96 hours for the 5-mg dose. Daily estradiol treatment at 1 mg/day resulted in positive estrus signs for the duration of administration. Of the two forms of estrogen with which we have extensive experience with ovariectomized mares (estradiol cypionate and estradiol 17-β), we have found estradiol 17-beta in oil[b] to typically result in more reliable, consistent, and full complement of estrus responses that more reliably elicits positive stallion response. On the basis of early success in maintaining good response for mares used in our research program, the estrogen treatment regimen that our clinical facility has adopted for use with average size light horse stimulus/mount mares, whether ovariectomized or with dysfunctional ovaries, is 0.5 mg estradiol 17-β administered subcutaneously every other day for the first three doses followed by 0.25 mg administered subcutaneously every other day for maintenance. It is our experience that the majority of mares (estimated three of four) maintain a strong estrus response at this level fairly long term (several weeks to many months, in some cases). Certain larger mares may do better with slightly higher doses. We have found that less frequent treatment with higher dosage typically results in less consistent estrus and may result in stallion-like behavior. We have also found that in the case of a mare that initially performs well with the low, frequent dose schedule but then becomes increasingly difficult to work with or off-putting to the stallion, a return to a better response can typically be achieved by discontinuing treatment for a brief period. As little as a few days to 1 week off before return to estrogen treatment has been effective. After the break, we resume treatment with the initial three 0.50-mg doses. Some mares as individuals may benefit from time off treatment (and work). We have also found that for rare instances, when it is judged that additional estrogen may be helpful on short notice, estrogen can be applied intravaginally, with what appears to be positive effects within 2 to 4 hours. Hyperemia of the vaginal mucosa generally follows intravaginal topical application, which typically resolves within a few days at most. Nonetheless, because of this untoward side effect that could be counterproductive to receptivity, we reserve the vaginal topical route of administration for rare situations requiring a rapid improvement in response.

Experience/Training

Despite careful selection and preparation of candidates, not all meet the challenges of our facility. In our experience, stimulus/mount mares can either become more or less tolerant and comfortable with time of service. Some appear to improve with positive experience. Some can become less comfortable and willing, possibly because of frequency and intensity of use, temperament of particular stallions, and experience with handling styles.

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[a]Ligasure, Covidien Surgical Solutions Group, Boulder, CO 80301.

[b]Estradiol 17-β, 3.33 mg/mL, Hagyard Pharmacy, Lexington, KY 40511.

How to Manage Cervical Incompetence by Application of a Cervical Cerclage Suture in the Pregnant Mare

Stefania Bucca, DVM

Placement of a cerclage suture over the caudal os of the cervix can effectively restore cervical closure in pregnant mares at risk of anatomical or functional incompetence, unresponsive to progestagen supplementation. Author's address: Qatar Racing and Equestrian Club, Muaither Rayyan, PO Box 7559, Doha, Qatar; e-mail: stefbucca@gmail.com. © 2013 AAEP.

1. Introduction

Cervical incompetence in the mare is widely recognized as a major cause of pregnancy failure. Failure to attain adequate closure during pregnancy is a key predisposing factor to ascending placentitis. In humans, cervical insufficiency is of major concern[1] and can result in preterm birth and late-term abortions, similar to observations in mares. Emerging clinical[2,3] and laboratory-based evidence[4] suggests that focusing on the uterine cervix may provide significant elements of diagnostic value to identify patients at risk for preterm delivery, offering guidelines to implement preventive intervention.[5] A variety of techniques have been used in human medicine to address cervical incompetence, including medical therapy with progesterone, cervical cerclage, and the placement of a cervical pessary,[6–8] some of which may be applicable in the mare. A recent study in human medicine compared vaginal progesterone versus cerclage, with the use of placebo/no cerclage as the control treatment.[9] The authors concluded that progesterone and cervical cerclage are equally effective for the prevention of preterm birth and adverse perinatal outcomes in patients with a short cervix and history of preterm birth.

A common treatment strategy used on mares that have previously lost a foal because of placentitis or that have anatomical abnormalities in the caudal reproductive tract making them at risk of ascending placentitis is to administer an antimicrobial, a non-steroidal anti-inflammatory drug and altrenogest daily until delivery of the foal.[10] The shortcoming of this treatment is that mares may still have a preterm delivery/fetal loss with histopathological evidence of ascending placentitis despite treatment. This report describes a technique for cervical cerclage applied to a population of mares with known cervical incompetence.

2. Materials and Methods

Six cervical cerclage procedures were carried out on four mares. Two mares underwent the cerclage procedure during two successive pregnancies. Three of the four mares were aged Thoroughbreds and the fourth was a 9-year-old Arabian mare. Cervical incompetence was detected through serial

NOTES

Table 1. Historical and Gestational Parameters of Six Cervical Cerclage Procedures

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Age, years	17	18	19	21	14	9
History of pregnancy loss	2 years	N/A	3 years	N/A	1 year	None
Gestation at initial diagnosis, days	220	205	170	154	218	213
Gestation at cerclage suture, days	234	226	191	183	240	226
Gestation at parturition, days	339	344	362	371	348	328

ultrasound examinations.[11] The gestational age at the time of diagnosis varied between mares but ranged from 5 to 8 months of gestation.

Case 1, a 17-year-old Thoroughbred mare, presented a 2-year history of abortion and a histopathological diagnosis of ascending placentitis despite weekly administrations of altrenogest and antimicrobials, each month of gestation. Case 1 was presented at 220 days of gestation for investigation of her history of pregnancy loss. Premature cervical ripening was evident on ultrasound examination, and a cerclage suture was placed at 234 days of gestation, after 2 weeks of increased altrenogest administration (of 0.088 mg/kg twice daily) with no improvement in ultrasound cervical parameters.

Case 2, the same mare as in case 1, the year after her first cervical cerclage treatment, was monitored for ultrasound evidence of cervical ripening from day 120 of gestation. Signs of cervical ripening were detected 205 days after ovulation, with cervical parameters progressing from grade 3 to grade 4[11] despite altrenogest administration. Cervical cerclage was then carried out.

Case 3, a 19-year-old Thoroughbred mare, had a 3-year history of pregnancy loss and histopathological evidence of ascending placentitis despite altrenogest/antimicrobial treatment administration. Signs of cervical ripening were detected at day 170 of gestation, and a cervical cerclage suture was placed 21 days later.

Case 4, same mare as in case 3, the year after her first cervical cerclage treatment, showed evidence of cervical ripening at day 154 of gestation and was treated with cervical cerclage after 3 weeks of altrenogest administration, with no improvement in cervical parameters.

Case 5 was initially presented for intermittent vaginal bleeding at approximately 7 months of gestation. The source of bleeding was readily identified in some vestibular varicosities and promptly corrected, but ultrasonography of the caudal reproductive tract showed evidence of premature cervical ripening. Case 5 had already been placed on altrenogest and antimicrobial treatment by the referring veterinarian because of the mare's history of abortion caused by ascending placentitis the previous year. After cervical assessment with evidence of premature cervical relaxation, treatment with altrenogest continued at an increased dose of 0.088 mg/kg twice daily for 3 weeks. A cervical cerclage suture was placed at day 240 of gestation because of the persisting state of cervical relaxation and the significant increase in the combined thickness of the utero-placental unit (CTUP) at the cervical pole (11 mm).

Case 6, a 9-year-old Arabian mare at her second gestation, was referred for mild signs of early mammary development at 7 months of gestation. The mare had a history of stillbirth and dystocia the previous year. She had been bred naturally very late that year and was not checked for pregnancy afterward.

No abnormalities were detected on clinical examination of the mare, but, on rectal palpation, the cervix was short and fibrotic. Ultrasonography of the caudal reproductive tract per rectum revealed a short (<6 cm) cervical canal with disrupted central linear striation and a heterogeneous echo pattern of the muscular layer. An area of increased CTUP (12.4 mm), markedly hypoechoic, was identified at the cervical pole, extending rostrally to the caudal body of the uterus for 6 to 8 cm. Transabdominal feto-placental evaluation was unremarkable. Ascending placentitis associated with cervical incompetence was suspected and was probably related to cervical damage received during dystocia correction the previous year. A cervical cerclage suture was placed 13 days after initial diagnosis.

Table 1 summarizes historical and gestational parameters relevant to the procedure.

Technique

The application of a purse-string suture over the vaginal os of the cervix was carried out on the sedated mare that was restrained in stocks. The mare's rectum was evacuated and the tail wrapped and tied to one side. The perineum was surgically scrubbed and dried. Sedation was administered before the procedure (detomidine HCL[a] 0.02 mg/kg in association with butorphanol tartrate[b] 0.04 mg/kg). The mare's cervix was visualized with the use of a modified Finochietto retractor (Fig. 1). The cervix was then grasped with long-handled Knowles forceps (Fig. 2). A purse-string suture was placed with the use of long-handled instruments and a single-stranded No. 2 nylon suture (Fig. 3). Three partial-thickness bites (avoiding penetration of the cervical lumen), approximately 1.5 to 2 cm long, were applied to the circumference of the external cervical os, starting dorsally at the

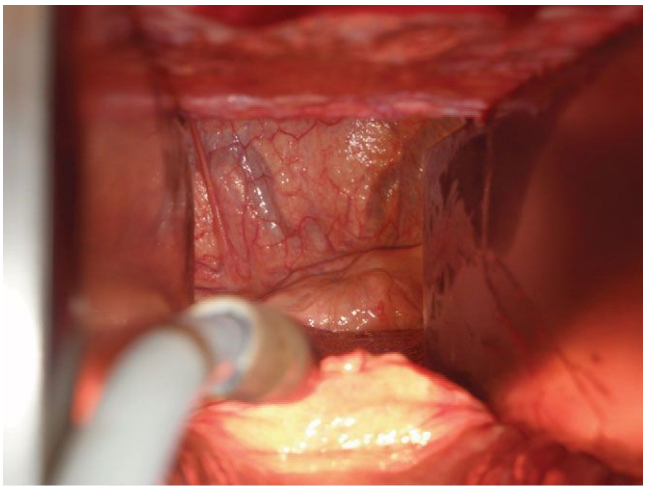


Fig. 1. The cervix is visualized through the use of modified Finochietto retractors.



Fig. 3. Placement of a purse-string suture over the vaginal os of the cervix, with the use of long-handled instruments and single-stranded No. 2 nylon suture.

2 o'clock position to end at the 11 o'clock position (Fig. 4). The length of the bites varied according to the state of relaxation of the cervical os and therefore its size. The knot was electively tied in an upper position (11 to 1 o'clock positions) to facilitate removal at the appropriate time.

Antimicrobials, non-steroidal anti-inflammatory drugs, and altrenogest were administered perioperatively and continued for about 5 days after intervention. After cerclage placement, treated mares were kept under close observation for signs of impending parturition, while cervical parameters and cerclage suture were monitored by transrectal ultrasonography. The cerclage suture was removed at the mare's due date or in the presence of early mammary development. Suture removal was performed in the standing sedated mare, restrained in stocks, with the use of long-handled scissors and Finochietto retractors (Figs. 5, 6, and 7).

3. Results

After cervical cerclage, all mares delivered live foals. Foals survived to at least 1 year of age. Serial evaluation of the caudal reproductive tract did not reveal abnormalities (placental changes, cervical shortening) after cerclage sutures were placed. Post-foaling inspection and examination at foal heat were also unremarkable for potential complications of the cerclage procedure.

4. Discussion

Cervical incompetence in the mare is commonly associated with difficulty conceiving or carrying a foal to term and is widely recognized as a major cause of



Fig. 2. The cervix is grasped with the use of long-handled Knowles forceps.

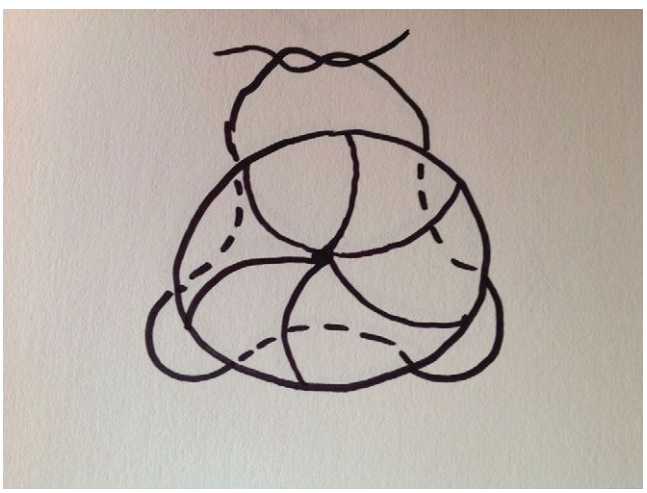


Fig. 4. Schematic drawing of the placement of a cervical cerclage suture.

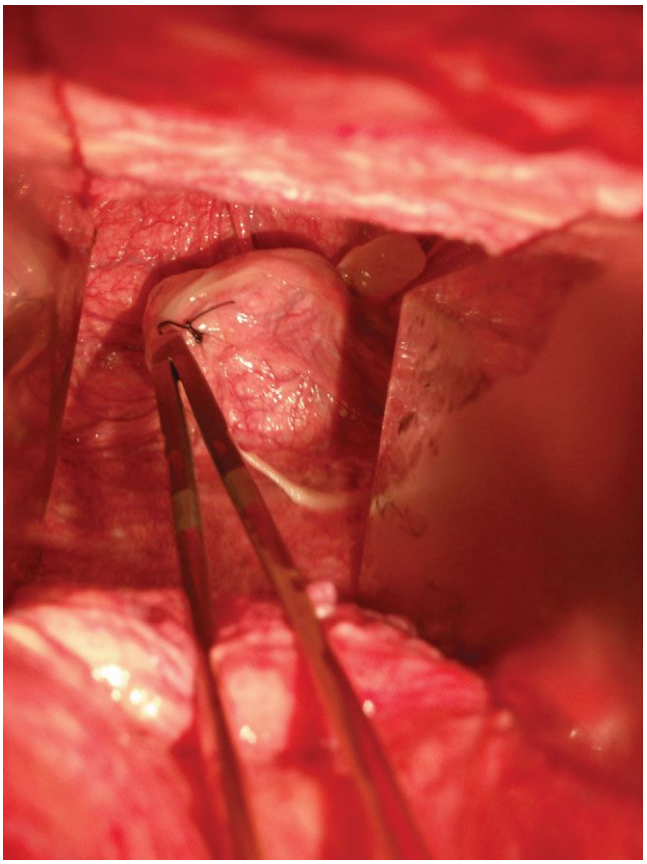


Fig. 5. Cerclage suture removal. The suture is grasped with the use of a long-handled needle holder.

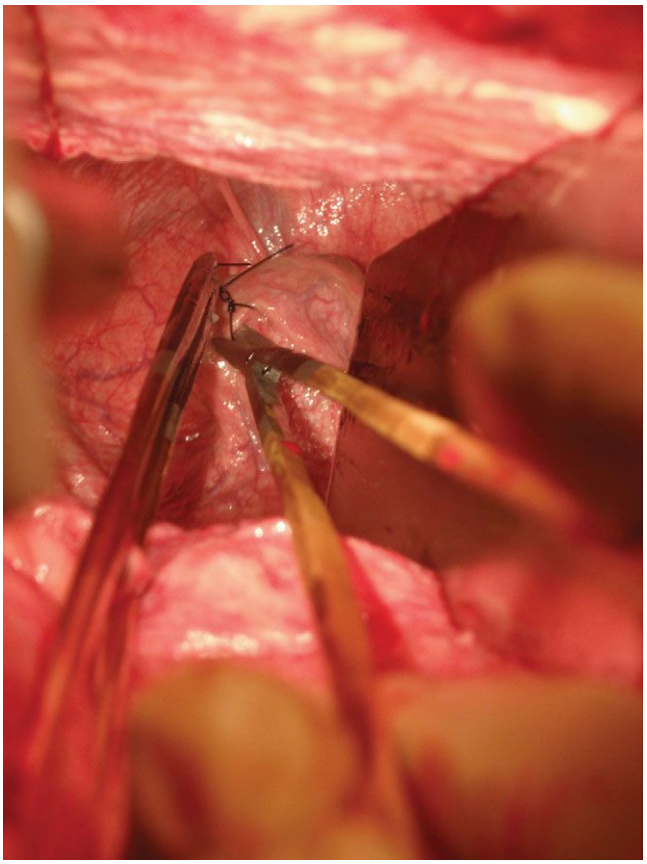


Fig. 6. Cerclage suture removal. The suture is cut with the use of long-handled scissors.

pregnancy failure. Yellon et al[12] have appropriately referred to the cervix as the "gatekeeper for pregnancy." The cervix functions as the final and most cranial barrier to ascending aggressors because it separates the uterine lumen from vaginal content. Failure of the cervix to form a functional barrier will expose the uterine environment to physical, chemical, or biological challenges from the caudal reproductive tract and predispose the mare to chronic endometritis and ascending placentitis. Cervical incompetence recognizes anatomical and functional origins. In humans,[13] it has been reported that remodeling of the cervix occurs slowly over a length of time and that the progressive changes in the cervix precede the uterine contractions by several weeks in normal pregnancy.[14] Similar conclusions may be drawn from the results of a recent study on the ultrasonographic features of the equine pregnant cervix.[11] The study indicates that in the mare, evidence of cervical remodeling leading to ripening may be observed during the last 2 months of gestation, with gradual changes in size and echotexture. Cervical ripening entails changes in cellular structure and molecular biology of the cervical cells,[14] and this gradual process will alter the mechanical attributes of the cervix, with a certain degree of variability among individuals. Premature remodeling/ripening of the cervix and shortening of the cervical canal may be suggestive of preterm birth, on the basis of loss of mechanical support and because the bacterial flora of the caudal genital tract is moved closer to the fetal membranes overlaying the internal os. Cervical ripening causes utero-placental instability at its interface, with resulting areas of detachment, hemorrhage, and inflammatory change, radiating from the cervical pole. Avillous detached areas may prolapse into the inner cervical os, under the mechanical forces of fetal extremities and fetal fluid pressing on it. Large detached areas may create a marked "funneling" effect, in which the free allantochorion may prolapse through the length of the cervical canal and become exposed to microorganisms ascending from the vagina, through the relaxed cervix. Premature cervical ripening may be a transient phenomenon and may spontaneously resolve; it may be observed in pregnant mares recovering from systemic disease and/or post-surgery. Gestation will progress undisturbed to term, providing that the local inflammatory process subsides and microorganism colonization of the allantochorion at the cervical pole is prevented.

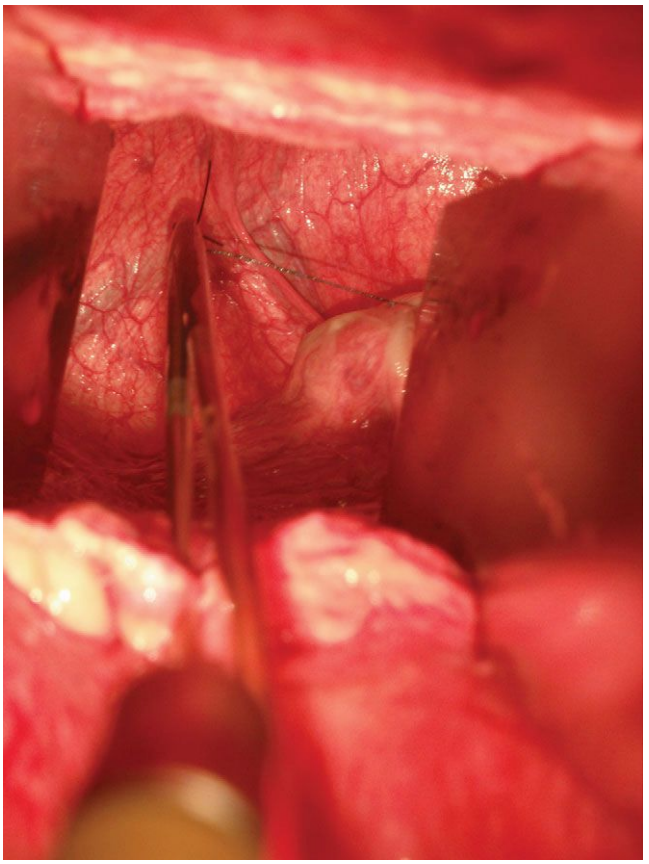


Fig. 7. Cerclage suture removal. The suture is gently pulled out.

Changes of the utero-placental unit at the cervical pole may be detected by transrectal ultrasonography as a consequence of premature cervical ripening in the mare, suggestive of ascending placentitis. The presence of sonographically identifiable collections of material referred to as "amniotic sludge," accumulated above the internal os, represents a correspondent change associated with adverse pregnancy outcome in women who have premature cervical ripening.[15] There is general agreement that some cases of premature cervical ripening are related to infection.[14] In humans, infection may account for up to one-third of preterm births, and the inflammatory response elicited by infectious agents has also become the accepted mechanism for preterm birth. In contrast, other instances associated with premature cervical ripening are attributed to less defined causes such as genetic background of the mother or fetus, environmental factors, intercurrent illness, or health conditions,[14] and this may very well hold true in the pregnant mare.

Progesterone is considered a key hormone for pregnancy maintenance, and a decline in progesterone action is implicated in the onset of parturition. If such decline occurs in the midtrimester in women, cervical shortening may occur, and this would predispose to preterm delivery. It is recognized that progesterone has a demonstrable effect on the rate of cervical shortening and preterm delivery when administered to women admitted for preterm labor between 25 and 33 +6 weeks of gestation.[16] A similar effect may be observed in mares with ultrasonographic evidence of premature cervical ripening during altrenogest supplementation, with significant improvement in cervical grading (author's observation) and gradual palpable increases in cervical tone and length. In the author's experience, failure to respond to altrenogest supplementation is the major indication for cervical cerclage in cervical incompetence cases, particularly when increments in CTUP are observed at the cervical pole. Mares that have chronic medical conditions, endocrine imbalances, and toxic disorders have been observed to show ultrasound evidence of premature cervical relaxation, even under progestagen supplementation (personal communication). Under these circumstances, a cervical cerclage procedure is likely to prevent the development of ascending placentitis.

In the case series presented in this report, cases 1 and 3 showed the inadequacy of progestagen treatment alone in promoting adequate cervical competence, as histopathology of fetal membranes indicated ascending placentitis as the cause of pregnancy failure for the previous two gestations in case 1 and for the 3 preceding years for case 3. Placement of a cervical cerclage suture prevents exposure of the cervical lumen and ultimately of the allantochorion to aggressors from the caudal reproductive tract. It also provides a means of stabilizing and strengthening the cervix from the mechanical forces applied by fetal parts and fetal fluid volumes shifting back and forth during episodes of activity. Penetration of the cervical luminal mucosa during placement of the cerclage suture should be avoided because it may create an intralumenal inflammatory focus and the opportunity for bacterial colonization from the caudal reproductive tract. Particularly during episodes of cervical funneling, the allantochorion may advance toward the vaginal os of the cervix, with ample opportunity for bacterial contamination.

5. Conclusions

Focus on the uterine cervix may identify mares at risk for ascending placentitis/preterm delivery and may yield approaches to prevent it. Ultrasonographic monitoring of cervical parameters can be easily applied under field conditions and should be implemented in mares with a repeated history of preterm delivery and evidence of ascending placentitis, starting as early as 4 months of gestation. Placement of a cervical cerclage suture is warranted when progestagen supplementation fails to achieve adequate cervical closure and significant changes of the utero-placental unit take place at the cervical pole. Although no complications have been reported in association with cervical cerclage, close supervision of treated mares for signs of impending

parturition is of critical importance to prevent cervical damage in the case of untimely delivery with the suture still in place.

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[a]Dormosedan® Orion Corporation, Espoo, Finland.
[b]Torbugesic®, Fort Dodge Animal Health, Fort Dodge, IA 50501.

How to Manage Hydrops Allantois/Hydrops Amnion in a Mare

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1. Introduction

Hydrops conditions of the placenta in the mare are uncommon, with hydrops allantois being reported more frequently than hydrops amnion.[1] The condition in the mare usually develops during the last trimester of pregnancy and is characterized by the excessive accumulation of allantoic (hydrops allantois) or amnionic fluid (hydrops amnion). The sequelae of unnoticed or untreated hydrops allantois/amnion can be significant, including abdominal wall hernias, prepubic tendon rupture, and cardiovascular shock associated with unattended foaling and dystocia[2–4] The condition is often detected by the horse owner as a sudden onset of abdominal enlargement (over a period of a few weeks), ventral edema, varying degrees of colic, lethargy, anorexia, tachycardia, and, on occasion, dyspnea associated with intra-abdominal hypertension (Fig. 1). Differential diagnoses include twins, other causes of colic, and causes of ventral edema.



Fig. 1. Mare with hydrops allantois. Note the pendulous "pear-shaped" abdomen.

Definitive diagnosis requires a trans-rectal examination of the reproductive tract and transabdominal sonogram to detect abnormally abundant fetal fluids. The allantoic fluid volume in mares with hydrops allantois can range from 110 to 230 L, whereas a normal volume at term ranges from 8 to 18 L.[5] Rectal examination will reveal a large, fluid-filled uterus of varying tightness with the dorsal

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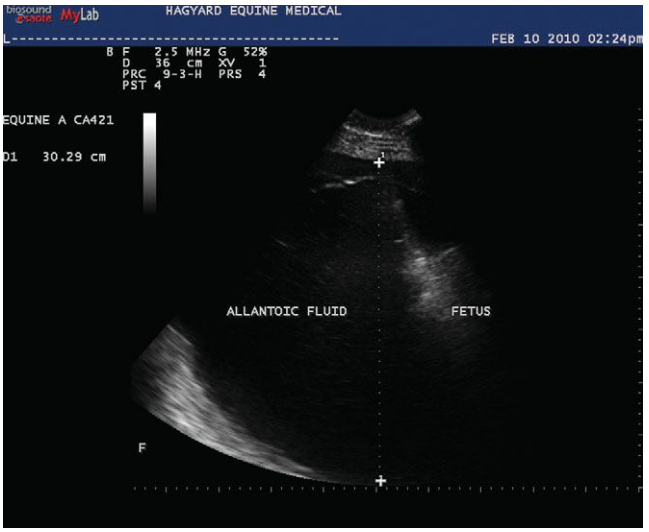


Fig. 2. Sonographic image of a hydrops allantois. Note the excessive allantoic fluid with a depth of 30.29 cm (normal allantoic depth : 4.7–22.1 cm).



Fig. 4. Several 5-gallon buckets used to quantitate the volume of allantoic fluid drained during the procedure. Quantitating the amount of fluid evacuate is important so that the veterinarian can calculate the amount of intravenous fluids needed to prevent hypotensive shock.

wall potentially protruding above the level of the pubis and absence of fetal ballottement.[6] Rectal exam for hydrops amnion is similar to hydrops allantois, except the uterus will not be protruding above the level of the pubis.[6] Transabdominal sonography for hydrops allantois will reveal excessive allantoic fluid, with depth >18 cm highly suggestive of hydrops allantois. The normal amount of allantoic fluid depth is 4.7 to 22.1 cm, with a mean maximal depth of 13.4 ± 4.4 cm (Fig. 2).[7,8] Imaging of the non-pregnant horn usually reveals an abnormal amount of allantoic fluid.

In hydrops amnion, the sonographic appearance of the amnion is turgid, without the normal undulating appearance. The average normal volume of amnionic fluid in the mare is 3 to 7 L.[9] Excessive amnionic fluid depth will also be noted. Normal fluid depth of the amnionic cavity is 0.8 to 14.9 cm (mean, 7.9 ± 3.5 cm).[7,8]



Fig. 3. Sharp, 32F Trocar[c] to penetrate the chorioallantoic membrane. Note the water proof "waders" worn by the veterinarian. This is important to stay dry and comfortable during the draining procedure.

Treatment for hydrops usually consists of the early recognition and drainage of the excess fluid, which usually leads to termination of the pregnancy. There has been one report in literature in which conservative medical management for hydrops amnion resulted in a viable foal.[10] This animal, however, was noted to be of small stature at 1 year of age and had limb deformities that had to have several procedures performed over the first 10 month of life until they were eventually corrected. The complicating sequelae associated with hydropic conditions may include rupture of the prepubic tendon or abdominal musculature, inguinal herniation, uterine rupture, abortion, and hypovolemic shock. Uterine inertia associated with the prolonged stretching of the uterus and abdominal musculature is common during parturition.[3,10] This inertia can result in dystocia and retention of fetal membranes. Given the severity of the complications associated with hydropic conditions, slow drainage of the excessive fluid(s) with resulting abortion is often recommended to salvage the mare.[3]

The following technique describes how to safely drain excessive fluid and perform a vaginally assisted delivery in mares with hydropic conditions.

2. Materials and Methods

The following diagnostic equipment and supplies are needed:

- Ultrasound machine
- 5- to 7-mHz linear transrectal probe
- 2- to 3.5-mHz transabdominal probe
- 10- to 14-gauge intravenous catheter
- Two 1-L bottles of 8% hypertonic saline
- 40 to 60 L of polyionic crystalloids
- High-flow fluid administration set
- Prostaglandin E (PGE) gel[a] (if cervical dilation is desired)
- or *N*-butylscopolammonium bromide gel[b] (compounded)
- Brown gauze
- Lidocaine and scissors for episiotomy ("open" caslicks)
- Sterile gloves and sleeve
- 28F to 32F thoracic trocar[c] (sharp tip)
- Sterilized stomach tube
- 5-Gallon bucket
- Euthanasia solution[d]
- Flunixin meglumine[e]
- Sedation
- Detomidine[f]
- Romifidine[g]
- Xylazine[h]
- Prophylactic treatment with antibiotics
- Head snare
- Chains and handles
- Sterile lubricant

Evaluation of the Mare

Once a diagnosis of hydrops allantois or amnion is confirmed, fetal viability is determined, and the udder development and milk electrolytes (when warranted) will be assessed to estimate the level of fetal maturity. Client communication is critical to establish the relative value of the mare or fetus, the risks posed to each, and the potential sequelae. Mares that present early in gestation may undergo elective termination of pregnancy by intramuscular injection of cloprostenol[i] (500 μg IM q 12 h until delivery), as long as the amount of fluid is not too excessive to send the mare into shock with spontaneous abortion. Cases that occur later in gestation, or those with profound abdominal enlargement with large volumes of fluid within the uterus, usually require controlled drainage of the hydropic fluid before expulsion of the fetus to control cardiovascular shock. Mares that present during the last 2 to 4 weeks of pregnancy may be managed by conservative therapy (maintain abdominal wrap for support, veterinary evaluation at least weekly, and have veterinary staff available for correction of dystocia and hypotensive shock) or partial drainage. The aim of the partial drainage is to maintain pregnancy for as long as possible for additional fetal maturation to occur. A physical exam of the mare will be performed along with complete blood cell count, fibrinogen, and serum biochemistry. In the authors' experience, mares with high muscle enzymes (creatine kinase >1200 U/L) are at risk for rupture of abdominal musculature or the prepubic tendon. These mares should have controlled drainage of the fluid because of the high risk for fatal complications. In our experience, it is not uncommon to have an animal that is hypocalcemic. Hypocalcemic patients will be more vulnerable to hypotensive shock when the abdominal pressure is released during hydropic drainage. These animals may require intravenous calcium supplementation before and during drainage.

The placement of a large indwelling intravenous catheter (10- to 14-gauge) is important for the administration of polyionic crystalloid fluids while the allantoic or amnionic space is slowly drained. The amount of fluid administered intravenously that is needed to prevent hypotensive shock is dependent on how rapidly the hydropic fluids are drained. Initially, 10 L of fluids will be given during the first 30 to 60 minutes of the procedure. By the end of the procedure, it will not be uncommon to have administered an equivalent of one fourth of the volume drained intravenously (ie, if 100 L was drained, then 25 L had been given intravenously over an 8- to 12-hour time interval). If the mare's heart rate becomes greater than 56 to 60 bpm or the animal becomes ataxic or shaking, then the drainage of hydropic fluids should be stopped and a bolus dose of colloids (Hetastarch,[j] 10 mL/kg IV) or 8% (2–4 mL/kg IV) hypertonic saline[k] will be warranted to correct the hypotension and normalize the heart rate.

Hydrops Drainage

The technique for drainage involves several considerations, with the most important being safety and comfort for both veterinarian and the patient. Location (stocks or stall) is determined by the individual preference and the condition of the mare.

The mare may have to be initially sedated and treated with flunixin meglumine (1 mg/kg IV) for ease of management. The tail should be wrapped and the perineal area should be surgically prepared. If present, the caslicks should be "opened." Sterile technique should be followed during the procedure. Prostagladin E[a] or *N*-butylscopolammonium[b] bromide gel is gently massaged onto the cervix if cervical dilation is desired. A PGE_1 gel can be made by crushing 200 μg of misoprostol[l] tablets, resuspending in 1.5 to 2 mL of saline, and further mixing with 1.5 to 2 mL of sterile lubricant. PGE_2 gel may also be used.[11] After 5 minutes, the cervix can be gently dilated manually. The veterinarian should be careful not to excessively dilate the cervix because he or she will not be able to control the rate of drainage of the fluid. If the veterinarian does not want to abort the fetus or induce the mare, then the dilation of the cervix may not be recommended. Once dilated, the mucus plug will be easily removed and the chorioallantoic membrane can be palpated. The use of either a 28F or a 32F, sharp-tipped trocar[c] is then used to penetrate the chorioallantoic membrane

(Fig. 3). If a hydrops amnion has been diagnosed, then the trocar must penetrate both the chorioallantoic and the amnion membranes. A small, sterilized stomach tube is then placed through the trocarized membrane(s) to aid in the drainage of the fluid. The hydropic fluid will be collected for culture. The allantoic or amnionic fluid is then drained in a slow (over 1–2 hours, depending on the volume of fluid to be drained) and controlled manner. To help control the drainage of fluid, a clamp on the tubing or a finger can be used as a "valve" at the end of the tube. During the drainage process, the heart rate of the patient is evaluated every 5 to 10 minutes. Should the heart rate increase above 56 to 60 bpm, then the drainage should be halted and the administration rate of intravenous fluids (colloids and/or crystalloids) can be increased until the heart rate returns to baseline. The fluid will be collected in 5-gallon buckets so that the amount of fluid drained can be calculated (Fig. 4). Knowing the exact amount of fluid drained will enable the practitioner to calculate the amount of crystalloids that the animal should receive during and after the procedure. During this process, pericervical separation of the placenta is common.

If partial drainage and maintenance of the pregnancy is the goal, then these mares will be treated with additional antimicrobial therapy, flunixin meglumine[e] (1 mg/kg PO or IV q 12 h for 5–7 days) and double-dose altrenogest[m] (0.088 mg/kg PO q 24 h).

Extraction of the Fetus and Fetal Membranes

Manual extraction of the fetus can be performed, or the natural sequence of parturition can be allowed to occur. Weakening of the abdominal musculature (from overstretching) as well as uterine inertia are common findings that result in an inadequate stage II abdominal press and delivery. Malpositioning and malpostures are very common, and assisted vaginal delivery is usually needed; however, care must be taken not to traumatize the cervix. The expelled fetus generally is alive, and humane euthanasia will be warranted. The fetus should have a full necropsy as well as appropriate cultures and viral isolation to rule out an infectious disease.

Retention of fetal membranes should be expected, and appropriate treatment for removal and prevention of metritis-laminitis complex is indicated. Treatment generally consists of the prophylactic antimicrobials, and a low dose of oxytocin[n] (10–20 IU) should be administered intramuscularly initially. Oxytocin[n] administration should be repeated at 1- to 2-hour intervals to effect. The sensitivity to oxytocin[n] may vary, and a positive response is indicated by the passage of fluid from the vagina. Oxytocin[n] stimulates uterine contraction by increasing calcium release from the myometrium. Oxytocin[n] concentrations are typically low throughout gestation and increase during parturition. Uterine sensitivity to oxytocin increases toward term, potentially as the result of receptor abundance or regulation of signaling pathways after receptor binding.[12] Because most of these mares are several weeks from their foaling date, the receptor regulation of oxytocin[n] may be altered and the response to a typical dose of oxytocin[n] may be blunted. If the mare has already received oxytocin[n] intramuscularly and failed to respond, then an intravenous drip of oxytocin,[n] 60 IU added to 1 L of saline, should be started. The drip rate will vary on the individual mare (30 minutes to 1 hour).

The key to preventing metritis, endotoxemia, and laminitis is uterine lavage. Uterine lavage can be performed between the membranes and endometrium or by use of the Burn's technique of expanding the allantoic cavity to facilitate separation. This can be repeated until the fluid comes back clear. If a section of membrane can be reached, then it may be gently twisted off the endometrium and removed. However, if it is firmly adhered, then traction should be avoided because it may result in uterine inversion or tearing. It is not uncommon to have fetal membranes be retained for 2 to 3 days. To help prevent toxic metritis the uterus may be lavaged with large volumes of fluids 2 to 3 times per day if necessary. Intrauterine infusion with antimicrobials is recommended by these authors but remains controversial.[13] Prophylactic treatment for laminitis should include deep bedding, with or without cryotherapy of the hoofs, flunixin meglumine[e] 1.1 mg/kg IV q 12 h (or q 8 h if endotoxemic), and hoof pads.[o]

3. Results

More than 30 mares over the past 10 years have been treated by these authors' with the use of this technique. Approximately 90% of these mares exhibited signs of hypotensive shock during the drainage of fluid, which included muscle tremors, elevated heart rate, and colic. These signs subsided with appropriate fluid/colloid therapy. The majority of the mares recovered well from the procedure and later were discharged from the hospital. One mare died after 72 hours as a result of rupture of a uterine artery. In 10 mares that were within 4 weeks of term, maintenance of the pregnancy has been attempted after partial drainage of the allantoic compartment. These mares were treated with additional antimicrobial therapy, flunixin meglumine[e] (1.1 mg/kg IV or PO q 12 h for 3–5 days), and altrenogest[m] (0.044–0.088 mg/kg PO q 24 h). In cases in which partial drainage has been attempted, fetal death can occur as a result of fetal asphyxia that results from varying degrees of placental separation. Iatrogenic fetal infection can also occur, secondary to contamination of the placental fluids during drainage. To date, all the authors' mares that were treated with partial drainage aborted a nonviable fetus.

4. Discussion

Neither hydrops allantois nor amnios are common conditions in the pregnant mare, but, if not detected early can lead to the death of both mare and foal. Hydrops amnion, unlike hydrops allantois, develops gradually over several weeks to months during the second half of gestation. Although swallowing in the fetus plays a role in the maintenance of fetal fluid balance, other mechanisms may be important. Amniotic fluid in mares is composed of secretions of the amnion and the nasopharynx of the fetus, fetal saliva, transudation from maternal serum, and fetal urine.[2,14] Whether the problem arises because of an increase in secretion or decrease in resorption or both is not clear.[14] It has been suggested that the fetus might actively regulate the volume and composition of the amniotic fluid by deglutition, and the prevention or impairment of swallowing may lead to hydramnios.[2]

The pathophysiology of hydrops allantois in the mare remains unknown. Some authors have suggested that the increase in fluid is a placental problem caused either by increased production of fluid or decreased transplacental absorption.[2] Others have proposed that the etiology is related to placentitis and heritability.[3] One of the authors (N.M.S.) has diagnosed two cases of hydrops allantois associated with leptospirosis that was isolated from the fetus and placenta.

Early management is aimed at preventing body wall tears and hypotensive shock, which improves the mare's prognosis, because obtaining a live foal has not been reported with hydrops allantois and has been reported only once with hydrops amnios.[2,10] Hydropic conditions have a higher incidence of a deformed fetus with facial, genetic, or congenital abnormalities.[15,16] Clients who therefore wish to invest in saving the fetus should be informed of these potential deformities and of the poor prognosis for fetal survival. The medical management for hydropic conditions described have been very efficacious in preventing life-threatening complications in the mare (100% success) but poor in regard to maintain a viable pregnancy (0% success). This procedure does not require a hospital setting and can be performed in the field, provided that the veterinarian has all of the necessary equipment. Careful patient assessment, which would include a full physical, complete blood cell count, and serum biochemistry, should always be performed before initiating the drainage of the allantoic or amnionic fluid. The technique for drainage may take several hours; therefore, comfort for both the patient and veterinarian would be required. Mares that present early in gestation may undergo elective termination of pregnancy by intramuscular injection of cloprostenol, as long as the amount of fluid is not too excessive to send the mare into shock with spontaneous abortion. If the allantoic or amnionic fluid is excessive, then partial drainage with or without manual extraction of the fetus can be attempted. Most of the mares presented to our hospital for drainage of hydropic fluid had signs of hypotensive shock (ie, muscle tremors, elevated heart rate, and colic). These signs subsided with appropriate fluid/colloid therapy. For successful medical management of hydropic conditions, the access to a central vein (ie, placement of a jugular intravenous catheter) and the use of crystalloids and, when warranted, colloids, should always be available when this procedure is performed. The fluid should always be drained in a control manner over a period of 1 to 2 hours. The slow drainage will allow the animal's blood pressure to compensate to the decreased intra-abdominal pressure that was associated with the hydropic condition. Once the partial drainage has been completed, then either manual extraction of the fetus or the natural sequence of parturition can be allowed to occur. Retained fetal membranes and fetal malpositioning/dystocia are very common with this medical condition. The technique described is very successful in decreasing the sequelae (ie, abdominal wall hernias, prepubic tendon rupture, and cardiovascular shock) of untreated hydrops allantois/amnion of the mare. Because it is possible that a heritable component to this condition exists, breeding to a different stallion may be prudent.

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[a]Prepidil, Pharmacia & Upjohn, Kalamazoo, MI 49001.
[b]*N*-butylscopolammonium bromide 5 mg/mL gel, Hagyard Pharmacy, Lexington, KY 40511.
[c]28F or 32F Argyle 41-cm Trocar Thoracic Catheter, Tyco Healthcare, Mansfield, MA 02048.
[d]Beuthanasia, Merck Animal Health, Summit NJ 07901.
[e]Banamine, Merck Animal Health, Summit, NJ 07901.
[f]Dormesedan, Pfizer Corporation, Orion Pharmaceuticals, Exton, PA 10017.
[g]Sedivet 1%, Boehringer Ingelheim Vetmedica Inc, St Joseph, MO 64506.
[h]Xylazine, IVX Animal Health, Inc, St Joseph, MO 64503.
[i]Estrumate, Merck Animal Health, Summit, NJ 07901.
[j]Hespan 6%, Braun Medical, Irvine, CA 92614.
[k]Hypertonic saline solution 7.2%, Nova-Tech Inc, Grand Island, NE 68801.
[l]Misoprostol 200-μg tablets, IVAX Pharmaceuticals Inc, Miami, FL 33137.
[m]Regumate 0.22%, Merck Animal Health, Summit, NJ 07901.
[n]Oxytocin 20 IU/mL, Bimeda Inc, Le Sueur, MN 56058.
[o]Soft-Ride, Soft Ride Inc, Vermillion, OH 44089.

How to Diagnose and Treat Hemospermia: A Review and Case Series

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1. Introduction

Disorders of breeding stallions have severe ramifications for not only the economics of the equine industry but also the welfare of affected animals. Hemospermia in particular is challenging to diagnose and manage, as there are many etiologies and treatment options. Importantly, one of the mainstays of treatment for all cases is sexual rest, which has severe economic consequences on the stallion's reproductive performance. Because hemospermia is associated with heavy breeding or collection schedules, the disease is often diagnosed in the beginning or height of the breeding season. Therefore, timely identification of the disorder, diagnosis of the pathophysiology, and implementation of a treatment protocol will provide the best opportunity to return the stallion to breeding function, albeit most often for the following season. Some causes of hemospermia are life-threatening, such as squamous cell carcinoma, and early diagnosis and treatment may improve survival rates.

Often, the first indication of hemospermia, especially in live-cover breeding operations, is infertility. Presence of erythrocytes within the ejaculate may dramatically reduce pregnancy rates as the result of effects of an unknown factor of the erythrocytes on spermatozoa. Alternatively, blood may be noted at the mare's vulva or stallion's penis on dismount. Stallions that are collected by means of an artificial vagina may be identified by blood-contaminated ejaculates. External lesions may be observed during washing before collection. Stallions may demonstrate normal libido but pain on erection, dilation of the glans, or ejaculation. Pain may also be observed in association with masturbation behavior or urination.

The objectives of this paper are 1) to provide an overview of the diagnostic evaluation of stallions that present for hemospermia; 2) to review the major causes of hemospermia and their treatment options; 3) to present a case series of stallions with hemospermia that represent cases that veterinarians commonly see in practice.

2. Diagnostic Protocol

Signalment

Hemospermia has been reported in stallions of variable ages and breeds; however, in one report of 18 cases, 15 were Quarter Horses (83.3%). Of the 18

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horses, the average age at onset of hemospermia was 7.1 years (range, 3–18) and the number of seasons at stud was 3.7 (range, 0–13).[1]

History

Hemorrhage may be noted during washing of the penis, from the penis after breeding or collection, on the phantom after collection, from the mare after live-cover mating, or associated with urination. The horse may have a history of pain during urination, masturbation, erection, or ejaculation. Self-mutilation behavior and colic have been described in a stallion with hemospermia caused by seminal vesiculitis.[2] Important history to acquire from the owner includes the following:

- Any previous episodes of hemospermia
- Any change in stallion body weight or condition
- Any change in stallion collection frequency
- Any recent adjustments to the collection phantom, such as height, type, and so forth (stallion use: collection only, live-cover mating, or both)
- On observation of hemospermia, was the blood expelled as large clots or progressive from blood-tinged to frank blood?
- Any observations of bleeding in the stall, such as associated with urination
- Any observations of bleeding when the penis is washed during erection

Physical Examination

Evaluation of the stallion that presents for hemospermia begins with a thorough physical examination. The horse may require sedation to facilitate examination of the external genitalia. The epithelium of the prepuce and penis should be evaluated for any lesions, including papules, pustules, erosions, ulcers, proliferative lesions, or lacerations. Special attention should be paid to the urethral process and urethral fossa because these are common sites of injury and neoplasia, although some lesions of the glans are not apparent until erection occurs. The epididymis should be palpated and imaged for enlargement or an increase in firmness. Transrectal ultrasonography may demonstrate abnormalities of the ampullae, seminal vesicles, bladder, or inguinal lymph nodes, which would indicate further diagnostic testing.

Semen Collection

The stallion is teased and washed, and semen is collected through the use of an artificial vagina. Some practitioners prefer to use an open-ended Colorado-style artificial vagina to fractionate the ejaculate, although in most cases this is not necessary to obtain a diagnosis.[3] Stallions with painful lesions of the glans penis or urethral process may not ejaculate because of pain associated with the lesion at the time of dilation of the glans. These stallions demonstrate normal libido but become acutely painful and dismount the phantom without ejaculating. Repeated collection attempts may result in frustration of the stallion. Often, examination of the penis after dismount will demonstrate the source of hemorrhage on the glans or urethral process. Stallions that ejaculate grossly hemorrhagic semen but do not display external lesions should be submitted for endoscopy within 20 minutes to identify the origin of hemorrhage. Stallions with a history of hemospermia that have had a period of sexual rest may not hemorrhage during initial collections but may do so after repeated collections over days to weeks.

Collected semen with hemospermia on gross or macroscopic inspection may appear pink to red in color. On gross inspection, semen with microscopic hemospermia may appear to be normal; the use of cytology is imperative to the diagnosis. Cytology that demonstrates the presence of large numbers of leukocytes and erythrocytes should direct the clinician to evaluate the urogenital tract for inflammatory or infectious processes of the bladder, epididymis, seminal vesicles, or urethra.

Endoscopy

In the absence of lesions of the glans penis and urethral process, endoscopy should be performed within 20 minutes of collection to examine the urethra, colliculus seminalis, seminal vesicles, and urinary bladder. The horse is sedated, often with an α-adrenergic agonist such as xylazine or detomidine and butorphanol intravenously. The penis is washed with warm water and non-irritating soap. Any accumulation of smegma in the urethral fossa should be removed. The penis is then dried. Endoscopic examination requires the use of three personnel: one to aseptically insert the endoscope into the urethra and hold the extended penis; one to operate the endoscope; and one to restrain the horse. Most adult light-breed horses can be examined with a sterilized flexible endoscope of 100-cm length and 10-mm diameter.[4] Small ponies and miniature horses require the use of a pediatric gastroscope of 8- to 9-mm diameter.[4] Large horses may require the use of a longer endoscope. Smaller-diameter endoscopes are easier to introduce into the seminal vesicles.

The endoscope is inserted into the urethra with a small amount of sterile lidocaine-containing lubricant by an operator who maintains one hand around the glans and one hand to advance or retract the depth of the endoscope, while wearing sterile examination gloves. The endoscope is advanced slowly to observe for signs of pathology. Small volumes of air may be applied to facilitate examination. Dilation of the urethra results in hyperemia of the underlying corpus spongiosum vasculature, which should not be confused with urethritis.[4] At the level of the ischial arch, the convex surface of the urethra should be closely examined for hemorrhage caused by idiopathic urethral rent formation. A

hypodermic needle may be placed percutaneously to identify the level of an identified rent.[4] Openings to the urethral glands and bulbourethral glands are located proximal to the ischial arch. The pelvic urethra should be examined for signs of urethritis. The colliculus seminalis should be examined for discharge from the seminal vesicle or presence of any cystic structures.[5] The seminal vesicles can be entered with the use of a sterile stylette and examined for purulent or hemorrhagic exudate. Transrectal massage of the seminal vesicles may elicit elimination of accumulated fluid and may improve diagnosis. Bacteriological samples may be obtained from the seminal vesicles or urethra. The bladder should be examined for cystitis or uroliths.

3. Differential Diagnoses and Treatment Options

Penile Neoplasia

Squamous cell carcinoma (SCC) represents the majority of diagnosed penile neoplasms. Hemospermia may occur as the result of ulceration of the glans or irritation of neoplastic tissues during erection. In a recent retrospective study of 3351 equine cutaneous neoplasms, 18.9% were SCC, as were 57.8% of penile or preputial tumors. In another study, 74 of 114 (65%) of equine penile or preputial tumors were SCC. The mean age of affected horses ranged from 16.4 to 19.8 years.[6,7] SCC is commonly identified in horses with unpigmented genitalia but can occur in any breed or color.[7,8] Appaloosa and American Paint horses are significantly more affected than other breeds.[7] Stallions appear to be less frequently affected than geldings, which may be a reflection of the respective population sizes.[9]

The pathogenesis of penile SCC has been hypothesized to result from infection with equine papillomavirus-2 (EcPV-2), which induces neoplastic changes in penile or preputial epithelium or existing papillomas.[10,11] In 16 penile SCC, 15 were positive for EcPV-2 DNA as was a metastatic lymph node.[12] Interestingly, 10% of healthy males were positive for EcPV-2 DNA in penile tissues, suggesting that some horses may be asymptomatic carriers.[12] Other factors relative to SCC formation include irritation of the skin by smegma or by ultraviolet sunlight exposure. The lesions are slow-growing, locally invasive, and metastasize late in disease to the inguinal lymph nodes. SCC which invade the cavernosus tissues of the penis may metastasize hematogenously. Lesions are often not identified in nonbreeding stallions until gross abnormalities are present, resulting in pain during collection, urination, or masturbation; hemorrhage; or a foul odor. Early lesions may be raised, discolored, or ulcerated; later lesions may become granulomatous or "cauliflower-like" in appearance. Large lesions may interfere with normal extension and retraction of the penis as well as urination.

Treatment options for penile SCC depend on the size and character of the lesion as well as the presence of metastasis. Small lesions may be treated with chemotherapy, cryotherapy, or laser excision. Chemotherapy options include topical 5-fluorouracil (5-FU), intralesional cisplatin, or oral cyclooxygenase inhibitors. Five-fluorouracil, a fluorinated pyrimidine, is an anti-metabolite that blocks the methylation of deoxyuridic acid into thymidylic acid in the formation of DNA.[13] Because thymidylic acid is high in SCC, 5-FU is thought to induce thymidylate deficiency in the neoplastic cells, which leads to apoptosis; normal non-cancerous cells are not affected.[13] A study of eight horses with penile or preputial SCC monitored the response to topical 5-FU after surgical debulking or as solitary treatment for small lesions.[13] The treatment was applied immediately after surgery or the following day, after hemostasis had occurred, and additional treatments were performed at 14-day intervals until regression of the SCC. Thereafter, horses were evaluated and treated every 6 months to prevent remission. The mean number of treatments to achieve remission was five (range, two to seven), and all horses were still in remission 5 to 52 months later.

Cisplatin, *cis*-diamminedichloroplatinum (II) or CDDP, is a platinum-containing chemotherapy agent that binds and cross-links DNA to induce apoptosis. It has been administered intratumorally to reduce the toxic systemic effects observed in some patients and to maximize tissue concentrations of this chemotherapeutic agent. Cisplatin in sesame oil emulsion was used to treat 151 SCC in 144 horses, either as solitary treatment or 10 to 14 days after surgery.[14] Eighteen of these SCC were of male genital origin. Four treatments were administered at 2-week intervals. The cure rate after one course of treatment was 88%.[14]

The use of cyclooxygenase (COX) inhibitors as chemotherapy agents has been researched in recent years. It has been demonstrated that COX is overexpressed in equine SCC and plays a role in cell growth and differentiation.[15] Inhibition of COX-2 induces apopotosis in neoplastic cells by blocking prostaglandin E2 synthesis and therefore angiogenesis and invasiveness.[16] Elce et al[16] demonstrated the expression of COX-1 and COX-2 proteins in tissue samples of four preputial and five penile SCC in horses. COX-2 expression was significantly increased in neoplastic versus non-neoplastic preputial tissues ($P = 0.04$).[16] This study also suggested that COX expression in horses was not limited to SCC but also occurred in normal tissues, a finding unique to horses. Another study demonstrated expression of COX-2 in 32 of 37 (86.5%) equine SCC, seven of which were preputial or penile in origin.[17] The use of piroxicam was extrapolated from use in other species, such as rats, dogs, and humans, to treat SCC.[16] Oral piroxicam was used to treat recurrence of non-genital SCC in a 16-year-old Morgan gelding.[18] The SCC had initially been treated with intralesional cisplatin; 3 months after initiation of piroxicam therapy, the lesion and enlarged regional

lymph nodes returned to a normal size. After 5 years, the horse was still receiving oral piroxicam once every 2 to 3 days, and there had been no signs of recurrence. Specific studies examining the COX selectivity and toxicity of piroxicam in horses have not been published, although in other species it is COX–non-selective.

Cryotherapy has been applied as solitary treatment for small lesions or in combination with surgery.[8] In one study, eight horses were treated with liquid nitrogen, which was applied with the use of a cryoprobe for two fast-freeze, slow-thaw treatments, as solitary treatment, or in combination with local excision or partial phallectomy. Of seven horses available for follow-up, SCC recurred in five animals.[8] Epithelialization after cryotherapy may be prolonged compared with surgical excision alone because of thermal damage.[9]

The prognosis for horses that undergo surgical treatment for SCC is variable. One study of en bloc resection with penile retroversion for penile SCC ($n = 4$) or squamous papilloma ($n = 1$) demonstrated that three of five ponies were alive at 3 to 20 months of follow-up (mean, 12 months).[19] One stallion with urethral process SCC underwent local excision and returned to breeding function without recurrence at 1 year.[20] Twenty of 31 pony geldings (64.5%) lived at least 18 months without relapse of penile SCC regardless of treatment method (penile amputation, local excision, or preputial ablation).[10] Six of nine (66%) that had penile amputation survived 18 months.[10] A study of 45 horses with genital SCC treated by en bloc penile resection, penile amputation, and/or segmental posthectomy demonstrated that recurrence occurred in 19% (six of 31) of horses for which follow-up was available. Seventy-one percent (22 of 31) of horses were alive at the time of follow-up, which ranged from 1 to 6 years after surgery. Notably, of the 45 cases examined, only 35 had histologic confirmation of SCC. Recurrence rates in a study of 77 equine preputial or penile SCC were 43.5% (17 of 39) for horses that had partial phallectomy with or without cryosurgery or that had incomplete removal of SCC; 12.5% (one of eight) for horses that had en bloc resection; and 25% (one of four) for horses that had confirmed lymph node metastasis. The overall recurrence rate was 29.5%. Successful removal and prevention of recurrence of SCC was 55.7%.

En bloc resection of the penis requires concurrent castration. Breeding stallions that have undergone partial phallectomy may be able to be collected through the use of an artificial vagina or induction of ex copula ejaculation and semen used for artificial insemination or cryopreservation. Other penile neoplasms of horses include sarcoid and melanoma, which typically do not result in hemospermia.

Penile Habronemiasis

Although uncommon in the United States, penile habronemiasis is an important differential diagnosis for penile SCC. Hemospermia results from collection from affected horses, with hemorrhage originating from inflamed or ulcerated areas of the glans penis. Its incidence has been drastically reduced because of the widespread use of avermectin anthelmintics. Stable and house flies, *Musca domestica* and *Stomoxys calcitrans,* respectively, serve as intermediate hosts that deposit the larvae of the stomach worms *Habronema spp.* or *Draschia megastoma* on mucocutaneous junctions of the genitalia or in open wounds.[21,22] Infection of existing SCC or lacerations are possible.[22] Resultant lesions are not the result of tissue damage caused by the larvae but rather by a severe hypersensitivity reaction to their presence, which results in exuberant granulation tissue production, ulceration, and hemorrhagic exudate that may result in hemospermia, hematuria, or dysuria.[23] Diagnosis is confirmed by excisional biopsy that demonstrates small, hard, yellow granules, larvae, and eosinophils and will rule out sarcoids, SCC, or pythiosis.[23] Complete blood count may demonstrate eosinophilia. Horses with penile habronemiasis should be suspected of having a large adult stomach worm population.[23] Treatment is performed through administration of avermectin anthelmintics followed by corticosteroid treatment to reduce the immune reaction. Development of avermectin resistance may lead to an increase in observed cases. Prednisolone has been used orally, and liquid nitrogen sprays have been used topically to kill larvae in small lesions. Management factors include the deworming schedule of horses, manure management, and fly control.

Penile Injuries

Hemospermia caused by penile injuries may be of several etiologies. The urethral process may be easily damaged by the mare's tail hairs during live-cover breeding. Penile lacerations, swelling, or hematomas may also occur as the result of kicks (either by the mare during breeding or by the stallion's hind legs during washing) or use of stallion rings. Some lacerations may not be apparent until erection is achieved and the lesion hemorrhages under increased pressure.[24] Injured tissues should be cleaned and a topical antibiotic and/or anti-inflammatory applied. Lacerations into the cavernosus tissues may require surgical closure.[25] Rapidly swelling tissue should be treated with a pressure bandage and abdominal support to prevent paraphimosis. Ability to urinate should be verified. Sexual rest is indicated until the lesion is healed because hemorrhage may recur with subsequent mating or artificial vagina use.

Equine Coital Exanthema (Equine Herpesvirus-3)

Infection with equine herpesvirus-3 can lead to pustules and ulceration of the penile surface, which can result in hemospermia on erection and service of an artificial vagina or mare.[26] The infection is self-limiting after several weeks. Sexual rest is indi-

cated because the infection is highly contagious and the lesions are painful. The disease is endemic in the United States.

Urethritis

In a review of 18 cases of hemospermia, urethritis was identified as the inciting cause in 10 cases.[1] Features of urethritis included pseudomembranous ($n = 1$), polypoid ($n = 1$), concurrent urethral stricture ($n = 1$), and non-specific inflammation ($n = 7$).[1] Bacterial cultures from 10 of 18 cases demonstrated growth of *Escherichia coli*, *Pseudomonas aeruginosa*, α- and β-hemolytic *Streptococcus* spp., *Streptococcus equiseminalis*, and *Proteus* spp. Four cases had histologic evidence of infection. Lesions were identified in the pelvic urethra in 44.4% of cases. Sexual rest was successful in returning two stallions to breeding function. Surgical treatment by subischial urethrostomy was performed in the remaining stallions with urethritis, and suppositories of nitrofurazone and hydrocortisone were administered. Eighty percent of stallions with urethritis treated by urethrostomy and suppositories returned to normal ejaculation.[27]

One case of viral urethritis has been suspected on the basis of identification of inclusion bodies in urethral epithelial cells, but virus isolation was unsuccessful.[28]

Although these and several other cases of bacterial urethritis are frequently cited, very few cases have been described in the scientific literature in the last 25 years. It is possible that the disease is under-reported or that most cases of hemospermia examined clinically or reported on in recent years have been of other etiologies.

Urethral Rent

Urethral rent development is a distinct disorder of the urethra in stallions that results in hemospermia. Urethral rents can occur in any age or breed, but Quarter Horses appear to be over-represented.[29] The disease can also occur in geldings, but results in hematuria, a clinical sign less often observed in stallions. These variable clinical signs are physiologically based in the fact that corpus spongiosum penis pressure during urination in geldings versus stallions is higher (25.5 ± 12.1 versus 15.3 ± 3.3 mm Hg)[30] and that corpus cavernosum penis pressures in stallions during teasing (107 ± 8.4 mm Hg) and breeding (4147 ± 142 mm Hg) are significantly higher than during urination or rest (13 ± 1.5 mm Hg).[31] Therefore, pressures during urination in geldings and erection in stallions are elevated enough to result in hemorrhage from an established rent. The authors have observed hematuria at the end of urination in some stallions with urethral rents, simultaneous with ischial muscle contractions. Therefore, observation of the stall for blood and the horse's hind limbs for dried blood are important. It should be noted that urethral defects can heal or begin to seal over with sexual rest and that not all ejaculates are contaminated. A stallion that presents for evaluation of a urethral rent may need to have semen collected several times over hours to days to incite hemorrhage, which can then be confirmed through urethroscopy as being caused by urethral rent.

Urethral rents are located on the convex aspect of the urethra at the level of the ischial arch. Although hypotheses have been made that this area of the urethra is inherently weaker,[29] histologic examination of this region in geldings and stallions with normal and affected urethras did not demonstrate any differences in the lamina propria, urethral mucosa, or corpus spongiosum penis thickness.[30] Defects are linear, longitudinal, and range from 3 to 10 mm in length.

Treatment for urethral rent begins with sexual rest. In some stallions, adequate sexual rest results in complete resolution of hemospermia and rent healing.[29] The most commonly used surgical technique to treat urethral rent is that of subischial urethrostomy. Alternatively, through the use of the same surgical approach, the urethra may not be incised. Dissection of these tissues allows for decreased corpus spongiosum penis pressure during erection at the level of the rent, which can facilitate healing. Topical corticosteroids and or nitrofurazone have been placed in the surgery site during healing.[1] The incisions are left to heal by second intention. Postoperative complications include urethral stricture, infection, fistula formation, or recurrence of urethral rent.

Semen may be collected after several months of convalescence. In some cases, the rent may not completely heal until the following breeding season.[1,29] In a recent report, a buccal mucosal urethroplasty successfully treated a urethral rent in one stallion and returned it to breeding function.[32]

Seminal Vesiculitis

Seminal vesiculitis is an important disease of stallions that is probably under-reported and can be associated with hemospermia.[33] Bacterial vesicular adenitis results in contamination of the ejaculate with mucopurulent debris, which may be apparent as chunks or pus.[34] The ejaculate ranges in color from bloody to brown. Often, the discoloration is observed in the final jets of an ejaculate.[35] On gross inspection, normal-appearing ejaculate may be diagnosed microscopically by the presence of a large number of neutrophils. Palpation per rectum of the glands may demonstrate enlargement of one or both glands, and, in acute cases, may elicit pain. In one case, colic and self-mutilation behavior were described in association with seminal vesiculitis.[2] Diagnosis is performed by endoscopic examination of the seminal vesicles, including collection of samples for bacteriology. Common isolates include *P aeruginosa*, *Klebsiella pneumonia*, *Streptococcus* spp., and *Staphylococcus* spp.,[35,36] although in some cases no specific pathogens are isolated.[2] The

pathogenesis and risk factors of the disease are not known. Treatment options include systemic antibiotics and anti-inflammatories as well as endoscopic-assisted lavage of the seminal vesicles and infusion of antibiotics. To attain high antibiotic concentration in the seminal fluids, an antibiotic must have a high pKa and lipid solubility; gentamicin has been demonstrated not to penetrate the seminal vesicles.[34] There are no large studies that provide epidemiologic data on prognosis for recovery from this disease. In some cases, resolution is possible. In others, the reproductive management of the stallion is altered to reduce risk of transmission of pathogenic bacteria to mares and improve pregnancy rates. One study of fertility that used semen from a *P aeruginosa*–infected stallion demonstrated pregnancy rates of 78% (seven of nine) when mares were artificially inseminated with semen that had been incubated in a polymixin-B–containing extender for 20 to 30 minutes.[37] Pregnancy rates of 50% (two of four) were achieved when mares were infused with the same extender immediately before live-cover mating.[37] A recent report described a stallion with *Streptococcus* spp. seminal vesiculitis that was medically managed to facilitate semen cryopreservation.[38]

4. Case Series

Materials and Methods

A retrospective study was performed of stallions that had presented to the Washington State University Comparative Theriogenology service between 2003 and 2012. Eight cases met the inclusion criteria of stallions with hemospermia. The mean age at the time of presentation was 10.1 years (range, 6–20 years). Quarter Horses represented 50% of the affected animals; other breeds represented were Appaloosas (25%) and Arabians (25%). Fifty percent of cases were referred from the primary veterinarian for advanced examination.

Case 1

An 11-year-old Appaloosa stallion was referred for evaluation of hemospermia. Blood had been observed during dismount after two live-cover matings. The horse had been placed on a 10-day course of trimethoprim-sulfamethoxazole by the referring veterinarian after urethral cultures were positive for bacteria (results of which were not available). After 1 month of sexual rest, semen was collected again, and hemospermia was noticed. The history also included unilateral testicular degeneration associated with a kick by a mare 5 years previously.

On presentation, the horse demonstrated normal physical examination parameters. The left testis was small and fibrotic, consistent with the history. Semen was collected through the use of an artificial vagina. On gross examination, hemospermia was observed, and blood continued to drip from the penis for several minutes after collection. Urethroscopy demonstrated a rent approximately 4 cm distal to the bulbourethral gland orifices on the convex surface of the urethra (Fig. 1). Induction of ex copula ejaculation was attempted, but ejaculation did not occur. The horse was discharged with instructions for sexual rest. The horse was examined 5 months later. Two ejaculates collected 30 minutes apart were free of erythrocytes. Bacteriological samples of the urethra before and after ejaculation were negative for reproductive tract pathogens. The horse resumed standing at stud for several years thereafter.

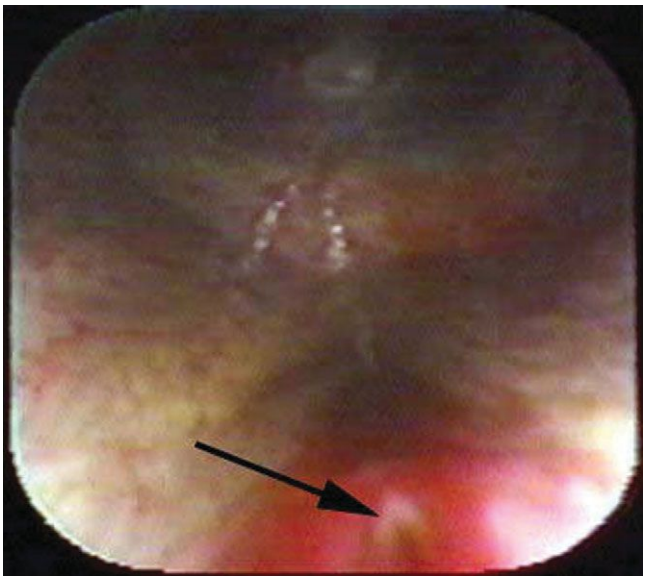


Fig. 1. Endoscopic image of a urethral rent (arrow) at the level of the ischial arch in an 11-year-old Appaloosa stallion that was referred for evaluation of hemospermia.

Case 2

A 20-year old Quarter Horse stallion was referred for evaluation of hemospermia. He was used for live-cover breeding. One year previously, he was diagnosed with penile squamous cell carcinoma, which was confirmed by biopsy. Information regarding treatment was not available. The horse covered eight mares in the current season, with occasional hemorrhage noted after erection or mating. Fertility data were not available. The stallion was referred for evaluation after live-cover mating was attempted, but the horse did not ejaculate, and frank blood was noted from the penis.

On presentation, the horse was in good body condition. The physical examination parameters were within normal limits. The horse was teased, and semen collection was attempted by means of an artificial vagina. The horse did not ejaculate, became acutely painful, and dismounted. Frank hemorrhage was noted from the urethral fossa. The horse was sedated for examination. The urethral fossa contained two ulcerated areas approximately 1 cm



Fig. 2. Glans penis of a 20-year-old Quarter Horse that had been previously treated for penile SCC and that was referred for evaluation of hemospermia.



Fig. 4. Urethral process lesion of an 8-year-old Arabian stallion that demonstrated hemospermia when vigorously washed or teased.

in diameter (Fig. 2). The dorsal surface of the glans penis as well as the body had depigmented, firm, raised areas. The lesions were suggestive of recurrence of SCC. Urethroscopy was unremarkable. Transrectal palpation and ultrasonography demonstrated a large, firmly adhered mass associated with the left inguinal ring, which may have represented metastasis to the internal inguinal lymph node (Fig. 3). Induction of ex copula ejaculation was attempted on two occasions, with no resultant ejaculation. Several treatment options were presented including partial phallectomy, en bloc resection of the penis and lymph nodes, radiation, and chemotherapy (oral piroxicam and topical 5-FU), all of which were declined by the owner. Follow-up by telephone revealed that after 6 months, the horse developed preputial and penile swelling, which resulted in penile prolapse. Treatment was unsuccessful, and the horse was euthanized.



Fig. 3. Transrectal ultrasound image of the left internal inguinal ring of a 20-year-old stallion with SCC. The mass was approximately 4.5 cm diameter, firm, immovable, and highly suggestive of metastasis.

Case 3

An 8-year-old Arabian stallion presented for semen collection and cryopreservation. On gross examination, initial semen collection resulted in hemospermia. Examination after collection demonstrated an erosion of the urethral process (Fig. 4). Cytology demonstrated a nondiagnostic cellular atypia. Urethroscopy was unremarkable. The horse was placed on a regular collection schedule for semen collection and cryopreservation. The horse continued to have intermittent hemorrhage from the urethral process, especially if teased or washed vigorously. Induction of ex copula ejaculation was unsuccessful. The horse was managed by increasing the intercollection interval to 4 to 7 days, with minimal handling and teasing time. This management resulted in successful collection of semen without hemospermia and healing of the urethral process. Frozen semen was shipped on several occasions and had acceptable fertility (data not available).

Case 4

An 8-year-old Quarter Horse stallion was referred for evaluation of hemospermia. He had successfully bred seven of eight mares in the previous year. At the time of presentation, he had been live-cover–mated to one mare, and blood was observed on the penis and the mare's vulva on dismount. On presentation, the horse's physical examination param-



Fig. 5. Hemospermia after ex copula induction of ejaculation in a 7-year-old Quarter Horse stallion with two urethral rents.

eters were within normal limits. There was dried blood on the penis. The horse was washed, and two ejaculates were collected at 30-minute intervals. No hemospermia was noted. The ejaculates were pooled and processed for cryopreservation. The horse resumed a successful career at stud.

Case 5

A 7-year-old Quarter Horse stallion presented for evaluation of hemospermia of 2 weeks' duration. Hemorrhage from the penis had been noted on erection, after breeding, and lasting for up to 30 minutes after breeding. On presentation, the physical exam was unremarkable. Semen was collected; blood was observed from the urethra on erection. On gross inspection, hemospermia was evident. Urethroscopy demonstrated two urethral rents, located 40 and 55 cm from the urethral process, respectively. Induction of ex copula ejaculation was successful, but hemospermia was evident (Fig. 5). After 6 months of sexual rest, no hemospermia was noted on semen collection. The horse was thereafter lost to follow-up.

Case 6

A 6-year-old Arabian stallion was referred for evaluation of hemospermia of 3 years' duration. After the initial observation, the referring veterinarian diagnosed a urethral process lesion. A biopsy of the urethral process demonstrated inflammation and dystrophic mineralization. The lesion was treated with a topical antimicrobial ointment for 10 days. One month later, granulation tissue was surgically removed from the urethral process. Subsequently, the horse was used as a teaser stallion. Semen was collected 1 month before presentation, and hemospermia was observed (the ejaculate was pink). Examination of the stallion demonstrated several small ulcerations of the urethral process that were not actively bleeding. Urethroscopy was unremarkable. Induction of ex copula ejaculation was unsuccessful. Semen was collected through the use of an artificial vagina. Semen was free of erythrocytes. After collection, the urethral process was erythematous and inflamed. Subsequently, the stallion was no longer needed for teasing, and castration was elected.

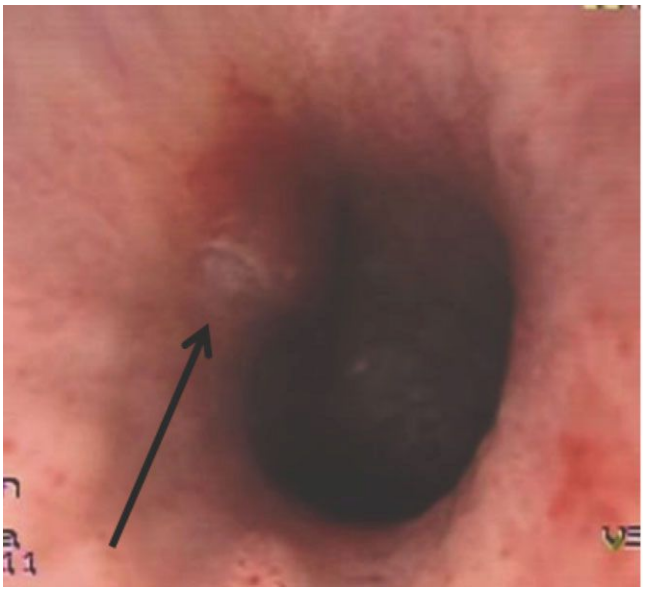


Fig. 6. Healing urethral rent (arrow) in a 13-year-old Quarter Horse stallion with recurrent hemospermia.

Case 7

A 13-year-old Quarter Horse stallion presented for examination of hemospermia of 1 week's duration. Blood clots had been observed from the horse's penis after live-cover mating. The horse had been submitted the previous season to our service for collection and cryopreservation of semen. Semen was collected twice, with no evidence of hemospermia; semen was processed for cryopreservation. Urethroscopy demonstrated a healing urethral rent at the level of the ischial arch (Fig. 6). After a period of sexual rest, the horse successfully bred several mares. Five months later, hemospermia was again noted. Semen was collected at the clinic three times within 35 minutes, and no hemospermia was noted; ejaculates were processed for cryopreservation.



Fig. 7. Overt hemospermia observed during semen collection in a 13-year-old Quarter Horse with a urethral rent.

After 7 months of sexual rest, semen from this horse was collected at the farm, and hemospermia was again observed. The owner of the horse indicated that the phantom used to collect semen at the farm was of a steeper angle than the phantom at our clinic; the significance of this to the lack of hemospermia observed at our clinic is unknown. Semen from this horse was collected twice at the clinic; the second ejaculate demonstrated overt hemospermia. Urethroscopy demonstrated a second urethral rent, parallel to the one observed the previous season. The rent was associated with an elevated area of tissue suggestive of corpus spongiosum prolapse into the urethra. Several options were presented to the owner, including subischial urethrostomy, which was declined. To manage the stallion medically, sexual rest was implemented for 9 days, after which semen was collected. Overt hemospermia was observed (Fig. 7). The semen was centrifuged with the use of density gradient centrifugation; however, too few spermatozoa for cryopreservation were recovered with the use of this technique. After another week of sexual rest, semen was collected 2 hours after administration of imipramine and minimal teasing to reduce the duration of corpus spongiosum pressure on the rent; hemospermia occurred. The horse had several observations of hematuria and blood dripping from the penis in the stall. Three attempts to induce ex copula ejaculation were unsuccessful. Currently, the horse is still under the care of our clinic and scheduled for examination after 10 months of sexual rest. Hematuria has not been recently observed.

Case 8

An 8-year-old Appaloosa stallion presented for semen collection and cryopreservation. Semen had been previously collected from this horse (4 years old) for cryopreservation at our clinic. The horse had an unremarkable health history and the physical examination was within normal limits. While being washed for semen collection, the horse kicked the penis with his left hind leg. On gross inspection, collected semen had hemospermia. The penis did not demonstrate any swelling or continued hemorrhage. After 2 days of sexual rest, semen was again collected and was not contaminated with erythrocytes. The horse resumed a normal collection schedule, with no further hemospermia.

5. Discussion

A common complaint in breeding stallions, hemospermia is detrimental to fertility and can result in considerable financial losses as well as reduced welfare of affected animals. The ability to diagnose hemospermia in breeding stallions is of significant importance to the equine practitioner.

Several breeds are over-represented in diagnosed hemospermia cases. A report of 18 cases demonstrated 15 Quarter Horses, three Appaloosas, and one Arabian.[1] Three cases of hemospermia caused by urethral rent were found in two Quarter Horses and one Appaloosa.[29] Buccal urethroplasty was performed in a Quarter Horse.[32] The breeds represented in our study were very similar to those previous studies (four Quarter Horses, two Appaloosas, and two Arabians). The mean age at presentation in our study (10.1 years; range, 6–20) was comparable to that in the study by Sullins et al[1] (7.1 years; range, 3–18).

There are many etiologies to hemospermia. Often, the history includes a change in collection phantom height or angle. This may change the pressure on the horse during collection and predispose to hemospermia. One example is the horse in Case 7, which demonstrated hemospermia when collected on the farm but less often when collected at the clinic. This may also be a feature of Case 4 and demonstrates that variation in washing technique, stallion handling, and the phantom may result in intermittent hemospermia that spontaneously resolves. The practitioner must be astute in examination of the horse to identify external lesions, such as urethral process lesions, habronemiasis, equine herpesvirus-3, SCC, or blunt-force trauma. In the absence of external lesions, the practitioner must be educated in the practice of or have means to refer the horse to a specialist for advanced diagnostics, including urethroscopy. In the current case series of eight affected stallions, diagnoses included urethral rents ($n = 3$), urethral process lesions ($n = 2$), squamous cell carcinoma ($n = 1$), kick to the penis ($n = 1$), and unknown origin ($n = 1$). Sexual rest returned four horses to breeding; one was castrated; one was not treated; and one is still under manage-

ment by our service. These diagnoses are in contrast to Sullins et al,[1] who diagnosed 18 cases of hemospermia caused by urethral rent ($n = 1$), urethritis ($n = 11$), urethral varicosities ($n = 4$), lymphosarcoma ($n = 1$), and unknown origin ($n = 1$). Of those 18 cases, 15 were treated by subishcial urethrostomy. Of three treated with sexual rest, one died (lymphosarcoma) and two returned to breeding function.[1] Of three stallions diagnosed with urethral rents, one returned to breeding function after 11 months of sexual rest, one was castrated 6 weeks after urethrostomy when hemospermia recurred, and one resumed breeding function 10 weeks after urethrostomy.[29]

If surgical options are not elected to manage hemospermia, whether caused by urethral rent or SCC, the practitioner must be prepared to manage affected horses medically. In our clinic, one stallion, for which subischial urethrostomy was repeatedly declined, was treated medially by use of imipramine to lower the ejaculatory threshold before semen collection. In two stallions, semen collection was attempted with minimal washing and teasing time to lower the risk of hemorrhage from erectile tissues. In six of the eight stallions (75%) presented here, semen collection was attempted ex copula. The one successful ejaculate was erythrocyte-contaminated. Ex copula ejaculation allows for collection of concentrated semen without high corpus spongiousum pressures, although stallions with urethral rents may still bleed, as in Case 5. Although the technique was not successful in the majority of our cases, it is a technique that we perform regularly in our clinic on debilitated stallions for genetic preservation.

If hemospermia is unable to be managed medically, ejaculates can be manipulated to increase pregnancy rates. Semen from one stallion in our study was density-gradient centrifuged to obtain uncontaminated semen. It has been demonstrated that placing an extender in the uterus before live-cover mating may counteract some of the negative effects of erythrocytes on spermatozoa. Semen from stallions with seminal vesiculitis can be extended with antibiotic-containing extender or placement of that extender in the mare.[37] Collection of semen in fractions may help to improve fertility if only erythrocyte-free jets are processed for artificial insemination.

The effects of hemospermia on fertility have been documented.[27] The presence of blood in the ejaculate has not been shown to affect spermatozoa morphology; however, in one study, the progressive motility and plasma membrane integrity were lower in semen with 10% or 20% added whole blood compared with unaffected ejaculates.[39] It was noted in that study that spermatozoa appeared to accumulate around the erythrocytes. In another study, semen with 20% added whole blood resulted in a per-cycle pregnancy rate of 7.7%, whereas per-cycle pregnancy rates with the use of unaffected semen or semen with 20% added serum were 28.6% and 50%, respectively.[27] These studies demonstrate that the infertility observed in hemospermia cases are the result of the erythrocyte presence in the ejaculate and not the presence of serum, although the mechanism whereby this infertility occurs is still unknown and warrants further investigation.

Despite the medical and surgical techniques available to practitioners to manage stallions with hemospermia, no technique is 100% effective. To ensure acceptable fertility from breeding stallions, practitioners must educate stallion owners that collection and cryopreservation of semen from stallions when the horses are young and healthy is the best guarantee against future financial losses caused by stallion debilitation or death, including hemospermia. In one of our cases, the horse was managed medically for urethral rent during the breeding season, yet mares were still able to be bred through the use of previously frozen semen, preventing the farm from losing a foal crop.

6. Summary

Hemospermia can be devastating to a stallion's fertility and welfare. The prognosis for recovery of breeding function and life are dependent on the diagnosis. Practitioners who work with breeding stallions should educate stallion owners on the importance of routine breeding soundness examinations at the beginning of each breeding season and routine collection and semen cryopreservation of healthy stallions. These practices will help to identify any pathology early, which can result in higher treatment success and survival rates, especially for SCC. Practitioners who diagnose hemospermia in breeding stallions but are unable to manage the horse should actively seek referral to a specialist.

Acknowledgments

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Prevention of Equine Herpes Myeloencephalopathy by Vaccination: A Pilot Study

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Immunization of horses with a high-antigen vaccine for equine herpes virus 1 decreased the clinical signs of equine herpes myeloencephalopathy, although the differences were not statistically significant. More definitive study of the ability of equine herpes virus 1 vaccination to protect horses from equine herpes myeloencephalopathy is warranted. Authors' addresses: Departments of Physiological Sciences, 264 McIlroy Hall (Maxwell, McFarlane), Veterinary Clinical Sciences, Room 0026 BVMTH (Gilliam, Holbrook), Veterinary Pathobiology, 250 McIlroy Hall (Eberle, Snider), and Oklahoma Animal Disease Diagnostic Laboratory (Rezabek), Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK 74078; e-mail: lk.maxwell@okstate.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The neuropathogenic form of equine herpes virus 1 (EHV-1) has become an increasingly important problem for the equine industry. The ability of vaccination to protect horses from equine herpes myeloencephalopathy is currently unknown because the disease has occurred even in well-vaccinated horses.

2. Materials and Methods

Twelve aged mares (>20 years old) were randomized to either saline control or vaccination groups. Three intramuscular injections with a high antigen vaccine[a] were administered at monthly intervals, followed by inoculation with a neuropathogenic strain of EHV-1. Ataxia scores, rectal temperatures, and clinical scores were determined.

3. Results

Control horses had more severe neurological signs, with five of six control horses versus one of six vaccinated horses progressing to at least a two-grade change in ataxia ($P = 0.08$). Mean body temperature was lower during the biphasic fevers occurring on days 2 and 5 in vaccinates ($P = 0.1$). The median clinical score was also 20% lower in vaccinates as compared with control horses ($P = 0.3$).

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4. Discussion

Immunization of horses with a high-antigen EHV-1 vaccine decreased clinical signs of infection when challenged with a neuropathogenic strain of EHV-1, although differences were not statistically significant. A larger number of horses would be needed for conclusive evidence of the ability of vaccination to protect horses from equine herpes myeloencephalopathy. However, the present pilot study provides justification for a more extensive and definitive study.

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Footnote

[a]Pneumabort-K®, Zoetis Animal Health, Kalamazoo, MI 49007.

Effect of Non-Steroidal Anti-Inflammatory Treatment at the Time of Vaccination

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Administration of a non-steroidal anti-inflammatory drug (NSAID) at the time of vaccination can impair the immune response to the vaccine. Authors' addresses: College of Veterinary Medicine, Michigan State University, East Lansing, MI 48824-1314 (Zoll); Maxwell Gluck Equine Research Center, University of Kentucky, Lexington, KY 40546 (Horohov, Page, Chambers, Betancourt, Stewart); e-mail: dwhoro2@uky.edu. *Corresponding author; †presenting author. © 2013 AAEP.

I. Introduction

Whereas horses are routinely vaccinated to prevent infectious diseases, adverse reactions remain a concern. The co-administration of a non-steroidal anti-inflammatory drug (NSAID) with vaccination is sometimes used to reduce the risk of an adverse reaction. Although the reasoning behind this may seem sound, such treatments could affect the ability of the horse to respond to the vaccine. We assessed the effect of treating horses with an NSAID on the response to a commercially available equine influenza vaccine.

2. Materials and Methods

Eighteen adult horses and 18 equine influenza–naive yearlings were used in this study. The horses were assigned to one of the following three groups containing six horses: NSAID treatment and vaccination; no NSAID treatment and vaccination; and no NSAID treatment and no vaccination. Blood samples were collected before the initial vaccination and on 7, 14, 21, and 28 days after vaccination for the determination of equine influenza–specific antibodies by enzyme-linked immunoassay (ELISA) and hemagglutination inhibition (HI) and cell-mediated immune responses.

3. Results

The use of the NSAID resulted in a significant ($P < 0.05$) decrease in the antibody response to the vaccine as measured by HI antibodies and ELISA. Likewise, there was a reduced cellular immune response to the NSAID-treated group.

4. Conclusions

The administration of an NSAID at the time of vaccination reduced both the antibody and the cellular immune response to the vaccine. This occurred in both previously immune and naive individuals.

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How to Evaluate the Equine Hoof Capsule

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1. Introduction

The hoof capsule is comprised of the hoof wall, sole, frog, and bulbs of the heels, which, through the unique continuous bond between its components, form a casing on the ground surface of the limb that affords protection to the soft tissue and osseous structures enclosed within the capsule.[1] The hoof wall is a viscoelastic structure that has the ability to deform under load and then return to its original shape when the weight is removed. It is well accepted that abnormal weight distribution on the foot or disproportionate forces placed on a section of the hoof will, over time, cause it to assume an abnormal shape.[1–4] These abnormal stresses within the foot will also predispose the foot to injury or disease. Increased stress or weight-bearing placed on a section of the hoof capsule may originate from a single source or it may be from multiple contributing factors such as abnormal limb conformation, strike pattern, amount of work, type of footing, and inappropriate farrier practices. Excess stress placed on one section of the hoof capsule can manifest itself in a variety of ways, such as compressed growth rings, flares or under-running of the hoof wall, dorsal migration of the heels, and either focal or diffuse displacement of the coronary band.[6,7] Distortion of the hoof capsule of the forelimbs appears to be related to limb alignment and load, whereas deformation in the hind feet appears to be different and related to propulsion. Because the hoof capsule distortion of the forelimbs is commonly associated with lameness and various disease processes, only the forelimbs will be considered in this report. Because the "normal" foot has never been defined, each view will begin with what is perceived to be an ideal, good, or healthy foot.[1,8] Palpation of the hoof capsule often complements the visual examination, and the areas where palpation is relevant will be included. The goal of evaluating the hoof capsule is to identify deformation and changes in growth pattern that indicate abnormal distribution of forces (stresses) on the foot. Because hoof capsule distortion and abnormal loading usually accompany lameness, farriery will form part of or sometimes the entire treatment. Farriery is used to help redistribute the load and help improve or resolve the hoof capsule deformation.

2. Mechanism of Distortion

Evaluation of the hoof capsule morphology will indicate where the hoof wall is unduly stressed; however, the evaluation must be coupled with an understanding of the abnormal distribution of forces that lead to hoof capsule deformation. Understanding the biomechanical forces leading to hoof capsule distortions is also helpful for the clinician in applying the appropriate farriery to modify these stresses. There are many excellent reviews of basic

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biomechanics of the hoof in the veterinary literature.[1–5] Increased load or weight-bearing by a portion of the wall has three consequences: (1) it may cause deviation of the wall outward (flares) or inward (under-running) from its normal position; (2) it may cause the wall to move proximally; or (3) it may decrease hoof wall growth. A reduction in load or weight-bearing generally has the opposite effect. Briefly, in the standing horse, the weight of the horse borne by the limb is supported by the ground, which opposes the weight with an equal and opposite force. The force exerted on the foot by the ground is termed the ground reaction force. The term center of pressure (COP) is the point on the ground surface of the foot through which the ground reaction force acts on the foot. The center of pressure varies among horses but is approximately located in the center of the solar surface of the foot in the standing horse. However, when the horse is moving, the location of the COP changes dynamically. The position of the COP at any point in the stride determines the distribution of forces between the medial and lateral and the dorsal palmar aspects of the foot. When the center of pressure is moved to one side of the foot, that side of the foot will be subject to increased forces. If the COP is moved in a palmar direction, the weight-bearing or load on the palmar hoof wall is increased. Relating this to hoof capsule distortions, if the COP is located more medially, over time, a medial hoof wall flare (bending) and a lateral under-running will develop. Or, if the COP is located more dorsally because of increased tension in the deep digital flexor tendon, the hoof capsule will develop a higher heel with a flare in the dorsal hoof wall. Farriery is used to change the location of the center of pressure (to some extent) and change the distribution of forces on the ground surface of the foot.

3. Limb Conformation

When evaluating hoof capsule deformation, limb conformation should be considered. Abnormal limb conformation affects the landing pattern and stance phase of the stride. Few horses have ideal limb conformation, and any change in conformation is going to change the distribution of forces within the hoof capsule, leading to deformation. In the frontal plane, the forelimbs should be of equal length and size and bear equal weight. A line dropped from the scapulohumeral joint to the ground should bisect the limb. Certain types of abnormal limb conformation have been described.[9] In the frontal plane, abnormal conformation is described as valgus (the limb's segment distal to the affected joint will deviate laterally) or varus (the distal segment of the limb will deviate medially). The joint most often affected is the carpus, and, to a lesser degree, the metacarpalphalangeal joint. Here, there will be excess load placed on the hoof opposite the direction of the deviation. If a line dropped from the metacarpalphalangeal joint through the digit to the ground



Fig. 1. Distal phalanx within hoof capsule will be offset laterally. Coronary band will be displaced proximally on media quarter/heel.

does not bisect the hoof capsule, the foot is considered offset to one side (usually laterally) and therefore increased load is placed on the opposite side of the foot (Fig. 1). In the transverse plane, conformation abnormalities are characterized by axial rotations of the limb or its segments, either laterally or medially. For example, a horse with a narrow chest and a lateral axial rotation will land on the lateral side of the hoof and then load the medial side resulting in proximal displacement of the quarter /heel on the medial side and causing the hoof deformation termed "sheared heels"[10,11] (Fig. 2). A limb with a medial (inward) rotation of the digit relative to the third metacarpal bone (toed-in) will develop a hoof with a diagonal asymmetry, with a narrow lateral toe and medial heel and a wide medial toe and



Fig. 2. Horse with one heel bulb displaced proximally (sheared heel conformation). Note the contour of the pastern above the displaced heel bulb.

lateral heel. The altered distribution of forces leading to hoof capsule deformations follow a logical pattern in which the overloaded sections of the hoof are less developed and the under-loaded sections are overdeveloped. In the sagittal plane, abnormal conformation can best be described by the position of the distal interphalangeal joint (DIP), either a flexural deformity or marked dorsiflexion (ie, extension) of the joint. The shape or conformation of the hoof in the sagittal plane will be dependent on the tension in the deep digital flexor tendon, the integrity of the laminar apparatus, and the digital cushion—all of which determine the angle of the solar margin of the distal phalanx. A flexural deformity will overload the toe, whereas marked dorsiflexion of the DIP joint will overload the palmar section of the foot.

4. Evaluation of the Hoof Capsule

A detailed morphological examination of the foot should begin with observing the horse in motion, both going away from and toward the examiner, on a firm, flat surface to note the landing pattern. The foot is then viewed from all sides while it is on the ground. Finally, the ground surface is examined with the foot off the ground. Additionally, small changes in the shape of the hoof capsule (such as the coronet and the digital cushion) may be better appreciated by careful palpation of the foot than by visual inspection.

5. Dorsal Aspect

When the foot is viewed from the dorsal aspect, the ideal hoof should be approximately symmetrical. An imaginary line drawn between any two comparable points on the coronary band should be parallel to the ground. The medial wall should be the same height as the lateral wall, but because it is often slightly steeper, it may be slightly shorter. An imaginary line that bisects the third metacarpal should bisect a line drawn between any two comparable points on the coronary band or the ground surface of the hoof. Similarly, the hoof should be symmetrically related to the distal limb such that an imaginary line that bisects the third metacarpal bone, bisects the pastern and the hoof, allowing for the slight asymmetry caused by the different angles of the medial and lateral wall (Fig. 3).[1] When the foot is viewed from the dorsal aspect, the shape of the forefeet may be asymmetrical, with one hoof being narrower than the other ("mis-matched feet"). Several abnormalities may be visible at the toe/quarters such as flares or under-running of the wall. The coronary band may be unevenly distributed, most commonly by an uneven slope from one side of the foot to the other. A distally directed arch in the coronary band in the dorsal portion of the foot usually indicates extensive remodeling of the distal phalanx at the toe. Examination of the growth rings below the coronet may show divergence of the rings from one side to the other indicating uneven or excessive load (Fig. 4). The angulation of the dorsal horn tubules toward the medial or lateral side of the hoof capsule should be noted; normally they are parallel, so when they appear tilted medially or laterally, it suggests that the whole hoof capsule may be tilted in that direction (Fig. 5). The position of where the pastern bisects the hoof capsule should be noted, that is, is its entry on the midline or displaced medially or laterally?

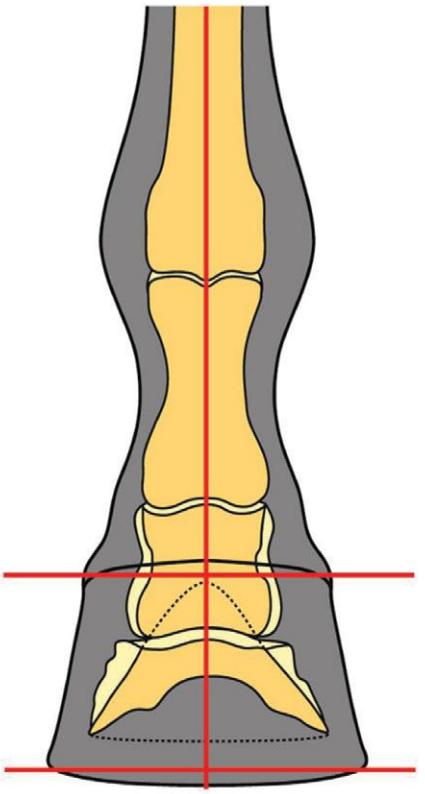


Fig. 3. Dorsal view: the hoof should be symmetrical. A line drawn between any two comparable points on the coronary band should be parallel to the ground. The hoof should be symmetrically related to the distal limb such that a vertical line should bisect the third metacarpal bone, the pastern, and the hoof (courtesy of Dr Andy Parks).

On palpation, the coronary band of a healthy hoof should feel thick and spongy. There should be no evidence of a "ledge" or "trough" behind the proximal margin of the hoof capsule when palpated. A depression in the coronary band indicates that the distal phalanx has displaced within the hoof capsule, a finding that can be present in sound horses.[12] This palpable depression will generally be accompanied by a thin, flat sole, narrow frog, and contracted heels. The dorsal aspect of the coronary band should also be palpated for effusion of the DIP joint. This is often seen with horses that have a broken back hoof–pastern axis.

6. Lateral Aspect

When viewed from the lateral aspect, the angle the dorsal hoof wall forms with the ground is variable



Fig. 4. Note divulgence of growth rings below the coronet from the lateral to medial side.

and typically related to the conformation of the digit. The heel tubules of the hoof capsule should form an angle with the weight bearing surface similar to the angle of the horn tubules in the toe region. Tradition has it that the angle of the wall at the heel should match that of the dorsal hoof wall; however, it is usually a few degrees less. As the foot accepts weight, it expands and the ground surface at the heels moves against the shoe, causing wear that decreases the heel angle. The amount of wear is dependent on the integrity of the structures in the heel. The length of the dorsal hoof wall is similarly variable but is determined by the amount of sole depth present. There are two guidelines that relate the proportion of the foot to the rest of the distal limb. First, the foot-pastern axis describes the relationship between the angles made by the dorsal hoof wall and the dorsal aspect of the pastern with the ground. Ideally, the dorsal hoof wall and the pastern form the same angle with the ground so that



Fig. 5. Hoof with a separation in the dorsal hoof wall. Note the angulation of the horn tubules toward the medial side of the foot. Also note the focal "arch" in the coronet on the lateral side.

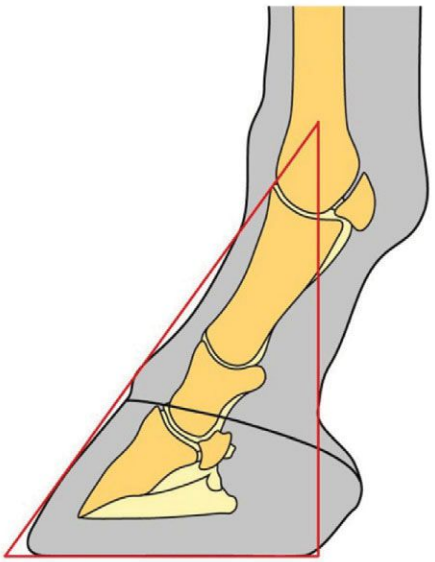


Fig. 6. Conformation of the foot as it relates to the digit can be depicted with a triangle. A line drawn down the dorsal surface of the pastern and hoof is the hoof-pastern axis. A vertical line that bisects the third metacarpal bone should intersect the ground at the palmar aspect of the heels of the hoof capsule. Connect these two lines with the angle of the dorsal hoof wall and the ground surface of the foot to form a triangle (courtesy of Dr Andy Parks).

the angle between them is 180° and the axis is considered straight. Second, an imaginary line that bisects the third metacarpal should intersect the ground at the most palmar aspect of the ground surface of the hoof. These two guidelines used in conjunction with the angle of the dorsal hoof wall and the ground surface of the foot combine to form a triangle of proportions that represents the relationship between the hoof and the distal limb regardless of the size of the horse (Fig. 6).[1] Evaluation of the hoof capsule from the side view should begin with the coronet as this structure can provide very useful information. The healthy coronary band should have a gentle, even slope from the toe to the heels, and the hair should lie flat against the hoof capsule; hair projecting horizontally may indicate excessive forces on the associated hoof wall.[13] The coronary band is dynamic, and its shape can be affected by chronic overloading.[14] A proximally directed diffuse arch at the quarters or a focal directed arch toward the heels is evidence of chronic overloading of that section of the foot (Fig. 7). A coronary band with an acute angle at the heels relative to the ground that bends distally at the heel to form a "knob" appearance is an indication that the foot has poorly developed or under-run heels and the hoof wall at the heels has migrated dorsally (Fig. 8). A coronary band that is horizontal relative to the ground and often accompanied by a flare in the dorsal hoof wall would denote an upright or clubfoot conformation (Fig. 9). Asymmetry of the height of



Fig. 7. A section of the coronet in the palmar section of the foot has been displaced proximally (focal arch). Note the relationship of the heel of the shoe and the origin of the defect, which denotes excessive load. The foot on the right shows a change in angulation of the horn tubules, curvature of the growth rings, and a proximal displacement of the coronet (dorsal arch).



Fig. 9. Clubfoot. Note the coronary band has lost the slope and is almost parallel with the ground. Also note the flare in the dorsal hoof wall.

the coronary band in the quarter/heel region on one side occurs when the horse develops a "sheared heel," a hoof capsule distortion resulting in proximal displacement of one quarter/heel bulb relative to the contralateral side of the foot.[15] The medial heel bulb/quarter is more commonly displaced proximally, as it is more common for the foot to be offset laterally. The angle of the coronary band can be used to estimate the position of the distal phalanx within the hoof capsule. One study described the angle of the coronary band of apparently normal front feet to be 23.5° ± 3°.[16] If the angle of the coronary band is >45°, the plane of the solar margin of the distal phalanx will decrease. At the other extreme, a coronary band parallel to the ground is indicative of a high palmar angle, which is often associated with a club foot or rotation of the distal phalanx. The width of the growth rings below the coronet should be equal from toe to heel. A disparity in the width of the growth rings between the toe and the heels is indicative of non-uniform circulation of the coronary corium or excessive forces below because wall growth is generally inversely related to load. An example of this disparity would be chronic laminitis typified by more horn growth at the heels than toe growth. However, regional irregularity in spacing of growth rings is not uncommon; the most frequently observed is a decrease in spacing at the quarter associated with proximal displacement of the coronary band as noted with sheared heels.[10] The angulation of the horn tubules from dorsal to palmar should be noted because horn tubules that are parallel with the ground in the heel area are associated with under-run heels. Flaring or concavity of the dorsal hoof wall accompanied by underrunning of the heels is readily appreciated from the lateral side. The presence of hoof wall flares or cracks are often caused by chronic, excessive overloading of the hoof wall in the region in which these defects are found.[10,11,14,17] Vertical cracks in the quarter are more likely to occur with a sheared heel. Horizontal cracks are usually the result of a disruption of production of horn caused by coronary band trauma or when a subsolar infection ruptures at the coronary band.



Fig. 8. Foot with under-run heels showing the "knob" appearance. Note the curve in the growth rings as the heels migrate dorsally. There will often be a depression ("thumbprint") showing the extent the heels of the hoof capsule have migrated forward (arrow). Palmar view shows the decrease in structural mass of the digital cushion.

7. Palmar Aspect

The heels are evaluated from the palmar aspect for their overall width and height. The heels frequently become narrowed when the foot itself is narrow. Additionally, the central sulcus of the frog may extend proximal to the hairline so that a cleft becomes apparent in the skin of the pastern between the heels. The overall height of the heels is readily assessed from the lateral aspect, but viewing from the palmar aspect is useful to compare the relative heights of the two heels. For example, in the case of the sheared heel, one heel is displaced proximally in relation to the other. Another example is mismatched feet in which there is a marked disparity in heel height. The contour of the junction of the heel bulbs with the skin can be evaluated relative to the width of the hoof wall at the heels and the thickness of the digital cushion (Fig. 10).

8. Distal or Solar Aspect

When viewed from the distal surface, the ground surface of the foot should be approximately as wide as it is long. The foot should be approximately



Fig. 10. A, Illustration shows various contours of the junction of the heel bulbs with the skin; B, wide heel; C, contracted heel with a poor digital cushion. Note also the sheared heel on this foot.

symmetrical about the long axis of the frog; the lateral side of the sole frequently has a slightly greater surface area that corresponds with the difference in wall angles at the quarters described in the dorsal view. The width of the frog should be approximately 60% to 70% of its length. The ground surface of the heels should not project dorsal to the base of the frog. Imaginary lines drawn across the most palmar weight-bearing surface of the heels and across the heel bulbs at the coronary band should be parallel and both lines should be perpendicular to the axis of the frog (Fig. 11).[1] If a three-dimensional object such as the foot changes in one plane, it will change in at least one other plane. Therefore, examination of the ground surface of the foot reveals much about the changes in the wall of the hoof capsule. For example, if the contour of the wall is displaced away or toward the median plane in the dorsal two thirds of the foot, this usually corresponds with a flare or under-running of the wall, respectively. If only one heel buttress is displaced dorsally in relation to the base of the frog, it usually corresponds with the proximal displacement of that heel plus or minus the quarter termed sheared heel.

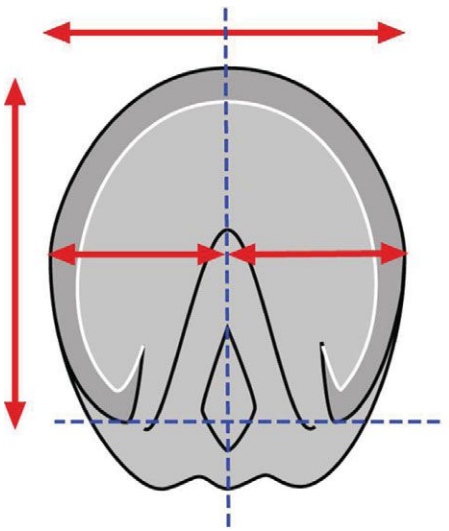


Fig. 11. The ground surface of the foot should be approximately as wide as it is long (red lines) and approximately symmetric about the long axis of the frog (blue lines). The heels should not project dorsal to the frog (courtesy of Dr Andy Parks).

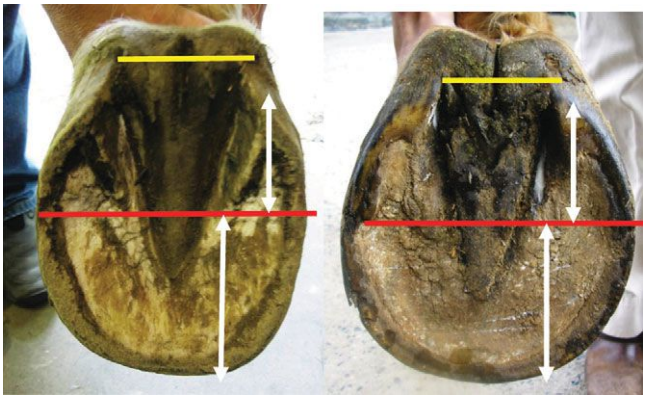


Fig. 12. Two examples of a line drawn across the middle or widest part of the foot to determine proportionality (red line) by use of the base of the frog (yellow line) and the perimeter of the toe. White lines are the distance from the middle of the foot to the end of the heel and to the toe, respectively. Note that both heels have migrated dorsally and both frogs are receded below the hoof wall.

The author begins the evaluation of the solar surface of the hoof capsule by drawing a line across the widest part of the foot. This line forms a consistent landmark and is located just dorsal to the center of rotation (of the distal interphalangeal joint). With the use of this line as a starting point, there should be approximate proportions from this line to the perimeter of the toe and to the base of the frog (Fig. 12). This creates a relative proportion from the front of the foot to the palmar aspect that is related to the alignment of the center of rotation in the middle of the foot or, when shod, the middle of the shoe. The normal solar surface of the foot may be wider laterally than medially. The width of a healthy frog should equal 60% to 70% of its length; therefore, the width and length of the frog should be critically evaluated using these guidelines.[1,8,18] The untrimmed frog should be on the same plane with the hoof wall at the heels; it should not be receded between the hoof wall or protrude beyond the solar surface of the hoof wall. In general, the frog is usually constant in length, and its axis is almost always aligned with the medial plane of the foot but its width is variable. As the frog functions as an expansion joint, a decrease in width is generally associated with contracture of the hoof capsule at the heels. The frog of a healthy hoof has sufficient depth at its dorsal aspect to reach the bearing surface.[17] The relationship of the untrimmed frog to the sole indicates the position of the distal phalanx within the hoof capsule (ie, the angle of the solar margin of the distal phalanx).[18] For instance, if the apex of the frog is deeply recessed and the frog appears to be angling toward the coronary band at the toe, the distal phalanx is probably similarly positioned, creating a negative palmar angle. The position of

the heels of the hoof wall relative to the base of the frog should be evaluated. Ideally, the most palmar extent of the bearing surface of the heel tubules would be at the base of the frog and very near a vertical line drawn thru the middle of the third metacarpal bone. When the ground surface of the heels is dorsal to the base of the frog, the heels are low, under-run and/or increased in length. The structures of the heel (hoof wall, buttress, angle of sole, bars) should be present and well-defined. If the heels have migrated dorsally relative to the frog and the structures are present, the heels are considered low; if the structures of the heel are absent or damaged, then the heels are considered under-run. The bars of the heel should be straight as curvature indicates contracted heels. The proportionality of the foot dorsal to the widest part of the foot should be evaluated. As the heels move forward, there will generally be a substantial increase in the proportion of the toe relative to the heels. A long toe will also be accompanied by an increased distance between the toe and the apex of the frog. The sole-wall junction should be solid and compact. Widening or fissures in the sole-wall junction and hoof wall separations dorsal to the sole wall junction occur with lengthening of the toe. The healthy sole tends to be concave and callused adjacent to the sole wall junction (white line). It should have a gradual slope from the apex of the frog to the sole wall junction and not a significant "trough." The sole should be between 10 to 15 mm thick beneath the margin of the distal phalanx and should not deform when hoof testers are applied. A ruler calibrated in millimeters can be placed within the collateral groove of the frog to measure the distance between the deepest part of the groove and the plane of the solar surface of the foot. The consistent distance (10 to 11 mm) between the distal phalanx and the collateral groove depth at the apex of the frog allows the clinician to predict sole depth. If one imagines moving the ground plane proximally so that the distance from the ground plane to the depth of the collateral groove decreases, it becomes clear that sole depth decreases as collateral groove depth decreases[13] (Fig. 13).

Horses with poor heel structure typically have a poorly developed digital cushion and thin collateral cartilages. These soft tissue structures determine the overall conformation of the palmar portion of the foot. Clinicians should gain an appreciation for variation in the consistency and overall size of the digital cushion and collateral cartilages. The digital cushion can be palpated between a thumb placed between collateral cartilages and the fingertips placed on base of the frog. A sense of "normal" can be acquired by palpating the digital cushions of sound horses with "good feet" and comparing those findings with those of horses with poorly conformed feet. The depth of the combined tissues of normal digital cushion and frog should be approximately 2 inches, but this can vary among different breeds.[13]



Fig. 13. A rasp is placed across the foot and a ruler is placed within the collateral groove of the frog to measure the distance between the deepest part of the groove and the plane of the solar surface of the foot. This simple technique can be used to approximate the sole depth (courtesy of Dr John Schumacher).

Horses with under-developed digital cushions typically have low or under-run heels that lack stability and can be easily distracted independently or they may have contracted heels and narrow, non–weight-bearing frogs.[19,20]

9. Conclusions

The clinical examination of the equine foot has been well-described and is generally performed in lameness cases. Evaluation of the hoof capsule during the lameness examination will provide additional information as to the etiology and treatment of the lameness but will also serve as a guideline to apply therapeutic farriery and other preventive measures to maintain a healthy hoof. The morphology of the hoof capsule reveals deformation and changes in growth that occurs after increased or reduced force. The relationship between the limb and the foot indicate conformations that predisposes the foot to abnormal weight-bearing. Inversely, with the use of abnormal distribution of forces and the subsequent hoof capsule distortion as a template, appropriate farriery or therapeutic farriery will form at least part of the treatment plan. Here it is essential to be familiar with the biomechanics of the foot and how these forces can be altered to change the distribution of forces or the focal stresses on a given section of the foot.

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How to Obtain Useful Information From a Grossly Blood-Contaminated Synovial Fluid Sample

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1. Introduction

Synovial fluid examination in the horse is a routine procedure in equine practice. One of the most valuable uses of synovial fluid (SF) analysis is to determine the presence or absence of infection within a synovial structure.[1–3]

Normal equine SF is clear to straw-colored and highly viscous. It is an ultrafiltrate of plasma, therefore similar concentrations of glucose and electrolytes are expected.[3] The total protein (TP) concentration and white blood cell count (WBC), however, are much lower, and the prevailing cell type is mononuclear. SF should contain very few to no erythrocytes. The reported normal values of SF WBC range from <300 to 1000 cells per microliter, containing mainly mononuclear cells with <10% neutrophils on cytologic examination.[3–7] Normal TP concentrations have been variably described as <25% to 35% of the systemic plasma TP and <0.8 to 2.5 g/dL.[3,4]

Values of WBC and TP above 10×10^9/L and 50 g/L, respectively, with >80% neutrophils, are highly suggestive of septic arthritis; however, lower levels do not necessarily rule this out.[3] The definitive diagnosis of a septic synovial structure of bacterial origin is a positive culture of the SF and/or synovial membrane.[5]

Many instances occur in equine practice, in which clients are faced with the difficult decision of whether or not to initiate aggressive treatment because of economic constraints. Unfortunately, it is not uncommon for an equine practitioner to be unable to provide a definitive diagnosis of septic arthritis for multiple reasons, including an iatrogenically blood contaminated sample. This report shows how to obtain diagnostically useful synovial fluid TP and WBC values from a blood-contaminated sample, given a concurrent complete blood count.

2. Materials and Methods

To be able to compute useful WBC counts and TP values from a blood contaminated synovial sample, one must obtain both a synovial fluid sample and a peripheral venous blood sample (usually by means of the jugular vein).

The hematocrit (HCT) and WBC counts of both the synovial fluid and blood sample are obtained, as well as the TP concentration of the synovial fluid in the laboratory.

NOTES

Table 1. Formula for Calculation of Uncontaminated Total Protein From a Blood-Contaminated Synovial Fluid Sample

Laboratory Values	Horse Name/Number
Synovial WBC, $*10^9$/L	3.49
Synovial TP, g/L	2.90
Blood WBC, $*10^9$/L	9.92
Blood TP, g/L	7.00
Synovial hematocrit, %	11.10
Blood hematocrit, %	30.50
Calculated values	
Uncontaminated TP, g/L	1.25
Uncontaminated WBC, $*10^9$/L	0.78

A ratio of HCT must be calculated to determine the degree of contamination for the synovial fluid sample.

$$\text{Ratio HCT} = \text{HCT}_{\text{SF}}/\text{HCT}_{\text{Blood}} \quad (1)$$

The difference between the values obtained from the contaminated synovial fluid sample and those that would have been obtained in a completely blood-free sample is then calculated as:

$$\text{TP}_{\text{DIFF}} = -0.063 - 0.0542\ (\text{HCT}_{\text{SF}}) + 6.34(\text{RatioHCT}) \quad (2)$$

$$\text{WBC}_{\text{DIFF}} = -0.01916 + 0.01373\ \text{WBC}_{\text{Blood}} + 0.08689\ \text{HCT}_{\text{SF}} + 0.01479(\text{WBC}_{\text{Blood}} * \text{HCT}_{\text{SF}}) \quad (3)$$

As TP_{DIFF} = TP (contaminated SF sample)−TP (theoretical blood-free sample), simple substitution is used to calculate the blood-free value for this variable. Similarly, WBC_{DIFF} = WBC (contaminated SF sample)−WBC (theoretical blood-free sample) can be used to calculate the blood-free synovial fluid WBC count.

This process seems cumbersome; however, in the authors' practice, a Microsoft Excel spreadsheet has been constructed such that entering the laboratory values will automatically generate blood-free–calculated values without having to resort to using a calculator every time (Tables 1 and 2).

3. Discussion

The mathematical models used above were generated in a two-part study.[8] Briefly, peripheral venous blood and SF was obtained from 10 adult horses. The SF samples were split (into six) and subsequently contaminated with the use of autologous blood added in 10% increments from 0% to 50%. In some horses, autologous plasma was also added to artificially raise the baseline total protein concentration. An automated cell count was obtained followed by direct smears in both the SF and venous blood samples. The packed cell volume (PCV) and the TP concentration were measured with a hand-held refractometer after sample centrifugation. Dummy variables (RatioHCT, WBC_{DIFF}, TP_{DIFF}, and NP_{DIFF}) were created as the difference between the variable count of a sample with a known percentage of contamination and one with 0% contamination. With the use of a combination of best-subset regression and linear regression techniques, mathematical models were generated that accounted for the change in TP concentration and WBC counts as a function of contamination. The models were then tested in five additional horses. Samples were contaminated by one author and the results presented to another author in blinded fashion. When noncontaminated TP and WBC counts were calculated and compared statistically with actual measured values, there was no significant difference ($P = 0.3$ and $P = 0.53$, respectively), despite calculated TP concentrations being 0.14 g/L higher and the calculated WBC count being $0.07*10^9$/L lower than measured.

In the original research, several interesting observations were made. First, it took almost 50% blood contamination (of normal synovial fluid) to raise the synovial fluid TP concentration toward the reported septic "cut-off value" of 2.5 g/dL. Adding blood also raised the neutrophil percentage; however, after the initial addition, further contamination did not change the percentage significantly and thus a "corrected" neutrophil percentage could not be calculated. Second, even in the most heavily contaminated samples, maximum values for synovial TP and WBC were 3.23 g/dL and $5.03 * 10^9$/L, respectively. This would indicate that even in blood-contaminated samples, a total WBC and TP above 20×10^9/L and 4 g/dL, respectively, would be highly suggestive of a septic process. Finally, our opinion that it is unlikely that SF harvested in a clinical situation would be submitted for subsequent laboratory analysis if >10% contaminated because at this point the sample is visually indistinguishable from blood.

The methods presented above are used clinically to calculate noncontaminated SF TP concentration and WBC counts from blood-contaminated samples. However, diagnosing a potential septic arthritis is

Table 2. Formula for Calculation of Uncontaminated White Blood Cell Count From a Blood-Contaminated Synovial Fluid Sample

Laboratory Values	Horse Name/Number
Synovial WBC, $*10^9$/L	3.49
Synovial TP, g/L	2.90
Blood WBC, $*10^9$/L	9.92
Blood TP, g/L	7.00
Synovial hematocrit, %	11.10
Blood hematocrit, %	30.50
Calculated values	
Uncontaminated TP, g/L	1.25
Uncontaminated WBC, $*10^9$/L	0.78

still a challenge when results are above normal but are not above the published cut-off values for septic arthritis. We believe that in these circumstances, use of other diagnostic tools, including cytology as well as the clinical picture and clinician experience, cannot be overrated.

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[a]Microsoft Excel, Microsoft Corporation. One Microsoft Way, Redmond, WA 98052–6399.

Associated Risk Factors of Equine Odontoclastic Tooth Resorption and Hypercementosis

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We suspect that a painful disease involving the equine incisors and canines called equine odontoclastic tooth resorption and hypercementosis (EOTRH) does not have a single determinant but rather occurs as the result of the cumulative impact of several risk factors: trauma caused by excessive dentistry, periodontal disease, type of feed, genetics and hormonal conditions such as equine Cushing's disease (pituitary pars intermedia dysfunction; PPID) and equine metabolic syndrome, and the presence of laminitis. Authors' addresses: Reata Equine Veterinary Group, 9100 East Tanque Verde, No. 100, Tucson, AZ 95748 (Pearson, Mansfield, Conaway); Eller College of Management, University of Arizona, 1130 East Helen Street, Tucson, AZ 85721 (Koput); e-mail: annpearson1960@yahoo.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Equine odontoclastic tooth resorption and hypercementosis (EOTRH) has been described in several publications as a painful disease affecting primarily the incisors and canine teeth.[1–4] In our experience, on gross inspection, the teeth appear as bulbous, irregular, and lytic inside. Gingival receding is often seen along with shifting of incisor positioning (Fig. 1). The radiographic findings include radiopaque densities involving the alveolar tooth roots, shifting of the teeth, osteomyelitis of the alveolar and surrounding bone, and widened periodontal spaces. Similar findings have been reported in the past in which on radiography, the teeth may appear as having bulbous enlargement with or without resorption.[2,3,5]

Recent literature has proposed that horses more than 15 years of age[1–4] and breed predilection such as Thoroughbreds and Warmbloods[6] may be risk factors associated with the incidence of EOTRH. Increased age has been previously noted as a cause of periodontal disease because of a loss of reserve crown and increased interproximal spaces.[7]

In addition to the amount of mastication time, efficient mastication is needed to prevent periodontal disease. Exposure of enamel ridges on the occlusal surface and preventing excessive tooth wear is needed for efficient mastication.[8] It was theorized that by excessive reduction of incisors and cheek teeth, the efficiency of mastication would be decreased. Trauma as a whole when dentistry is performed excessively or by some unlicensed dentists may also weaken the periodontal ligaments. The horses with excessive dental procedures may have a predisposition for lack of salivary bathing time caused by the lack of ridges left after aggressive floating. Our hypothesis is that excessive dentistry may be a risk factor in the development of EOTRH.

Initially, the proposed etiology of EOTRH was thought to be secondary to dental disease, whereby

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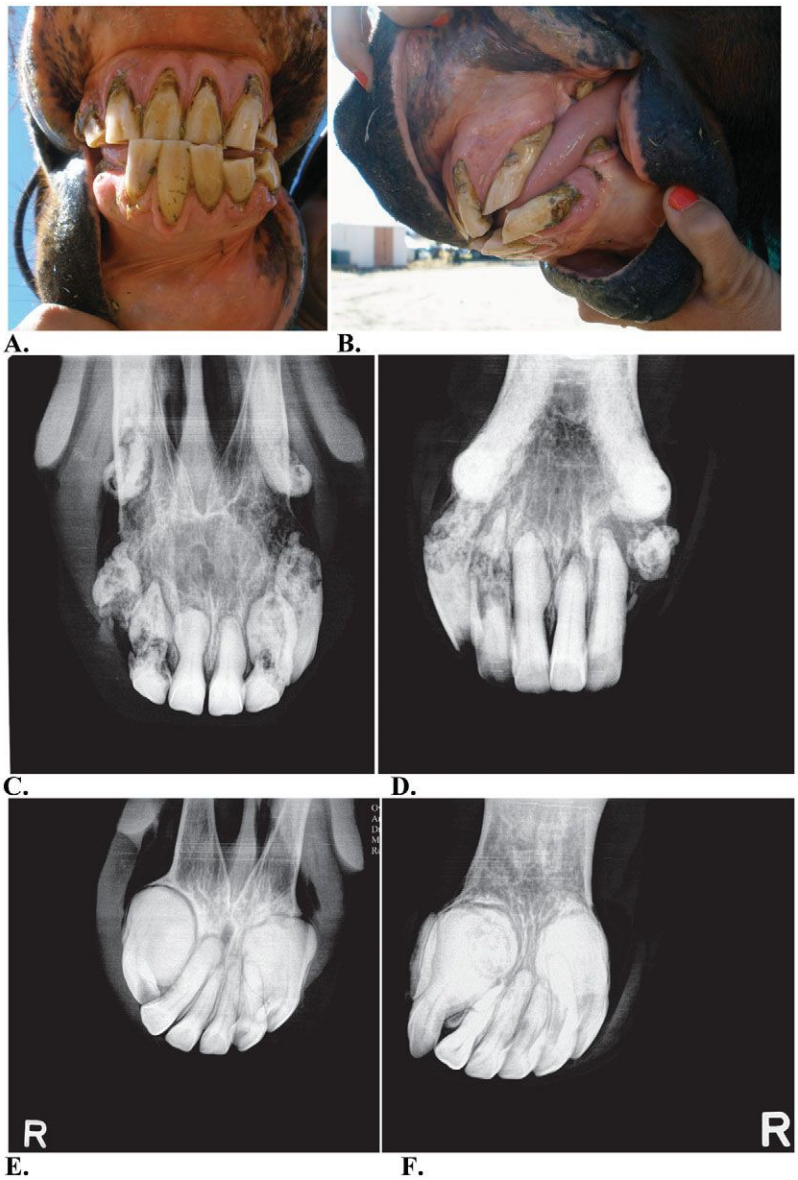


Fig. 1. Images show incisor drifting and gingival recession (A and B) in a 20-year-old Thoroughbred gelding with history of excessive dentistry (incisor and canine reductions). Radiographs of the same horse are shown of the maxillary (C) and mandibular (D) incisors showing resorption and lysis of teeth and bone. Radiographs were taken from a 24-year-old Quarter Horse mare with minimal outward clinical signs (history of avoidance behavior around her mouth). Both maxillary (E) and mandibular (F) incisors exhibit severe hypercementosis of the corner incisors, resulting in crowding of central incisors and loss of rostral incisive and mandibular bone. F, Remnant of canine 304 is superimposed over 303.

the inflammation of the periodontal ligaments were instrumental in the cause of resorptive lesions in calcified incisors.[2,9] Studies were performed by Stazyk in 2008, whereby the diseased teeth were analyzed histologically and grossly. The surrounding cementum was histologically different in affected teeth when compared with normal teeth. The type of collagen fibers as well as the arrangement and size of blood vessels were different. The arrangement of fibers were disordered, and the vascular channels were large within the diseased cementum. An independent variable asked on the retrospective study involved the question of whether or not the horse had ever been on pasture. It is possible that the lack of grazing with the associated tug and tearing motion and lack of movement with the incisors may be associated with a stagnant effect, thereby producing a venous congestion with enlarged vascular channels and lack of blood pumping into the gums. Our hypothesis is that the presence of periodontal disease is a risk factor that may be a predictor of future EOTRH. The development of periodontal disease may be multifactorial and may not only include lack of blood supply to the

gums but also lack of salivary bathing time and hormonal influences.

Reducing food stasis is very important in preventing periodontal disease. Preventing dental interlocks and promoting normal movement of mastication aids in the bathing with saliva.[10] According to Gieche,[10] horses rely on normal salivary bathing of the periodontium and continual movement of food matter through swallowing. By increasing pasture or grass hay, an increase in masticatory time will be instituted. We are hypothesizing that the lack of grazing time will not allow the horse to have the head down in proper position to allow the saliva to have the full effect of bathing the incisors and removing stagnant feed.

The associated evidence of endocrine disorders with accompanying laminitis has been studied as to the mechanisms involved.[11] With the equine metabolic syndrome (EMS) present in several horses, it is possible that the glucose and insulin levels in the blood are much higher and may be associated with the periodontal disease that was present in these horses. In several studies, as will be discussed, the end results of EMS on laminitis include vasoconstriction, thrombosis, and catabolism of laminar proteins.[12] All risk factors should be considered with the anatomical structures around the tooth. It has been found that the tooth actually fits into a fibrous socket called a gomphosis.[10,13] The joint consists of cementum (which is a living tissue),[14] periodontal ligament, bone lining of the alveolus, and gingival that is facing the tooth. In small animals, recent research indicates that there is a much higher prevalence of ligament laxity with associated canine Cushing's disease, presumably because of the hormonal effects of cortizol.[15] It has been widely acknowledged that horses with pituitary pars intermedia dysfunction (PPID) and EMS are much more predisposed to infection. It is hypothesized that horses with clinical signs of PPID, EMS, and laminitis may be more at risk for development of EOTRH. The hormonal effects of cortizol may weaken the periodontal ligaments, and the high levels of glucose and insulin in the blood may affect essential components of the cementum and periodontal ligament adversely, thereby increasing the risk of periodontal disease. The risk factor of horses that have been diagnosed with hormonal diseases such as PPID and EMS with associated laminitis are hypothesized to be of significant value.

2. Materials and Methods

We used detailed medical records on more than 13,000 veterinary calls involving 3461 horses over a 12-year period for an equine veterinary group located in the desert southwest to conduct an archival study that matches the statistical characteristics of a prospective, repeated-measures, longitudinal field study. Horses were included in the analysis on the basis of having been visited by a veterinarian in the group for any reason from 2000 to 2012. Horses were then followed at each subsequent visit by any vet in the group for the duration of the study period or until the horse was no longer a patient. Content analysis was used to code the notes attendant to each visit for key terms indicative of the outcome and risk factors, as illustrated in Table 1. Risk factors were age, breed, sex, lay dentist, incisor reductions, pasture history, PPID, EMS, presence of periodontal disease, signs of involvement with water (playing, standing by the water tank, etc), salivation, laminitis, and the exclusive history of feeding alfalfa.

Table 1. Examples of Terms Used in Content Analysis of Medical Records

Variable	Example Key Terms
Excessive dentistry	Lay dentist, incisor reduction
Dental disease	Periodontal disease
Low-mastication feed	Alfalfa, not pasture, not grazing
Endocrine	Cushing's disease, PPID, Prascend,[b] crested neck, EMS, metabolic syndrome, fatty deposits, laminitis
External/behavioral symptoms	Excess salivation, drooling, mouthing water bucket

Control variables, such as age, breed, and sex, were coded from extant fields in the veterinary group's database. We then performed logistic regression analyses to test for the predictive relationships between risk factors and outcomes entailed in our hypotheses. For the regressions, we restricted our analyses to horse-visit records up to and including the first incidence of the outcome and used statistical corrections for the non-independence of observations inherent in any repeated-measures design.

To gain further insight into and validate the measurement of the outcome in our prospective study, we also conducted a retrospective study on 20 cases in which the outcome was identified. This study involved a clinical examination, radiographs before tooth extraction, and a questionnaire given to the horse owners. The clinical exam included examination of the teeth, evaluation for regional adiposity, presence of long haircoat, and overall body condition. When evidence of EOTRH was present, radiographs were advised, and those patients in the study had radiographs taken before surgical tooth extraction. The questionnaire involved 16 questions covering the risk factors, symptoms, and history items revealed as important in the statistical analyses.

3. Results

Our sample consisted of 3461 horses, ranging in age from 1 to 40 years, covering more than 70 breeds and with a mix of mares, geldings, and stallions. The average age was 9.3 years. Nearly a third of the sample were Quarter Horses, with another fifth be-

Table 2. Describing Risk

	Entire Sample	Coded as EOTRH
Total (number)	3461	64
By risk factors (number)		
Excessive dentistry	269	12
Periodontal disease	39	17
Alfalfa-exclusive diet	79	12
Endocrine-related disorder	676	42
EMS	135	
Cushing's disease	59	
Laminitis	577	
External/behavioral symptoms	40	7
Average age (years)	9.3	12.6

ing Arabians. Geldings accounted for 45% of the horses, with mares accounting for another 35%.

In terms of the prevalence of our hypothesized risk factors, in the entire sample, there were 269 cases of excessive dentistry, 39 with periodontal disease, 79 on alfalfa-exclusive diets, and 676 with an endocrine-related disorder. Of the horses with endocrine-related disorders, 577 had laminits, 135 had EMS, and 59 had Cushing's disease. There were 40 cases that exhibited external or behavioral symptoms that were hypothesized manifestations of EORTH.

The outcome was defined by the coincidence of hypercementosis, incisor/tooth extraction, and resorptive/resorption syndrome; 248 horses had some mention of one of these, 174 had mention of two, 64 horses had all three and were coded for the statistical analysis as having EOTRH.

Table 2 displays the characteristics of the sample described above for the entire sample as well as for those coded as having the outcome.

We present the results of our statistical analyses in Table 3. The table consists of three columns, the first of which contains the variable with our hypothesized risk factors listed first. The columns labeled (1) and (2) present two models predicting EOTRH from the risk factors. In model (1), disorders are aggregated, whereas in model (2) we separately estimate the effects of EMS, Cushing's disease, and laminitis. For each model, we present the exponentiated logistic regression coefficients, or odds-ratios, and their robust standard errors (in parentheses).

When reading Table 3, keep in mind the following: the odds ratios act as measures of the strength or magnitude of the relationship between the predictor and outcome variables, interpretable as multipliers of the odds of the outcome occurring when a risk factor is present. The standard errors provide a sort of "on-average" margin for sampling error. We used one-tailed P values to test our hypotheses because each posits a directional (ie, positive) relationship between predictor and outcome. Significance levels are indicated in the table by asterisks next to the odds ratios. This choice of α implies a conventionally accepted 5% risk of "type I" error, that is, of falsely inferring that a risk factor relates to an outcome.

Table 3. Predicting EOTRH: Logistic Regression Odds Ratios for Hypothesized Risk Factors

	Dependent Variable (DV) = Coded as EOTRH	
Predictor	(1)	(2)
Risk factors		
Excessive dentistry	5.17‡ (0.79)	4.83‡ (0.70)
Periodontal disease	5.24‡ (2.53)	6.05‡ (2.86)
Low-mastication feed	2.01* (0.69)	2.20* (0.81)
Endocrine disorder	1.55† (0.22)	
EMS		1.98* (0.66)
Cushing's disease		2.30† (0.58)
Laminitis		1.073 (.143)
Manifestations		
Behavioral symptoms	3.57† (1.30)	3.57† (1.30)
Controls		
Breed		
Arab	1.28~(0.15)	1.28~(0.15)
Thoroughbred	0.85 (0.14)	0.85 (0.14)
Sex		
Mare	0.61~ (0.10)	0.69~ (0.11)
Gelding	0.67~(0.10)	0.77~(0.11)
Age	0.99 (0.6)	0.99 (0.6)
Model-fit χ^2	199‡	199‡
Pseudo-R^2	.1426	.1426

Robust standard errors are given in parentheses.

Notes: N = 3461 horses (13,707 total visits). Odds ratios are obtained by exponentiating the logistic regression coefficients. Standard errors are adjusted for non-independence caused by repeated observations per horse.

Significance levels: *<0.05;†<0.01; ‡<0.001.

In Table 3, rows 1 through 4 contain the results for our hypothesized risk factors. As an overview, all of our risk factors have statistically significant coefficients, all in the predicted direction. EOTRH thus appears to have a number of antecedents, so let us go through these results in more detail. H1, that excessive dentistry increases the risk of obtaining EOTRH, is strongly supported. An indication of prior treatment by a lay dentist or a procedure such as incisor reduction make a horse nearly 5 times more likely to subsequent development of hypercementosis/resorptive syndrome. H2 is likewise supported, with prior periodontal disease also increasing the chances of EOTRH by a factor of 5. Horses on feed with low mastication time, operationalized in this study as a diet of alfalfa with no time on pasture, are twice as likely to develop EOTRH, supporting Hypothesis 3.

We turn now to endocrine disorders as risk factors for EOTRH. Looking first at model (1) in which we use an aggregate measure, we find, as hypothesized in H4, that they are positively related to EOTRH. In aggregate, having an endocrine disorder increases the odds of EOTRH by >50%. In (2), we obtained separate estimates for each of the endocrine disorders, revealing that EMS is most strongly

related to EOTRH, with an odds ratio of 2.5. Cushing's disease is also a significant predictor, nearly doubling the odds of the outcome. The coefficient for laminitis, when taken separately from the primary endocrine disorders, was close to unity and not statistically significant.

Moving beyond the tests of our hypotheses, we can remark on the other variables that we included as manifestations of EOTRH, or as controls. The results in Table 3 indicate that the external and behavioral symptoms are significant correlates of EOTRH. External signs, such as excess salivation, and behaviors such as mouthing a water bucket, are symptomatic of EOTRH. We note, without remark, that Arabians are at greater risk than other breeds and that mares and geldings are less prone than stallions. Age is not a significant predictor in our study. This is perhaps somewhat surprising but tells us that it is not maturation in and of itself that puts a horse at risk of EOTRH. Rather, it is the other risk factors that attend to aging.

The follow-up clinical exams and owner survey served to validate the statistical results just presented, in terms of clinical findings, signs and symptoms, and histories. There were many common findings involving the clinical exams for the selected cases: (1) periodontal disease, including petecchiation of the gingiva over the teeth, draining tracts, purulent discharge, inflamed gingiva, loose teeth, enlarged, bulbous teeth, pain elicited when tapping affected teeth, and a foul odor present, (2) signs of PPID including a long haircoat, and (3) body condition and signs of EMS, including regional adiposity.

The follow-up questionnaire given to owners also revealed a series of similar clinical signs and symptoms for the subsample of horses coded as EOTRH: (1) inappetance and slow eating, (2) salivation/drooling and foaming of the mouth, (3) exhibiting a smiling appearance at rest and when riding whereby the lips are pulled back, (4) evidence of previous periodontal disease and the exhibition of a painful response when the teeth were tapped, (5) standing at the water tank, rinsing the mouth, or holding the mouth in the water tank (one owner reported that the horse's muzzle was always wet, and one owner reported that the horse's tongue was always held between the teeth), 6) late shedding of the haircoat and the presence of an unusually long haircoat.

Finally, a review of the histories of the horses selected for follow-up converged on these points: (1) excessive reduction of the incisor teeth and/or cheek teeth, (2) lack of grazing, (3) the feeding of primarily alfalfa hay, and, (4) the history of laminitis.

4. Discussion

History of excessive dentistry was nearly 5-fold higher when predicting the presence of EOTRH. Dr Carsten Staszyk in 2010 hypothesized that mechanical stress within the periodontal ligament may be the initiating factor of EORTH.

Regarding trauma, as in excessive dentistry, cementoblasts are present in the periodontal ligament.[15] Cementum can respond to harmful events quickly after harmful stimuli by further rapid deposition of cementum. Cementum has a high percentage of mineral present (approximately 65%). Within the mineral content, a large portion is calcium hydroxyapatite.[16–18] The calcium content of alfalfa is disproportionately high when compared with phosphorus. Could feeding straight alfalfa along with excessive dentistry be compounding risk factors?

The prevalence of periodontal disease was shown to be a supporting piece of evidence when predicting a positive risk factor for EOTRH, as previous research has indicated. Horses with prior periodontal disease increased risk by factor of 5.

As mentioned in the Results, horses fed alfalfa without pasture or grazing are twice as likely to have EOTRH. Hypervitaminosis A has been listed as a possible etiology of EOTRH.[5] Alfalfa and other legume hay have a high percentage of calcium (1.2–1.8%) compared with grasses that tend to be relatively low in calcium (0.3–0.7%). Phosphorus tends to be low in most forage (0.23–0.33%). Legume hay is also a source of provitamin A (beta-carotene), which is metabolized to vitamin A in horses within the small intestine.[19] In addition to the lack of chewing time and elevation of the horse's head while chewing, alfalfa requires much less chewing time. As mentioned earlier, the lack of chewing time and difference in elevation of the head will decrease the amount, time, and path of bathing the teeth and gums with saliva.

Although laminitis was not found to be a significant risk factor for EOTRH in our sample, further research is warranted. The associated laminitis that has been found to be as a result of horses having the disease of EMS may have similar mechanisms of producing EOTRH. It has been found in horses with EMS-induced laminitis that the insulin-induced lesions showed cellular changes and stretching elongation of the secondary epidermal lamellae.[11,12,20] According to another author, levels of matrix metalloprotease 9 were increased 48 hours after the corresponding neutrophil infiltration of the lamellae.[21] In addition, thrombosis, hypertension, catabolism of structural laminar proteins, vasoconstriction, and dysregulation of laminar cell growth have been listed.[12] It would be interesting to compare in further research the effect of high insulin levels and hyperglycemia on the periodontal ligament and Sharpey fibers to see if stretching and cellular changes would also occur with those structures. As mentioned before, it is the author's opinion that there may be a correlation as to the level of cortizol seen in small-animal joints and stretching of the periodontal ligaments as well.

The existing treatments for EOTRH include removal of affected teeth with regional anesthesia and standing sedation,[22,23] rinsing with 0.12% chlorhexi-

dine, and treatment with trimethoprim sulfa 24 mg/kg q12 hours orally for 28 days[14] and splinting to prevent drifting of adjacent teeth after removal of affected incisors.[a] This disease is very painful for the horse, and the treatment regimen is very difficult for the patient to endure. The follow-up after removing teeth will be continuous to make sure the mouth stays in balance with occlusion.

The equine veterinarian must consider the existing risk factors when examining the horse and taking the history. These factors may be helpful when managing the prevention of the disease of EOTRH.

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[a]Personal communication from Dr Tom Johnson, Advanced Equine Dentistry, Grass Lake, Michigan.

[b]Prascend®, Boehringer Ingelheim Vetmedica GmbH, Binger Str. 173, 55216 Ingelheim, Germany.

Optimization of a Navicular Bursal Injection Technique That Avoids Penetration of the Deep Digital Flexor Tendon

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Injection of the navicular bursa can be performed from the lateral aspect of the distal pastern region, avoiding deep digital flexor penetration with the use of radiographic guidance. Authors' addresses: Clinical Sciences Department (Daniel, Goodrich), Orthopedic Research Center (Goodrich, McIlwraith), and Department of Environmental and Radiological Health Science (Barrett, Valdes-Martinez), Colorado State University, 300 West Drake Road, Fort Collins, CO 80523; University of Florida, College of Veterinary Medicine, PO Box 100125, Gainesville, FL 32610–0125 (Werpy); e-mail: laurie.goodrich@colostate.edu. *Corresponding author; †presenting author. © 2013 AAEP.

1. Introduction

Injection of the navicular bursa is commonly performed from the palmar aspect of the limb, which results in penetration of the deep digital flexor tendon (DDFT). We investigated an injection of the navicular bursa from the lateral aspect of the limb to determine soft tissue penetration with the use of this approach.

2. Materials and Methods

Cadaveric specimens were collected and placed in a stand to simulate a standing position. Injection of the bursa was performed in specimens without synovial distension or with distension of the distal interphalangeal joint or digital flexor tendon sheath. A 100-mm, 20-gauge, titanium MRI-safe needle was placed into the bursa with the use of radiographic guidance to confirm accurate needle placement. Dilute contrast was then injected into the bursa before a final radiograph was performed. With the needle in situ, a complete MRI examination was performed.

3. Results

Successful navicular bursal injection was achieved in all specimens, and no penetration of the DDFT occurred on the basis of MRI examination. The optimal angle of needle penetration was determined relative to the horizontal and vertical planes from

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dorso-palmar and latero-medial radiographs. When the distal interphalangeal joint was inadvertently injected with contrast, a small correction in needle angle resulted in accurate bursal injection before MRI was performed.

4. Conclusions

With the use of the 1-Tesla MRI to gauge tissue penetration, the lateral approach of the navicular bursa appears to avoid penetration of the DDFT and the palmar fibrocartilage.

How to Perform the Basilar Sesamoid Approach for Digital Flexor Tendon Sheath Injection in Horses

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1. Introduction

Access to the digital flexor tendon sheath (DFTS) is needed for synovial fluid sample collection, administration of local anesthetics or other medication, tendon sheath lavage, and creation of portals for tenoscopy. Because of intimate associations between the DFTS and various tendons and ligaments in the distal aspect of the limb, the DFTS is commonly involved in orthopedic problems in horses.[1–5] Diagnosis of lameness can be facilitated by administration of local anesthetic solution within the DFTS, which can be necessary in horses that have little or no DFTS effusion. The distal sesamoidean ligaments, distal digital annular ligament, deep digital flexor tendon (DDFT), superficial digital flexor tendon (SDFT), palmar/plantar annular ligament, and intersesamoidean ligament are intimately associated with the DFTS, and these structures can be desensitized by deposition of local anesthetic solution within the sheath.[2–4] Historically, collection of synovial fluid from the DFTS or injection into this structure has been difficult if little or no effusion is present.[4,6] An efficient approach to the DFTS that simplifies performance of these procedures is needed.

The DFTS is a complex synovial structure that surrounds the DDFT and SDFT in the distal aspect of the limb.[7] This sheath begins at the junction of the proximal two-thirds and the distal third of the metacarpus or metatarsus and extends distally past the metacarpophalangeal or metatarsophalangeal joint and pastern region, terminating just proximal to the collateral sesamoidean ligament within the hoof.[8] The DDFT is completely enveloped by the DFTS in the fetlock and pastern regions; however, the branches of the SDFT exit the DFTS at midpastern level to insert on the proximopalmar or proximoplantar aspect of the middle phalanx.

Two sites typically accessed for DFTS synoviocentesis and injection in horses with effusion of this structure are the proximolateral pouch and the distal palmar or plantar pouch.[9] When distension is present, fluid can accumulate in relatively large amounts in these locations. The proximolateral pouch is located abaxially at the proximal extent of the sheath between the suspensory ligament

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branches and the DDFT. The distal palmar or plantar pouch is located at midline on the palmar or plantar surface at the distal aspect of the pastern between the proximal and distal digital annular ligaments. A third site used for DFTS synoviocentesis and injection, accessed through a palmar or plantar axial sesamoidean approach, has also been described. This approach, in which a needle is inserted into the DFTS from a location axial to the midbody of either proximal sesamoid bone, has been shown to be superior to the proximolateral approach in regard to the speed at which the DFTS is entered and decreased number of attempts to penetrate the DFTS in a study of equine cadaver limbs.[6] These approaches can result in successful DFTS penetration, but the time it takes to enter the sheath and the ability to obtain fluid can be quite variable,[6] indicating the need for development of a more reliable technique.

The proximal and distal collateral pouches of the DFTS are less commonly discussed in the literature. The proximal collateral pouch is found at the base of the proximal sesamoid bones between the distal aspect of the palmar or plantar annular ligament and the proximal aspect of the proximal digital annular ligament on either side of the palmar or plantar midline. The distal collateral pouch is found abaxially between the proximal and distal attachments of the proximal digital annular ligament.[8] Neither of these pouches are easily visualized, even with DFTS effusion, but the proximal collateral pouch can be accessed by use of the base of a proximal sesamoid bone and the edge of the SDFT as landmarks.

The proximal collateral pouch has been used clinically for many years as an alternative approach for DFTS access,[1,2,9] but the approach has not been fully described in the literature and the advantages and disadvantages of this approach have not been evaluated in a controlled study. Clinically, this basilar sesamoid approach (BSA) has been used in standing and recumbent horses for administration of local anesthetic or medication and as a site for gaining access for lavage and tenoscopy.[3,9,10]

The objectives of the present study were to describe the BSA to the proximal collateral pouch of the DFTS and to evaluate the ease of injection and synovial fluid sampling with this approach in standing horses.

2. Materials and Methods

Twelve healthy adult mares from a university-owned breeding herd were included in the evaluation of the BSA. The horses had no evidence of lameness at a walk, no palpable effusion of the DFTS, and no obvious conformational abnormalities related to the distal aspects of the limbs. There were eight Quarter Horses, three Thoroughbreds, and one mixed-breed horse; median age was 12 years (range, 3 to 19 years) and body weight was 480 kg (range, 400 to 600 kg). One forelimb and one hind limb of each horse was randomly assigned for DFTS synoviocentesis and injection through the use of the BSA. This resulted in 24 limbs (12 forelimbs and 12 hind limbs).

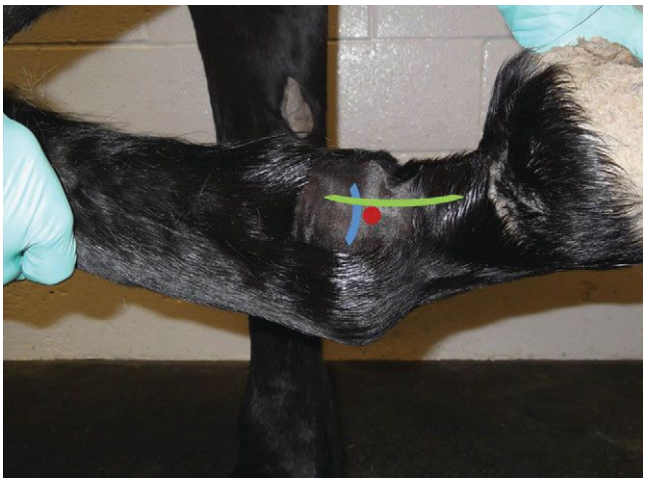


Fig. 1. Lateral view of the left forelimb held in a non–weight-bearing position by an assistant and mildly flexed between 200° and 220°. The edge of the superficial digital flexor tendon is outlined in green, the base of the lateral proximal sesamoid bone is outlined in blue, and the site of injection for the basilar sesamoid approach is indicated by the red dot. Proximal is to the left.

For the BSA, hair is clipped from the lateral and palmar or plantar aspect of the fetlock region with a No. 40 blade (Fig. 1), resulting in a clipped area centered over the lateral proximal sesamoid bone. The clipped area is prepared for sterile injection. Horses in this study were sedated with detomidine hydrochloride (4 to 12 μg/kg IV), but restraint can also be achieved with the application of a nose twitch if the BSA is being used for local anesthetic deposition during lameness evaluation.

The metacarpophalangeal or metatarsophalangeal joint is held by an assistant in a mildly flexed position at an angle of approximately 200° to 220° from the dorsal surface of the third metacarpal or metatarsal bone (Fig. 1). This is accomplished most easily with the assistant facing the rear of the horse and holding the canon bone with the right hand and the hoof with the left hand when injecting the left forelimb or hind limb, and with the assistant holding the canon bone with the left hand and the hoof with the right hand when injecting the right forelimb or hind limb. This positioning provides easier access to the injection site. For hind limbs, the limb should be extended as much as possible toward the ground and behind the horse to decrease the spontaneous fetlock flexion that occurs when the limb is lifted off the ground. The injection site is then identified in the non–weight-bearing limb by palpating the depression created by the base of the lateral proximal sesamoid bone proximally and the lateral border of the SDFT axially (Fig. 2). This palpation can be facilitated by having the assistant



Fig. 2. Palmarolateral view of the left forelimb held in a mildly flexed position by an assistant. The palpable divot is identified with a finger at the base of the lateral proximal sesamoid bone. Proximal is to the left.

slightly flex and extend the distal limb as the palpation is occurring. An 18-gauge, 3.8-cm needle is directed into the palpable depression at an angle of approximately 45° to the transverse plane (in a lateromedial direction) and 45° to the dorsal plane (in a distoproximal direction), to a depth of ≤1 cm (Fig. 3). Synovial fluid is commonly obtained with very superficial placement of the needle; therefore no further attempt should be made to advance the needle until it is determined that the initial needle placement is not successful, either by visualizing synovial fluid in the needle hub or by lack of resistance to injection.

Synoviocentesis and Injection

For this study, all synoviocentesis and injection procedures were performed by the same veterinarian who had no prior clinical experience with the technique being evaluated but had practiced the technique on cadaver limbs before beginning the live horse study. For each injection, 8 mL of sterile contrast material[a] (iohexol [300 mg of iodine/mL]) was injected, with confirmation of positioning on the basis of spontaneous appearance of synovial fluid in the needle hub or by lack of resistance to injection if no synovial fluid was obtained. Immediately after injection of each limb, a lateromedial radiograph was obtained under conditions of weight-bearing to determine whether the contrast material was delivered accurately. All procedures were recorded with a digital video camera to ensure accurate documentation of time required for the procedure, number of attempts to enter the DFTS, and presence of synovial fluid in the needle hub.

Assessment of Injection

For each injection in this study, the number of attempts needed to insert the needle into the DFTS was recorded. Any redirection of the needle constituted a new attempt to enter the DFTS. The presence of synovial fluid in the needle hub (yes or no) was recorded. All lateromedial radiographs were evaluated for the presence or absence of contrast material within the DFTS, and location of contrast material was recorded and described. Successful injection of contrast material was defined as obvious contrast material filling the DFTS throughout its proximal to distal extent of the sheath (Fig. 4). Elapsed time for DFTS injection was measured from needle insertion through the skin to the beginning of contrast injection into the sheath. The association between time to successful injection and horse was also assessed to determine whether the investigator gained proficiency with the technique during the course of the study.

Statistical Analysis

Visual assessment of the results for injection time and number of times the needle was redirected indicated the data were not normally distributed. Consequently, nonparametric methods of analysis that accounted for the hierarchic structure of the data were used.[b] For each outcome, the data were ranked and analysis of variance was performed. An estimation of the covariance parameters with a repeated statement specifying horse identity as the subject, limb as the group, and an unstructured covariance structure was obtained. Limb interaction (front versus hind) was initially included in the model as a fixed effect but was found to have no significant bearing on the outcome. Linear regression was used to assess the association between



Fig. 3. Palmarolateral view of the left forelimb held in a mildly flexed position by an assistant and an 18-gauge, 3.8-cm needle is placed sterilely into the digital flexor tendon sheath through the use of the basilar sesamoid approach. The needle is angled at 45° to the transverse plane and 45° to the dorsal plane. Proximal is to the left.



Fig. 4. Lateral radiographic projection of the left front limb of a horse with contrast material filling the digital flexor tendon sheath immediately after injection through the use of the basilar sesamoid approach. Dorsal is to the left.

horse order and injection time. Values of $P < 0.05$ were considered significant.

3. Results

The median elapsed time for needle placement and injection of contrast material into the DFTS was 11.5 seconds (range, 4.2 to 114.9 seconds). The median number of times the needle was redirected for DFTS injection was 0.5 (range, 0 to 7). Synovial fluid was obtained spontaneously in 87.5% of limbs. There was no gross evidence of blood in synovial fluid obtained with any of the injections. Injection of contrast material into the DFTS was successful in 24 of 24 limbs. Comparison of results over time revealed that the time from initial needle insertion to injection decreased with increasing horse order ($R^2 = 0.30$; $P = 0.006$). There was no significant difference between front and hind limbs in any evaluated parameter.

4. Discussion

The BSA provided access to the DFTS with a high likelihood of synovial fluid retrieval in a relatively short period of time. Injection of contrast material into the DFTS was successful in all limbs. It is important to properly position the limb to expose the BSA in order to facilitate successful injection.

The anatomy of the proximal collateral pouch of the DFTS, the targeted entry point for the BSA, provides a superficial recess with an absence of overlying structures that makes this a practical alternative to conventional approaches and results in shallow needle penetration into the limb. It is likely that the ease of entry into the DFTS in the present study was in part because of the easily identifiable landmarks (specifically, the lateral border of the SDFT and the base of the lateral proximal sesamoid bone).

Clinically, an important aspect of this study was the ability to obtain a synovial fluid sample in 21 of 24 limbs. It was thought that the use of an 18-gauge, 3.8-cm needle was necessary to have the best chance of retrieving synovial fluid, and this size needle is commonly used for synoviocentesis. It is possible that a smaller diameter and shorter needle could be used for this approach, but other needle sizes were not evaluated in this study. On the basis of the depth of penetration needed for the approach in this study, a 2.5-cm needle is considered to be of sufficient length to enter the DFTS in horses with no swelling of the limb.

In the present study, synovial fluid retrieval was determined solely on the basis of visual detection of synovial fluid in the needle hub. This decision was made before beginning the study because the use of suction with a syringe to obtain fluid could result in tissue plugging the end of the needle and this could hinder subsequent interpretation of needle entry into the DFTS. When synovial fluid was not obtained in the hub of the needle, needle placement within the DFTS was determined by lack of resistance to injection.

A potential advantage of the BSA is the ability of a veterinarian unfamiliar with this approach to gain proficiency over time, although this was evaluated for only one veterinarian. Results of linear regression modeling indicated a significant association between horse order and time for DFTS injection. Given the number of other factors that could contribute to this outcome, the effect of horse order was shown to have a substantial effect by explaining 30% of the variation in time. Gaining efficiency quickly is particularly important to veterinarians who are attempting to learn a new procedure.

One important limitation of the present study was the inability to grossly or microscopically evaluate the limbs internally for trauma from needle penetration because none of the horses in this study were euthanized. However, it is theorized that the BSA may be a less traumatic means of entering the DFTS because the needle does not need to enter into deep structures of the limb and there is minimal interference to needle placement by surrounding structures.

In the present study, injections were performed in standing sedated horses, and there was some movement of the limb as there would be when performing the same techniques on standing non-sedated horses in a clinical setting. The BSA has been used by the author (SNS) in many equine patients for lameness diagnosis, medication administration, diagnosis of sepsis, evaluation of wound penetration, and tendon sheath lavage in nonsedated, sedated, and anesthetized horses as well as for tenoscopic portals in anesthetized horses. In the author's (SNS) experience, the BSA is no more difficult than a metacarpophalangeal or metatarsophalangeal joint injection and does not elicit more uncooperative behavior than the latter procedure; thus, it can be done with minimal restraint in many horses.

Results of the present study provide evidence that the BSA is a useful method for DFTS synoviocentesis and injection in forelimbs and hind limbs of normal standing horses and may be superior to other DFTS approaches in regard to ease of entry into the DFTS and the likelihood of obtaining a synovial fluid sample. Given the high frequency of obtaining a synovial fluid sample with the BSA in clinically normal horses (21/24 limbs), the use of the BSA should be considered when DFTS synovial fluid analysis is desired. Also, the use of the BSA should be considered when DFTS synoviocentesis is to be performed on limbs with minimal or no DFTS effusion.

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The study procedures were approved by the Institutional Animal Care and Use Committee at Mississippi State University.

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Disposition of Methylprednisolone Acetate in Plasma, Urine, and Synovial Fluid After Intra-Articular Administration to Exercised Thoroughbred Horses

**Heather K. Knych, DVM, PhD, Diplomate ACVCP*;
Linda M. Harrison, DVM, PhD, Diplomate ACVS; Haley C. Casbeer, BS; and
Dan S. McKemie, BS**

The withdrawal time for intra-articular administration of methylprednisolone acetate (MPA) before a performance depends on the specific threshold established by the racing jurisdiction. In the current study, MPA was detectable in plasma, urine, and synovial fluid for up to 13, 21, and 70 days, respectively. Authors' addresses: K.L. Maddy Equine Analytical Chemistry Laboratory, School of Veterinary Medicine, University of California, Davis, CA 95616 (Knych, Casbeer, McKemie); Willow Oak Equine, 33558 County Road 24, Woodland, CA 95695 (Harrison); e-mail: hkknych@ucdavis.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Although its use remains somewhat controversial, methylprednisolone acetate (MPA) is one of the more commonly used intra-articular corticosteroids in performance horses. The goal of the current study was to describe the disposition of MPA in plasma, urine, and synovial fluid after intra-articular administration.

2. Materials and Methods

Sixteen healthy, exercised adult Thoroughbred horses received a single intra-articular MPA administration (100 mg) into the right antebrachiocarpal joint. Blood, urine, and synovial fluid samples were collected before and at various times after drug administration. Synovial fluid was collected from the right and left antebrachiocarpal and the right and left middle carpal joints. All samples were analyzed with the use of liquid chromatography-mass spectrometry (LC-MS). Pharmacokinetic analysis was conducted with the use of non-compartmental analysis.

3. Results and Discussion

The plasma terminal elimination half-life was 1.33 ± 0.80 and 0.843 ± 0.414 days for horses that had synovial fluid collected (group 1) and those that did not (group 2), respectively. MPA was undetectable by day 6.25 ± 2.12 (group 1) and 4.81 ± 2.56 (group 2) in plasma and day 17 (group 1) and day 14 (group 2) in urine. MPA was detected in synovial fluid from all joints sampled. MPA remained above the limit of detection for up to 70 days in the joint of administration. Results from this study will aid in establishing threshold concentrations and appropriate withdrawal times for racehorses.

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Pharmacokinetics and Clearance of Triamcinolone Acetonide After Intramuscular and Intra-Articular Administration to Exercised Thoroughbred Horses

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The results of this study support an extended withdrawal time for triamcinolone acetonide given intramuscularly and suggest that plasma and urine concentrations are not a good indicator of synovial fluid concentrations. Authors' addresses: K.L. Maddy Equine Analytical Chemistry Laboratory (Knych, Casbeer, McKemie), Department of Surgery and Radiology (Vidal), School of Veterinary Medicine, University of California, Davis, CA 95616; e-mail: hkknych@ucdavis.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Corticosteroids are potent anti-inflammatory agents used commonly in horses to treat performance-related injuries. Because of their potential to mask injuries that would otherwise prevent a horse from performing, regulation of corticosteroids is of the most importance. The goal of the present study was to describe plasma, urine, and synovial fluid concentrations of triamcinolone acetonide (TA) after intra-articular and intramuscular administration.

2. Materials and Methods

Twelve fit, racing adult Thoroughbred horses received a single intramuscular administration of TA (0.1 mg/kg). After an appropriate washout period, the same horses then received a single intra-articular TA administration (9 mg) into the right antebrachiocarpal joint. Blood, urine, and synovial fluid samples were collected before and at various times up to 60 days after drug administration and analyzed with the use of liquid chromatography–mass spectrometry. Plasma data were analyzed by means of non-compartmental analysis.

3. Results and Discussion

The plasma terminal elimination half-life was 11.4 ± 6.53 and 0.78 ± 1.00 days for intramuscular and intra-articular administration, respectively. After intramuscular administration, TA was below the limit of detection by days 52 and 60 in plasma and urine, respectively. After intra-articular administration, TA was undetectable by day 7 in plasma and day 8 in urine. TA was also undetectable in any of the joints sampled after intramuscular administration and remained above the limit of quantitation for 21 days after intra-articular administration.

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Efficacy of Intramuscular Meperidine Hydrochloride Versus Placebo in Experimental Foot Lameness in Horses

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Meperidine may provide a suitable nonsteroidal anti-inflammatory drug alternative analgesic for acute foot pain, but it requires more frequent administration. Authors' address: University of Illinois at Urbana-Champaign, College of Veterinary Medicine, Department of Veterinary Clinical Medicine, 1008 West Hazelwood Drive, Urbana, IL 61802. Dr Ruemmler's current address is Boston Equine Associates, 28 Tremont Street, Rehoboth, MA 02769; e-mail: jhf@illinois.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

There are no refereed, blinded, controlled documentations of the skeletal analgesic efficacy of intramuscularly administered meperidine. The objective of this study was to test the hypothesis that meperidine (pethidine) administered intramuscularly would be more efficacious in alleviating lameness than would a saline placebo in an adjustable heart bar shoe model of equine foot pain.

2. Materials and Methods

Eight healthy adult Thoroughbred horses randomly underwent weekly intramuscular treatments 1 hour after lameness induction: saline placebo (1 mL/45 kg body weight) or meperidine HCl (1 mg/kg IM). Heart rate and lameness score responses were assessed by a blinded observer every 20 minutes for 5 hours after lameness induction and then hourly through 12 hours after treatment. Jugular venous blood samples were obtained at hours −1, 0, 0:05, 1, 2, 4, 6, 8, 10, and 12 and were subsequently analyzed for drug concentrations (lower limit of detection, 1 ng/mL). Repeated-measures analysis of variance and post hoc Tukey's test were used to identify analgesic effects at a significance level of $P < 0.05$.

3. Results

Mean (±SE) heart rates were lower in meperidine trials at 2.3, 3.3, and 3.7 hours after administration ($P < 0.05$). Mean lameness scores were lower in meperidine trials from 2.0, 2.3, and 3.3 hours after administration ($P < 0.05$). Mean plasma [meperidine] peaked at 227 ± 52 ng/mL at 1 hour after administration and decreased to 2.7 ± 0.3 ng/mL at 12 hours after administration. In three of eight subjects, plasma [meperidine] was below the lower limit of detection at 12 hours after administration.

4. Conclusions

It was concluded that intramuscular meperidine was more effective than saline placebo but only for a short period of time (2.0–3.67 hours after administration) compared with previous 8- to 12-hour durations of efficacy in this same model when horses

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were treated with nonsteroidal anti-inflammatory drugs.

Acknowledgments

This study was funded by the Maria Caleel Fund for Equine Sports Medicine Research at the University of Illinois. Horses were purchased with funds provided by the Grayson/Jockey Club Foundation, Keeneland Racing Association, and Thoroughbred Owners and Breeders Association, Kentucky Thoroughbred Association. The authors thank Drs Jason Bundy and Keith Zientek for technical assistance.

Physiologic Effects of Long-Term Immobilization of the Equine Distal Limb

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The objective of this study was to describe the effects of distal limb immobilization and remobilization in the equine metacarpophalangeal joint. Authors' addresses: University of Pennsylvania, New Bolton Center, 382 West Street Road, Kennett Square, PA 19348 (Stewart); Orthopaedic Research Center, Colorado State University, 300 West Drake Road, Fort Collins, CO 80523 (Kawcak, McIlwraith); University of Florida, Department of Small Animal Clinical Sciences, Gainesville, FL 32610 (Werpy); e-mail: hstewartvet@gmail.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

One forelimb of eight horses was immobilized in a fiberglass cast for 8 weeks, followed by a 12-week standardized training program. The third metacarpal (MC3), proximal phalanx (PP), and proximal sesamoid bones (PSB) were examined by means of radiography, computed tomography, nuclear scintigraphy, and magnetic resonance imaging.

2. Materials and Methods

Serum and synovial fluid were collected for biomarker analyses. Lameness, decreased range of motion, joint capsule thickening, and joint effusion were observed in the immobilized metacarpophalangeal joint (MCP). Significant increases in bone sclerosis and lysis, osteophyte, enthesiophyte, and fragment formation were observed radiographically in the immobilized limb during the exercise period.

3. Results

Computed tomography examination revealed a significant time-by-cast interaction on bone density in MC3 and PSB. Magnetic resonance imaging revealed a significant increase in synovial proliferation, articular cartilage degeneration, osteophyte and enthesiophyte formation, and thickening within the soft tissues of the immobilized MCP, specifically the deep digital flexor tendon. Gross lesions in the MCP included wear lines, articular cartilage erosion, osteochondral fragmentation, and palmar arthrosis. Serum and synovial fluid biomarkers varied significantly with immobilization and exercise.

4. Discussion

Eight weeks of single-limb immobilization is sufficient to induce significant changes to bone mineral density, articular cartilage, and surrounding soft

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tissues structures and alter the physiologic environment of the MCP in an immobilized equine forelimb. Twelve weeks of exercise is insufficient for recovery to pre-immobilization bone density and resulted in significant changes to the deep digital flexor tendon and periarticular soft tissue structures.

Anesthetic Diffusion Following Two Approaches to Block the Deep Branch of the Lateral Plantar Nerve

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The diffusion of anesthetic was highly variable after either approach was used to block the deep branch of the lateral plantar nerve. High-volume injections may increase the risk of false-positive results. Inadvertent penetration of synovial structures is possible when blocking the deep branch of the lateral plantar nerve. Authors' address: Large Animal Surgery, Department of Large Animal Medicine, University of Georgia, College of Veterinary Medicine, Athens, GA 30602; e-mail: kclaunch@uga.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The objectives of this study were to compare the diffusion patterns and risk of inadvertent injection into synovial structures for two different techniques that have been described to block the deep branch of the lateral plantar nerve (DBLPN) with the use of two different volumes of anesthetic.

2. Materials and Methods

In 16 horses, either 2 mL or 8 mL of mepivacaine hydrochloride-iohexol (50:50 mixture) was injected with the use of one of two different techniques to block the DBLPN. Radiographs were obtained before and at multiple time points after injection. The extent of diffusion of the contrast solution and whether or not contrast appeared to be present in any synovial structures were determined.

3. Results

A high degree of variability in diffusion was noted among injections. High-volume injections diffused significantly further than did low-volume injections. Contrast agent was documented within the tarsal sheath in 16% of injections (five of 32) and within the tarsometatarsal (TMT) joint in 6% of injections (two of 32). A significant difference in diffusion pattern was observed between the two techniques.

4. Discussion

The 2-mL injection appeared to be in the correct location of the DBLPN. A low-volume injection may be superior to a high-volume injection to decrease the number of false-positive responses to local anesthesia. A perpendicular needle approach may be the preferred technique to avoid proximal diffusion of anesthetic.

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In Vivo Diffusion Characteristics After Perineural Injection of the Deep Branch of the Lateral Plantar Nerve With Mepivacaine or Iohexol in Horses

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Diagnostic analgesia of the deep branch of the lateral plantar nerve can result in inadvertent involvement of the tarsal sheath and/or tarsometatarsal joint. Authors' address: Department of Clinical Sciences, Colorado State University, Equine Orthopaedic Research Center, 300 West Drake Road, Fort Collins, CO 80523; e-mail: erincontino@yahoo.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Proximal suspensory ligament (PSL) desmitis is a common injury in sport horses. Accurate diagnosis of the condition can be difficult, partly because diagnostic analgesia of this region lacks specificity. Perineural analgesia of the deep branch of the lateral plantar nerve (DBLPN) to diagnose PSL desmitis has been described but not evaluated in-vivo.

2. Materials and Methods

The DBLPN was injected perineurally with 3 mL of either mepivacaine ($n = 8$) or contrast media ($n = 8$) in live horses. Contrast-injected limbs were radiographed 5, 15, and 30 minutes after injection, and diffusion characteristics were described. In mepivacaine-injected limbs, synovial fluid from the tarsometatarsal joint was obtained 10 and 20 minutes after injection, and mepivacaine concentrations were analyzed.

3. Results

At 5, 15, and 30 minutes after injection, the contrast media extended, on average, 19.6, 20.6, and 21.0 mm proximal and 38.0, 43.5, and 51.9 mm distal to the injection site, respectively. Three of eight (37.5%) limbs had evidence of contrast media in the tarsal sheath. Two of eight (25%) limbs had tarsometatarsal joint mepivacaine concentrations sufficient to produce analgesia (>300 mg/L) at 10 minutes after injection.

4. Discussion

Analgesia of the DBLPN is commonly used to diagnose PSL desmitis; however, this technique can result in inadvertent involvement of the tarsal sheath and/or tarsometatarsal joint. This is important to consider when evaluating the response to analgesia of the DBLPN and reiterates that subtarsal analgesia is not specific to the PSL.

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Comparison of Cellular Properties of Equine Multipotent Mesenchymal Stromal Cells After the Use of Two Different Isolation Techniques

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Enzymatic tissue digestion and explant technique showed no major impact on characteristics of isolated multipotent mesenchymal stromal cells (MSCs). MSCs isolated by digestion might be advantageous for application in equine tendon regeneration caused by higher expression levels of the tendon marker scleraxis. Additionally, higher MSC numbers are available. Authors' addresses: Large Animal Clinic for Surgery, University of Leipzig, An den Tierkliniken 21, 04103 Leipzig, Germany (Gittel, Brehm, Burk, Ribitsch); Translational Centre for Regenerative Medicine (TRM), University of Leipzig, Philipp-Rosenthal Str 55, 04103 Leipzig, Germany (Juelke); Department of Veterinary Anatomy, Histology, and Embryology, Faculty for Veterinary Medicine, Justus-Liebig-University, Giessen, Frankfurter Str 98, 35392 Giessen, Germany (Staszyk); Equine Hospital, Clinic for Equine Surgery, University of Veterinary Medicine, Veterinärplatz 1, 1210 Vienna, Austria (Ribitsch); e-mail: Claudia.Gittel@vetmed.uni-leipzig.de. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Mesenchymal stromal cells (MSCs) are a promising tool in regenerative medicine for treatment of tendon lesions in horses. MSCs can be isolated from various solid tissues either by enzymatic digestion or by explant technique. Thereby, differences in cell characteristics caused by different preparation techniques can be expected. Therefore, the aim of this study was to investigate and compare cell features of MSCs from solid tissues after different isolation methods are used.

2. Materials and Methods

Equine adipose tissue, tendon, and umbilical cord matrix were harvested, and MSCs were isolated by enzymatic digestion with collagenase and by explant technique. Subsequently, cell yield, growth and differentiation potential, and tendon marker expression were compared.

3. Results

Isolation of MSCs by enzymatic tissue digestion yielded significantly more MSCs in a shorter period

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of time. No major impact of the isolation method on proliferation, migration, and differentiation behavior of the MSCs could be detected. Interestingly, compared with MSCs isolated by explant technique, a significantly higher expression level of the tendon marker scleraxis was found in MSCs isolated by collagenase digestion.

4. Discussion

Differences in cell characteristics caused by isolation methods could make enzymatically isolated MSCs advantageous for certain clinical applications. Furthermore, higher obtainable cell numbers could enable an efficient practice of enzymatically isolated MSCs.

Acknowledgment

This work was funded by the Mehl-Muelhens Foundation.

Dual-Axis Gene Therapy With the Use of Stem Cells Overexpressing Transforming Growth Factor-β3 in Combination With Interleukin-1β and Tumor Necrosis Factor-α RNA Silencing for Osteoarthritis Control in a Large-Animal Osteochondral Chip Fracture Model

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Intra-articular injection of mesenchymal stem cells co-expressing a growth factor and cytokine-suppressing genes was effective in reducing progression of osteoarthritis in this osteoarthritis model. This beneficial effect may occur by modulation of synovial fluid constituents, inflammation, cytokine profile, or direct cartilage repair. Authors' addresses: Texas A&M University, 4475 TAMU, College Station, TX 77843 (Watts); Cornell University, Department of Clinical Sciences, College of Veterinary Medicine, Ithaca, NY 14853 (Nixon); e-mail: awatts@cvm.tamu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Dual-axis gene therapy with anti-cytokine and anabolic growth factors may provide synergistic effects not apparent in single-target manipulation. The purpose of this study was to investigate the potential of intra-articular injection of mesenchymal stem cells (MSCs) overexpressing the anabolic transforming growth factor-β3 gene, concurrent with an RNA interference motif to suppress the catabolic interleukin (IL)-1β and tumor necrosis factor (TNF)-α cytokines, in an equine model for treatment of osteoarthritis (OA).

2. Materials and Methods

Early OA was induced in one middle carpal joint by osteochondral fragmentation in 13 skeletally mature Thoroughbreds. The contralateral joint was sham-operated. Second-passage autologous bone marrow–derived MSCs (10e6) were transfected with long hairpin silencing construct against IL-1β and tumor necrosis factor-α and transduced with Ad-transforming growth factor-β3. Treatments with either MSCs ($n = 6$) or placebo ($n = 7$) were injected into OA joints 14 days after OA induction. Sham joints were injected with placebo. Observations were a mix of blinded and nonblinded.

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3. Results

No adverse treatment effects were observed. When comparing MSC versus placebo-injected OA joints, there were significant improvements in range of motion and effusion in the week after injection, higher glycosaminoglycan (GAG) content of opposing third-carpal bone cartilage, significantly improved gene expression of cartilage matrix metalloproteinase-13 and synovial membrane IL-1β, and reduced synovial fibrosis.

From Paternalism to Partnership: Collaborative Care for Your Clients

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1. Introduction

Over the past decade, veterinarians have witnessed substantial changes in the profession. One of the major changes is the increasing recognition of the relationships that people may have with their companion animals.[1] When asked about their relationship with their pets, 85% of pet owners reported that they viewed their pets as family members.[2] In conjunction with this, there is a growing recognition that provision of veterinary services in a manner that acknowledges the human-animal bond will lead to better outcomes for veterinary practices and their patients.[1] Appreciating the impact of animal companionship on the health and well-being of humans creates a new dimension for veterinarians in public health. Veterinarians' responsibilities have expanded to include the mental health and well-being of their clients, as well as their clients' pets.[1]

In a recent address, Blackwell[1] stated that today's veterinarians are faced with educated clients armed with questions and greater expectations. Veterinarian's responsibilities for addressing questions and providing client education are increased. In an increasingly litigious society, consumers are not forgiving of unprofessional services.[1] Most complaints to regulatory bodies are related to poor communication and deficient interpersonal skills,[3] with breakdowns in communication being a major cause of client dissatisfaction.

An adaptive response is integral to successfully addressing these societal and professional changes. Given growing client expectations, the strong attachment between people and their pets and increasing consumer knowledge, demands a shift in communication style from the traditional paternalistic approach to a collaborative partnership. Clients are no longer content with taking a passive role in their animal's healthcare and want to take an active role in decision-making on their pet's behalf.

2. Models of the Veterinarian-Client-Patient Relationship

The balance of power between veterinarian and client is based on three criteria[4]:

(1) Who sets the agenda for the appointment (ie, the veterinarian, the veterinarian and client in negotiation, or the client).

(2) Importance placed on the client's values (ie, the veterinarian assumes that the client's values are the same as the veterinarian's, the veterinarian explores the client's values with the client, or the veterinarian does not explore the client's values).

(3) Functional role of the physician (ie, guardian, advisor, or consultant).

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On the basis of these criteria, three models of the veterinarian-client-patient relationship have been described.[5] At one end of the relationship spectrum lies paternalism, characterized as a relationship in which the veterinarian sets the agenda for the appointment, the veterinarian assumes that the client's values are the same as the veterinarian's, and the veterinarian takes on the role of a guardian. At the opposite end of the spectrum lies consumerism, which is characterized by a reversal of the traditional power relationship between veterinarian and client: the client sets the agenda for the appointment; the veterinarian does not explore the client's values; and the veterinarian plays the role of a technical consultant, providing information and services on the basis of the client's demands. The paternalism model has been criticized for ignoring the client's perspective, but the consumerism model errs in limiting the role of the veterinarian.

Between these two extremes is the relationship-centered care model, which represents a balance of power between veterinarian and client and is based on mutuality.[6,7] In the relationship-centered model, the relationship between veterinarian and client is characterized by negotiation between partners, resulting in creation of a joint venture, with the veterinarian taking on the role of advisor or counselor. Respect for the client's perspective and interests and recognition of the role the animal plays in the life of the client are incorporated into all aspects of care.

Principles of relationship-centered care are associated with significant outcomes:

(1) Broadening the explanatory perspective of disease beyond the biomedical to include lifestyle and social factors is related to expanding the field of inquiry and improved diagnostic reasoning and accuracy.[8,9]

(2) Building a strong relationship is associated with increased accuracy of data gathering,[9] patient satisfaction,[10-12] and physician satisfaction.[13,14]

(3) Encouraging participation, negotiation, and shared decision-making promotes patient satisfaction,[10,11] adherence,[15] and improved health.[16]

3. Shared Decision-Making

An interactive approach is promoted in giving information, in contrast to direct transmission.[9] With a direct transmission approach, the sender assumes that his or her responsibilities are complete once the message has been formulated and sent, whereas with an interactive approach, the interaction is considered complete only if the sender receives feedback about how the message was interpreted, whether it was understood, and what impact it had on the receiver.

Silverman et al[9] have recommended using a "chunk and check" method when giving information to avoid giving a one-sided speech and providing a large amount of information all at once. The aim of this technique is to increase recall, understanding, and commitment to plans. It consists of giving information in small pieces (ie, chunks), followed by checking for understanding before proceeding further (ie, check). In this manner, the information-giving process is responsive to the client's needs and provides an opportunity for the client to participate.

Taking the client's perspective into account and establishing mutual understanding and agreement encourages the client to fully participate in the discussion and commit to the treatment plans. This entails encouraging the client to contribute ("What questions do you have?"), picking up on client cues ("You seem a little hesitant about surgery"), asking for client suggestions ("What options have you and your husband discussed?"), and checking for the client's understanding ("What will be most difficult for you?"). Creating a shared understanding and encouraging shared decision-making enhances compliance and subsequently improves health outcomes.[16]

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Equine Lameness: Clinical Judgment Meets Advanced Diagnostic Imaging

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1. Introduction

When I graduated, it was accepted wisdom that lameness was assessed by watching horses move in hand. As an experienced rider, I became increasingly aware of the value of ridden exercise to assess lameness and performance. This has become an essential part of lameness and poor performance diagnosis. Diagnostic analgesia is now commonplace, but it is only comparatively recently that we have become aware of some of its limitations. Since I graduated, diagnostic imaging has evolved from radiography to include ultrasonography, nuclear scintigraphy, magnetic resonance imaging (MRI), and computed tomography. We are still learning how to interpret what we find and to integrate this with clinical findings. I aim to discuss the science and art of lameness diagnosis.

Accurate lameness diagnosis requires recognition of the lame limb(s), identification of any significant palpable abnormalities, determination of the source(s) of pain, and appropriate diagnostic imaging to identify the potential cause(s) of pain, with high quality images, correctly interpreted. This is an acquired art requiring careful observation, pattern recognition, a logical deductive process, and a questioning mind. By continuing to look, I believe that we can all continue to learn and to make new discoveries. My aim is to review some of the advances in lameness recognition, diagnostic analgesia, and advanced imaging, especially MRI. With respect to MRI, my focus is on the foot, because this is the area about which we have learned most and that has the best validation by comparison of imaging findings with postmortem examinations. However, throughout, I aim to emphasize the complexities and the many unanswered questions that remain. This is a challenging field.

2. Looking at the Horse and Learning to Read the Horse

The identification of lameness is an art, a skill that with practice and guidance can be enhanced. It requires an understanding of normal gaits and how they may be modified under a variety of circumstances. I use a simple grading system (0–8)[1] that is applied independently at a walk and a trot, in straight lines and in circles (on both soft and firm and/or hard surfaces and both to the left and to the right), and ridden (at rising trot [sitting on either the left or right diagonals] and sitting trot, and both to the left and the right, and if necessary when performing specific movements, eg, a 10-m diameter circle to the left, or half pass to the right). In my view, this gives a much more accurate picture of the lameness than the American Association of Equine Practitioners (AAEP) 0 to 5 scale,[2] especially if also embellished by some verbal description of the gait, for example, "a shortened cranial phase of the stride," "marked irregularity of rhythm" (appreciated by both visual assessment and listening to the gait), and "toe drag," or an "intermittent, hopping lameness." The system is effectively a numerical rating scale, because the grades have no definitions other than mild, moderate, and severe (0 = sound; 2 = mild; 4 = moderate; 6 = severe; 8 = non–weight-bearing). Thus, a subtle lameness may be grade 1, whereas an obvious lameness may be grade 5. This requires a good understanding of the breadth of gait abnormalities that may be encountered. I find it a workable system, because the manifestations of lameness vary so much among horses, depending on the source of pain and its severity, and each individual horse's response to that pain. Some features, such as extension of the fetlock, are more accurately assessed at a walk than at a trot because of the slower foot-fall. The longer stance phase at a walk,

and therefore the greater extension of the distal interphalangeal (DIP) joint, may make some foot-related lameness (eg, injuries of the deep digital flexor tendon [DDFT] or a collateral ligament of the DIP joint) more obvious at a walk than at a trot. In my opinion, a 0 to 8 system applied independently for each situation under which the horse is examined, supplemented by verbal qualifications, offers a far more flexible system than that of the AAEP grading scale and provides sufficient grades to cover a wide spectrum of gait abnormalities and facilitates grading of change in lameness after local analgesia.

With both forelimb and hindlimb lameness, it is useful to assess the horse walking in small circles, which often accentuates foot-related lameness but is not specific for foot pain. This also gives the opportunity to assess the flexibility of the horse's neck and back, the ease with which it crosses over its hindlimbs, and to detect any gait abnormalities suggestive of a component of ataxia, such as toe drag, hindlimb circumduction, leaving limbs in abnormal positions, irregularly sized steps, and interference between limbs. Evaluating the horse step backward can also give additional information about the presence of shivering and flexibility of the lumbosacral region.

Whichever lameness grading system is used, it is important to recognize that for consistency of results, the circumstances in which a horse is examined must remain the same. The speed of trot may influence the degree of lameness; some horses may try to slow the speed to protect themselves. Lameness may be accentuated on a slight downward slope compared with a horizontal surface or on a deeper sand surface than on a firm, waxed rubber and sand arena. Detection of these variations under different conditions may give an indication of the likely source of pain causing lameness. For example, hindlimb lameness that is worse on a circle on a hard surface is most likely to reflect foot pain.

Assigning a grade is not always straightforward because there are so many ways in which the gait may be modified. When a horse with unilateral hindlimb lameness is moving in straight lines, there is usually some degree of asymmetry of movement of the hindquarters. There may be reduced extension of the fetlock joint, reduced flexion of the hock, toe drag, or alteration in stride length; the horse may move on three tracks, usually drifting away from the lame limb; alternatively, the lame hindlimb may deviate axially during protraction or less commonly is swung outward during protraction. The rhythm may be altered both audibly and visually. Irrespective of the degree of hindquarter asymmetry, there may be a head-nod mimicking ipsilateral forelimb lameness.

Our ability to detect asymmetry of movement of the hindquarters is limited. A computer model was devised to determine how capable we are of assessing hindlimb lameness, on the basis of evaluation of movement of the tubera coxae.[3] The model had two forms, one in which there was random asymmetry of movement between two objects and the other that simulated the patterns of movement seen in a lame horse. In the first model, no differences were seen in the skill of inexperienced assessors compared with experienced clinicians, suggesting that there are no innate differences in the ability to detect asymmetry. However, the experienced clinicians performed best with the real lameness-based data. Nonetheless, even with lameness-based data the accuracy of detecting asymmetry of movement simulating low-grade lameness was poor: asymmetries in movement of $<25\%$ could not be detected. In a more recent study of so-called normal horses, assessed by veterinarians with a range of experience, and also objectively with the use of inertial measurement units, an expert was able to detect lameness manifest as asymmetries of 10%.[4] However, the expert was not confined to assessing asymmetry alone and could use any technique to determine whether the horse was lame or sound.

I focus on both the tubera coxae and the tubera sacrale, although it may be impossible with a horse with a naturally high tail carriage, such as Arabians, some Warmbloods, and gaited horses, when the tail conceals the tubera sacrale. A skewbald or piebald horse with one white hindquarter and one colored hindquarter, or a horse with unilateral gluteal muscle atrophy in either the lame or the non-lame limb, or a horse with asymmetry in height of the tubera sacrale, or an excitable horse that will not trot straight potentially confound our interpretation. A bilaterally symmetrical hindlimb lameness may manifest merely as a short stride, stiffness, and lack of hindlimb impulsion. All of these factors potentially compromise our ability to detect the lame limb(s). Some can be solved—an excitable horse can either be worked or sedated, but you cannot change the markings of a colored horse or the positions of the tubera sacrale. The use of markers placed on the tubera coxae may facilitate assessment.[5]

With forelimb lameness, there is usually abnormal movement of the head and neck, although in a horse with a naturally short-striding gait, with a tendency to roll from side to side, this may be difficult to discern. Assessment of head and neck posture is also important for detection of neck pain that can result in alterations in gait.

I believe that it is important to assess the horse moving from behind, from the front, and from the side to assess all aspects of the gait and to both watch and listen to the horse. Irregularities of gait may be emphasized by listening to the limbs striking the ground or hearing a toe drag during protraction. An abnormally deliberate placement of hindlimbs to the ground may suggest a neurological component to the gait abnormality or a mechanical component such as stringhalt or fibrotic myopathy. Assessment of foot placement—toe first or flat-footed, lateral side first or flat, in line with the ipsilateral foot

or to one side—and breakover may reflect the site of pain or help to explain why lameness may have developed. Viewing the horse from behind may reveal that a hind foot is placed axially and the horse appears to collapse laterally over the fetlock, with an unusual wobble of the hock. This may be normal for the horse but may be a predisposing cause for lameness. Abaxial sliding of a hind foot during landing may reflect ataxia. Axial deviation of a hindlimb during protraction may be a way of reducing proximal limb flexion associated with lameness.

Careful observation of the horse from the front at the walk may reveal slight bulging of a shoulder: "shoulder-slip," usually a manifestation of brachial plexus injury. Watching the hocks carefully from behind may reveal intermittent, slight movement of the superficial digital flexor tendon (SDFT) reflecting subluxation secondary to a partial tear of the retinaculum. Unequal height of the hocks may reflect partial disruption of gastrocnemius or coxofemoral joint subluxation.

No grading system can take into account a bilaterally symmetrical lameness. A lameness grade ascribed to the lamer limb or lamest limb in the presence of an asymmetrical bilateral lameness or concurrent forelimb and hindlimb lameness can be potentially highly misleading. With a bilaterally symmetrical forelimb lameness, the horse may show only a subtle shortening of stride. Abolition of pain in one limb by local analgesia may reveal either a low-grade lameness in the contralateral limb or a moderate to severe lameness, but this can be highly unpredictable on the basis of the initial clinical assessment.

Many but not all forelimb lamenesses are accentuated on a circle. I prefer to see a horse lunged on a circle rather than led, because it is easier to assess any adaptations of the horse's balance, posture, and rhythm. Although historically it has been assumed that most horses with foot pain have lameness accentuated with the lame(r) limb on the inside of a circle, approximately one-fifth of 718 horses were lamest with the lame(r) limb on the outside of a circle.[a] Forelimb lameness associated with proximal suspensory desmitis is usually worse with the lame(r) limb on the outside of a circle on a soft surface. Bilateral forelimb lameness sometimes manifests as a shortness of stride, hurried rhythm, loss of balance, and a tendency to look out of a circle. Foot-related pain and some other sources of pain causing lameness may be worse on a firm or hard surface compared with a soft surface. However, selection of an appropriate hard surface is crucial because the horse must move confidently; a slippery surface may result in marked shortening of stride and apparent loss of balance in a clinically normal horse, especially those that move extravagantly. I use a gravel surface on an incline immediately adjacent to purpose-built modified tarmac; the gravel provides excellent grip, and the downward slope often accentuates lameness. A horse that trots sensibly can be assessed moving from the gravel to the harder tarmac surface and back onto the gravel. A horse may move with a rather restricted stride, and, if asked to move forward more freely, may break to canter rather than increase the stride length. This is usually a manifestation of hindlimb lameness but can also reflect forelimb lameness.

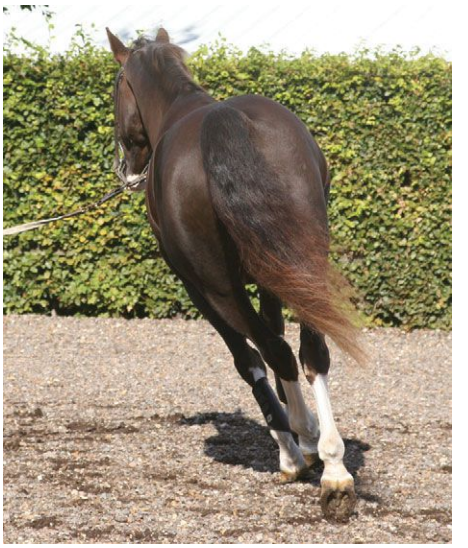


Fig. 1. A medium level 7 year old Warmblood dressage horse being lunged to the left. The left hindlimb is crossing in under the body during protraction, toward the contralateral forelimb, crossing in front of the right hindlimb. The horse is leaning in, looking to the outside, reflecting a bilateral hindlimb lameness (left>right) associated with proximal suspensory desmopathy. No gait abnormalities were detected when the horse was trotted in hand.

Evaluation of the horse moving in circles on the lunge can also be helpful for assessment of hindlimb lameness, with some lamenesses becoming more evident with the lame hindlimb on the inside of a circle and some with the lame hindlimb on the outside of a circle, and may change depending on the circumstances under which the horse is assessed. In my experience, this is not necessarily related to the source of pain causing lameness. However, with mild lameness that is not modified by being on a circle, detection of hindlimb lameness may be more difficult than in straight lines. The pelvis tilts inward on the lunge, therefore comparison of movement of the tubera coxae is more difficult than in straight lines. Lameness may modify the horse's body posture, with a tendency to lean in so that the body is no longer perpendicular to the ground (Fig. 1). This makes evaluation of pelvic symmetry even more difficult, but alterations in rhythm may make it easier to detect the lame limb. The lame hindlimb may cross in under the horse's body when on the inside of a circle and may have an accentuated toe drag. When on the outside of a circle, a



Fig. 2. A six year old showjumper mare (A) on the lunge and (B) ridden in left canter. Note the horse's croup high posture. In canter to the left and to the right the horse felt on the forehand and very stiff in the back. She was unwilling to engage the hindlimbs. In trot the horse stepped slightly short behind and lacked hindlimb impulsion. The horse had bilateral proximal suspensory desmitis and sacroiliac joint region pain. Her performance was dramatically improved after bilateral perineural analgesia of the deep branch of the lateral plantar nerve and infiltration of local anaesthetic solution around the sacroiliac joints.

shortened cranial phase of the stride of the lame hindlimb may be exaggerated.

It is also crucial to recognize the influence that forelimb lameness can have on the hindlimb gait and the effect that hindlimb lameness can have on forelimb gait. It is well recognized that hindlimb lameness can induce a head-nod mimicking ipsilateral forelimb lameness.[6] It is less well recognized that forelimb lameness may induce asymmetry of the hindlimb gait. By observing the horse under a variety of circumstances, it may be possible to differentiate between genuine concurrent forelimb and hindlimb lameness and an apparent lameness that is secondary. However, it may or may not be possible to determine whether there is concurrent forelimb and hindlimb lameness without using local analgesia. Thus, each lameness must be graded independently initially, in the knowledge that abolition of pain in, for example, the hindlimb may abolish both the hindlimb lameness and the apparent (secondary) forelimb lameness.

Assessment of a horse moving on the lunge can help to determine if a head-nod present in conjunction with a hindlimb lameness is related to the hindlimb lameness or reflects a forelimb lameness. If the hindlimb lameness deteriorates on the lunge but the head-nod remains unchanged, or if the head-nod is worse but the hindlimb lameness stays the same, it can probably be concluded that there is coexistent forelimb and hindlimb lameness. This requires verification with the use of local analgesic techniques, further discussion of which is beyond the scope of this commentary.

Although overt lameness is rarely seen on the lunge in canter, assessment of canter and transitions from trot to canter and canter to trot can give useful additional information. A bilateral forelimb lameness may manifest as a short-striding, jarry canter. Hindlimb lameness may result in the hindlimbs being placed closer together than normal, so-called "bunny-hopping" when extreme. There may be reduced extension of the distal limb joints of the lame hindlimb, especially with the lame limb on the inside of a circle. The fetlock may appear to knuckle forward slightly during the stance phase, with incomplete loading of the heel. Alternatively the horse may be on the forehand and croup high in canter (Fig. 2). Curiously this gait adaptation is sometimes also seen in a horse with bilateral forelimb lameness. The horse may repeatedly become disunited (ie, change legs behind, while maintaining the correct forelimb lead); however, this may be a normal feature of a young unbalanced horse that lacks hindlimb strength and coordination. Irregular steps behind in a transition from trot to canter or from canter to trot are a manifestation of hindlimb lameness. Stepping short behind and maintaining a croup-high posture in a downward transition may reflect bilateral hindlimb lameness.

Mild hindlimb lameness may still be barely detectable, and assessment of the horse ridden may be crucial. It is important to recognize the influence of the weight of a rider on hindlimb gait, especially when in rising trot. The lameness is usually accentuated when the rider sits on the diagonal of the lame hindlimb and the horse feels more uncomfortable to the rider on this diagonal. The horse may adjust its rhythm to try to shift the rider back to the opposite diagonal. A heavier rider is more likely to accentuate lameness than a lighter rider. A subtle lameness may still be unapparent and riding consecutive 8- to 10-meter diameter circles to the left and to the right may induce irregularities of rhythm reflecting lameness and a change in the speed, with the horse slowing down when uncomfortable. Likewise, performing so-called lateral movements such as shoulder-in, travers, and half-pass may induce gait irregularities that are otherwise not apparent. In some horses, the only manifestation of a hindlimb gait abnormality may be a tendency to change limbs



Fig. 3. A, Rear view of a 9 year old Prix St Georges Warmblood dressage horse, with a low-grade left hindlimb lameness, most apparent in left half-pass. The saddle has slipped to the left (toward the side of the lame limb), a persistent feature on the left rein in both trot and canter. Saddle slip was resolved when the lameness was abolished by diagnostic analgesia. B, Rear view of a small riding horse, with left hindlimb lameness. There is saddle slip to the right on the left rein, which was worse in circles than in straight lines. Abolition of the lameness by diagnostic analgesia resolved the saddle slip.

behind in canter, to become disunited. Alternatively, the horse may have difficulties in performing canter pirouettes in one direction, or flying changes either from left to right, or from right to left. It is important to be aware of the influence of a rider; an unskilled rider who lacks balance may induce lameness, whereas a highly skilled rider may conceal lameness. A rider who is constantly moving their hands may cause irregular movement of the horse's head, which may mimic forelimb lameness. A rider who restricts the horse with their hands may cause a loss of hindlimb propulsion and/or irregular steps. Unless the rider is sufficiently skilled, lameness that is only apparent when a horse is working in maximum collection may not be apparent. Evaluation of the horse working to a contact, "on the bit" compared with on a long rein, may reveal that lameness is apparent under one circumstance but not the other.

The tendency of the saddle to consistently slip to one side may be a manifestation of hindlimb lameness,[7] with the saddle usually slipping to the side of the lame(r) limb but sometimes slipping to the side of the non-lame or less lame limb (Fig. 3). This presumably reflects the different ways that horses modify their gait in the face of hindlimb lameness. Saddle slip with two riders was present in 38 of 71 (54%) horses with hindlimb lameness and was abolished when lameness was resolved with the use of diagnostic analgesia in 37 of 38 (97%) horses, verifying a causal relationship.[7] The saddle slipped to

the side of the lamest limb in most horses (32/37 [86%]). Saddle-slip persisted in one horse in which the flocking of the saddle was asymmetrical. When ridden in a better-fitting saddle, no saddle-slip was observed. Saddle-slip associated with hindlimb lameness was not related to the degree of lameness. The observation of saddle-slip may actually highlight the presence of lameness. Saddle-slip was usually worse in rising trot than in sitting trot, in circles compared with straight lines (irrespective of the appearance of the lameness), in canter than in trot, and with a lighter rider compared with a heavier rider. No horse had significant left-right asymmetry of the shape of the thoracic region. However, it is important to recognize that saddle slip may also be induced by an ill-fitting saddle, crookedness of the rider, or asymmetry of the horse's back.

The way in which a multilimb lameness may influence a horse's performance when ridden must also be understood. There may be no overt lameness; therefore grading the degree of lameness may be impossible, although the gait of the horse is altered and performance compromised. For example, there may be no detectable lameness, but on the lunge a horse may lean the body inward and look to the outside, while the inside hindlimb crosses in under the body toward the contralateral forelimb during protraction. This may well be a manifestation of lameness, but is not quantifiable and can only be described. It is also not specific for lameness, because a young unbalanced horse, lacking musculoskeletal strength and coordination, may show a similar gait. When ridden, the same horse with poor performance may lack hindlimb engagement and impulsion and tend to be croup-high in downward transitions. Superficially, to an inexperienced eye, the horse may appear normal and the appearance may markedly underplay the discomfort experienced by the horse. Leaning on the bit, taking an uneven contact, tilting the head (Fig. 4), opening the mouth, raising the head, becoming overbent, stiffness in the neck or back, crookedness, difficulties to turn in one direction, reluctance to go forward or undue hurrying, evasiveness, and spookiness can all be manifestations of lameness. The complete transformation of balance, impulsion, engagement, quality of contact with the bit, and willingness to work after appropriate local analgesia to abolish subclinical lameness will emphasize the degree of pain that the horse is experiencing.

Problems that are only evident ridden have frequently been attributed to pathology in the thoracolumbar-sacral spine. Primary back pain may induce back stiffness and limited hindlimb impulsion or a restricted gait all round. However, lameness can induce similar symptoms. Likewise, neck stiffness is often attributed to neck pain but can also be a manifestation of lameness. Comparison of a horse ridden in rising and sitting trot can help to identify a component of primary back pain. In sitting trot, a horse with back pain is more likely to alter its rhythm and/or speed and alter the position of the head and neck, becoming slightly above the bit, compared with a lame horse.



Fig. 4. A 7 year old Thoroughbred-cross, amateur-owned novice event horse. The horse had become difficult to ride, being unwilling to bend correctly. The horse is on the right rein, but is leaning in and looking slightly to the outside with the nose tipped to the right. The bit has moved so that there is more on the right side. No overt lameness was detected in hand; the horse had low-grade right forelimb and bilateral hindlimb lameness evident when ridden. When lameness was abolished by local analgesia, the horse was much more compliant and took an even contact on both reins, and bent easily in the correct direction on both reins. The horse is being ridden in draw (running) reins because otherwise it would intermittently throw its head up and charge forward.

Some lameness may only be apparent under specific circumstances. For example, with forelimb lameness, the horse may be unwilling to land with the left forelimb leading in canter. The ground reaction force is greater in the trailing (non-lead) forelimb on landing,[8] so unwillingness to land with the left forelimb leading is protecting the right forelimb from concussion. However, there is greater stress on the suspensory apparatus of the leading forelimb, so unwillingness to land with the left forelimb leading could reflect suspensory injury in the left forelimb. Pushing off the hindlimbs unevenly can cause a horse to jump crookedly across a fence, with the hindlimbs drifting toward the lame limb. With low-grade hindlimb problems, lameness may manifest as unwillingness to perform flying changes from left to right in canter (or vice versa) or inability to maintain a regular rhythm in piaffe, passage, or pirouettes. The hindlimbs may be unable to sup-

port weight normally, so there may be a tendency to place them more closely together in canter. Particularly when the lame limb is on the inside of a circle, it will not be protracted as far as normal and will therefore be placed closer than normal to the outside hindlimb. A stiff, stilted canter or poor hindlimb propulsion in canter, with the hindlimbs trailing, may be the most obvious manifestations of a bilateral hindlimb lameness.

There are also some specific lamenesses that are generally only evident when a horse is ridden. There is a hopping-type forelimb lameness, characterized by a shortened cranial phase of the stride and a marked lift of the head and neck as the lame forelimb is protracted. It can vary in degree within a work period and may disappear if the horse is ridden on a long rein. It is often but not invariably worse with the affected limb on the outside of a circle. It may be sensitive to the diagonal on which the rider sits. Such lameness is often refractory to any diagnostic analgesic technique, is non-responsive to non-steroidal anti-inflammatory drugs (NSAIDs), and comprehensive investigation with the use of diagnostic imaging usually fails to identify a cause.

Unilateral primary strain of a braciocephalicus muscle may manifest as lameness only discernible when the horse is ridden at walk. It is characterized by lifting of the head and neck as the limb is protracted and a shortened cranial phase of the stride. Some horses with injuries of the tendon of biceps brachii have also shown lameness only when ridden, which was markedly more severe at the walk than at the trot. Low-grade shoulder-slip may be more obvious at a walk than at faster gaits and when ridden compared with in-hand.

There is a lameness typified by a shortened cranial phase of the stride of one hindlimb at the walk, when ridden "on the bit" (or driven) but not when ridden (or driven) on a loose rein, nor when lunged, even with side reins adjusted to simulate the position of the horse's head and neck when working "on the bit." No lameness is detectable at other gaits. This lameness is also refractory to any diagnostic analgesic technique, is non-responsive to NSAIDs, and comprehensive investigation with the use of diagnostic imaging usually fails to identify a cause. Prolonged rest does not alter the problem, nor does physiotherapy, acupuncture, chiropractic treatment, or osteopathic treatment.

There remains a small but important group of horses in which even a highly skilled and experienced lameness clinician cannot see lameness, but a skilful rider can feel lameness and/or compromise in performance. I never cease to be amazed by the large degree of apparent asymmetry of movement I can feel when riding a horse that I cannot detect by visual appraisal. However, it must also be recognized that some highly successful competition riders are apparently unable to feel even quite obvious lameness, whereas some far less talented riders do have the ability to feel even low-grade lameness.

Ridden exercise is not, however, a substitute for assessment in hand and on the lunge, because there are some lamenesses, especially forelimb lameness, which may only be apparent in hand and /or on the lunge. It is curious that even when ridden on a long rein under identical circumstances, such lameness may be concealed. It should also be borne in mind that a horse that shows lameness that is evident when trotted in hand, which can be abolished with the use of diagnostic analgesia, may also demonstrate another lameness due to an unrelated cause when ridden.

Low-grade hindlimb ataxia can mimic hindlimb lameness, and hindlimb lameness and ataxia may coexist. Concurrent existence of hindlimb lameness and low-grade ataxia can confound interpretation. Horses with mild ataxia may have a bouncy, croup-high, and stiff-legged hindlimb gait when decelerating from trot to walk, with steps of irregular height. There may be more deliberate placement of the hind feet to the ground, with sideways movement of the feet. There may be a toe-drag and lack of hindlimb propulsion. If turned in small circles, there may be a toe-drag, abnormal limb placement, delayed movement of limbs, circumduction of the outside hindlimb, and sometimes one limb may collide with another. Using a tail-pull test at the walk may highlight the presence of weakness. An affected horse may have a croup-high canter on the lunge.

There are some gait characteristics that can be highly suggestive of the primary source of pain causing lameness. For example, a short pottery forelimb gait, soreness when turning, and accentuation of the lameness on a firm surface on the lunge are features highly suggestive of foot pain. A forelimb lameness that is worse with the affected limb on the outside of a circle on a soft surface is suggestive of proximal suspensory desmitis. A forelimb lameness that is markedly accentuated by carpal flexion is likely to reflect carpal pain. However, many gait modifications are non-specific, and horses may adapt their gaits differently despite similar sources of pain. Flexion tests are also non-specific. Whereas exacerbation of lameness after fetlock flexion may reflect fetlock region pain, lameness associated with proximal suspensory desmitis may also be accentuated, presumably because of release of tension on the suspensory apparatus during flexion and then sudden loading when the limb is placed to the ground.

The importance of clinical observation cannot be over-emphasized—looking *and* seeing; assessment of stance at rest, conformation, balance and symmetry; systematic palpation; determination of the baseline lameness; and the response to flexion tests. At this stage in an investigation, it may be possible to determine accurately a potential source of pain causing lameness, but in many cases local analgesia

will be required. Although careful assessment of gait may be suggestive of the primary source of pain causing lameness, in my experience, many of the findings are non-specific and the same injury may be manifest differently among horses. Moreover, more than one source of pain may coexist in one limb, therefore accurate diagnosis can only be achieved with the use of diagnostic analgesia. The gait characteristics may change after abolition of one source of pain if another source coexists. For example, a horse had a low-grade bilateral forelimb lameness evident only on the lunge on a firm surface or ridden, as mild (grade 2/8) left forelimb lameness on the left rein and subtle (grade 1/8) right forelimb lameness on the right rein. After palmar digital nerve blocks of the left forelimb, right forelimb lameness was accentuated (grade 3) on the right rein, but only when ridden. Palmar digital nerve blocks of the right forelimb abolished this lameness, but then a more severe right forelimb lameness (grade 4) became apparent on the left rein when ridden. This was ultimately abolished by palmar metacarpal (subcarpal) nerve blocks. Foot pain and proximal suspensory desmitis coexisted.

There are differences of opinion, reflecting personal experiences about whether a horse is lame enough to block, for example, is it likely that the observer would be able to detect an improvement if lameness was abolished? This can certainly be tough if a horse with subtle lameness is only assessed in-hand and on the lunge. However, it must always be borne in mind that subtle lameness may reflect bilateral lameness and if pain is abolished in one limb a much more obvious lameness may become apparent in the contralateral limb. If a horse is also assessed ridden and other aspects of performance are considered together with lameness, I believe many horses with subtle lameness can be nerve-blocked with meaningful results. This is likely to be much more rewarding than resorting to survey radiography or nuclear scintigraphic examination, which often yield false-negative or false-positive results.

Nerve blocks must be performed in a systematic way; there are few shortcuts, and this time-consuming procedure cannot be rushed without risks of misinterpretation of the results. However, on the basis of the findings of an initial clinical assessment, a logical decision can be made about where to start. For example, if there is a markedly positive response to distal limb flexion, then intra-articular analgesia of the fetlock may be performed, bearing in mind the potential to influence closely related anatomical structures, such as the suspensory ligament branches. In a hindlimb, in the absence of clinical signs related to the fetlock and more distal aspects of the limb, it would be reasonable to start by perineural analgesia of the plantar (at the junction of the proximal three-quarters and distal one-quarter of the metatarsus) and plantar metatarsal (distal to the "button" of the second and fourth metatarsal bones) nerves (a "low four-point-block"). If a horse showed lack of hindlimb propulsion but no detectable lameness, bilateral perineural analgesia of the deep branch of the lateral plantar nerve may result in substantial improvement in the horse's performance when ridden, whereas a unilateral block may confusingly result in little change. However, the distal aspect of the limb should first be excluded as a potential source of pain by use of a "low four-point-block."

Nerve blocks can paradoxically result in an increase in lameness severity. If the foot is desensitized and it is not the source of pain causing lameness, lameness may deteriorate. This is a non-specific finding but is often seen in association with suspensory ligament pain. I believe that the foot serves a proprioceptive function and the horse can modify its gait to reduce pain. Desensitization of the foot reduces its proprioceptive function, and the horse is less able to adapt its gait to reduce loading and therefore increased strain is placed on the suspensory apparatus, resulting in accentuation of lameness.

Thus, interpretation of the response to local analgesia is not always straightforward. How much improvement is expected after apparent desensitization of a single source of pain? This depends on both the severity of the pain, the cause(s) of pain, and how that pain is mediated and whether there may be a mechanical component to the lameness. For example, severe foot pain associated with a subsolar abscess, a fracture of the distal phalanx or navicular bone, laminitis, or adhesions of the DDFT may be unaffected or only partially improved by palmar nerve blocks performed at the base of the proximal sesamoid bones. Pain associated with a neuroma may be minimally influenced by perineural analgesia. However, it must also be borne in mind that more than one source of pain may coexist, and more proximal nerve blocks may be required. This is where the art and science of lameness diagnosis must be combined.

3. Some Further Observations Concerning Local Analgesia

Diagnostic analgesia is frequently required to localize the site(s) of pain causing lameness, but it is crucial to be aware of the limitations of the techniques used and how confusion may arise, a problem I first addressed in 1986.[9] It is well-recognized that perineural analgesia may abolish pain distal to the sites of injection, but at some sites proximal diffusion of local anesthetic solution may result in abolition of pain at sites considerably proximal to the site of injection. Pain associated with the proximal interphalangeal (PIP) joint was induced by injection of bacterial lipopolysaccharide, and baseline lameness was recorded.[10] Local anesthetic solution (1.5 mL per site) was injected around the palmar digital nerves at sites 1, 2, and 3 cm proximal to the cartilages of the foot (ungular cartilages). The

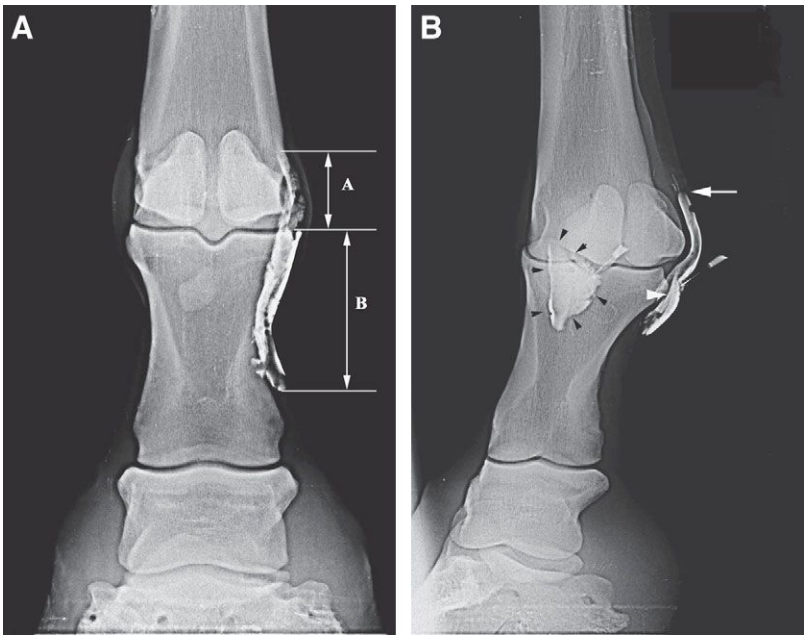


Fig. 5. A, Dorsopalmar radiographic image of a left forelimb, obtained immediately after subcutaneous injection of 2 ml radiodense contrast medium over the lateral palmar nerve at the base of the lateral proximal sesamoid bone. There is an elongated distribution of the contrast medium and marked proximal and distal diffusion, within the fascia surrounding the neurovascular bundle. The extents of proximal (A) and distal (B) diffusion were measured from a line drawn perpendicular to the medial/lateral aspect of the proximal articular surface of the proximal phalanx, to the most proximal/distal aspect of the contrast 'patch'. B, Dorsomedial-palmarolateral radiographic image of a right forelimb, obtained immediately after subcutaneous injection of 2 ml radiodense contrast medium over the medial and lateral palmar nerves at the base of the medial and lateral proximal sesamoid bones. The needles were left in position. Note the elongated distribution of the contrast medium and the marked proximal diffusion from the tip of the needle (white arrowhead) within seconds after injection at the medial injection site (right side of the image, white arrow). At the lateral injection site (left side of the image), the distribution of the contrast medium was more diffuse (black arrowheads), compared with the elongated pattern laterally, suggesting that it was outside the fascia surrounding the neurovascular bundle. (Reproduced from *Equine Vet J* 2009;41:379–383, with permission).

median lameness score improved after injection at the two most proximal sites. I have seen 80% improvement in lameness associated with severe subchondral bone trauma of the proximal aspect of the middle phalanx with the lesion communicating with the PIP joint, or in association with advanced osteoarthritis (OA) of the PIP joint, within 10 minutes of perineural analgesia of the palmar digital nerves immediately proximal to the cartilages of the foot. Moreover, it has been reported that perineural analgesia of the palmar digital nerves may resolve lameness associated with lesions of the metacarpophalangeal joint.[11]

After perineural injection of 2 mL of a radiodense contrast medium (iohexol) around the palmar nerves at the base of the proximal sesamoid bones, there was immediate proximal spread of the contrast medium detected radiologically, and, within 10 minutes, the mean proximal spread was 16.9 ± 13.7 mm and further spread up to 30 minutes after injection (mean, 20.8 ± 15.1 mm) (Fig. 5A).[12] Spread was not significantly different in horses that stood still or those that were walked after injection. Diffusion is influenced by molecular weight, and iohexol has a larger molecular weight than mepivacaine; thus, mepivacaine may diffuse further. This explains clinical observations that horses with incomplete fractures of the sagittal groove of the proximal phalanx often have lameness that is abolished by perineural analgesia of the palmar nerves performed at the base of the proximal sesamoid bones (Fig. 6).[13] Lameness associated with injuries of the palmar annular ligament or the insertion of the suspensory ligament branches, the fetlock joint, and the digital flexor tendon sheath may be similarly abolished.

Distribution of contrast medium in a patch outside the neurovascular bundle occurred occasionally (Fig. 5B), or proximal spread occurred in a lymphatic vessel.[12] These observations may explain in a clinical setting either a delayed onset of analgesia or false-negative results of palmar nerve blocks performed at the base of the proximal sesamoid bones.

Perineural injection of iohexol around the medial and lateral palmar nerves at the junction of the proximal three-quarters and distal one-quarter of the metacarpus and over the palmar metacarpal nerves immediately distal to the second or fourth metacarpal bones, simulating a "low four-point block," resulted in significant proximal diffusion



Fig. 6. Dorsoproximal-palmarodistal oblique radiographic image of the right forelimb of a 9 year old Warmblood. Lameness was substantially improved by palmar nerve blocks performed at the base of the proximal sesamoid bones. There is a small incomplete fissure in the sagittal groove of the proximal phalanx, extending into a more diffuse area of reduced opacity (arrow) distal to which is an area of increased opacity (arrowheads). This lesion was associated with focal intense increased radiopharmaceutical uptake.

over time but never proximal to the mid-metacarpal region.[14] Distribution of the contrast medium within the neurovascular bundle was achieved in only 77.5% of sites compared with 89% when injecting at the base of the proximal sesamoid bones (Fig. 7).[12] There was no difference in successful injection into the neurovascular bundle with injections performed in a weight-bearing or a non–weight-bearing position. Delayed desensitization or failure to achieve analgesia may occur if injection is not into the neurovascular bundle. The most proximal extent of the contrast medium, measured from the tip of the needle in the radiograph obtained immediately after performing the palmar injection, was 80.9 mm.[14] The most proximal extent of a palmar patch of contrast medium was measured as 156 mm proximal from the most distal aspect of the sagittal ridge of the third metacarpal bone. These results suggest that structures in the mid-metacarpal region (superficial and deep digital flexor tendons and the accessory ligament of the DDFT [ALDDFT]) could be desensitized after a "low four-point nerve block," but it is unlikely that diffusion of local anesthetic solution can be responsible for desensitizing structures in the proximal metacarpal region. However, the proximal-distal location of the injection site for anaesthetizing the palmar nerves obviously has an effect on the extent of proximal diffusion. There was significant proximal diffusion with time, indicating that the longer the time that elapsed between a perineural block and the assessment of its effect, the more likely it is that proximal structures are desensitized. Depending on the location of the distal extremity of the second and fourth metacarpal bones, the proximal margin of the patch of contrast medium after injection of the palmar metacarpal nerves was sometimes seen between the distal one-third and one half of the metacarpal region. It is therefore unlikely that after a palmar metacarpal injection immediately distal to the distal aspect of the second and fourth metacarpal bones, diffusion would result in improvement of pain in the proximal metacarpal region. The explanation for a low four-point block improving lameness associated with proximal suspensory desmitis[15] may be caused by painful lesions in the suspensory ligament extending further distally than can be appreciated ultrasonographically or by the presence of a concurrent site of pain in the fetlock region.

Paradoxically, in some horses with subchondral bone pain in the palmar aspect of the condyles of the third metacarpal bone associated with extensive mineralization, lameness is improved but not abolished by a "low four-point block".[b,c] Palmar metacarpal nerve blocks performed just distal to the carpus are required to resolve the lameness, although no lesions in the carpus or proximal metacarpal region have been identified through the use of radiography, ultrasonography, and MRI. It is presumed that there are nerves that enter the third metacarpal bone proximal to the sites of injection for the "low four-point block," which require anesthesia to abolish pain and lameness. Similar observations have been made in hindlimbs.

In two of 40 limbs in the "low four-point block" study, there was radiodense contrast medium within the digital flexor tendon sheath (DFTS) (Fig. 7), which raises concerns about potential iatrogenic synovial sepsis.[14] The injection sites were not clipped before injection, and, although the sites were thoroughly cleaned, the limbs were not prepared for aseptic injection. Neither of the two horses whose DFTS was penetrated developed effusion of the DFTS or lameness after the study, but we recommend extra care (thorough cleaning and clipping if the hair does not permit adequate cleaning) when preparing for blocking the palmar nerves when performing a low four-point nerve block. To minimize the risk of iatrogenic infection of the DFTS, the palmar injection should be performed proximal to the proximal extent of the DFTS. This, depending on the degree of DFTS effusion may, however, run the risk of desensitizing more proximal structures. Since performing this study, I saw one horse with no palpable distension of the DFTS that developed sepsis of the DFTS within 15 hours of a low four-point block.

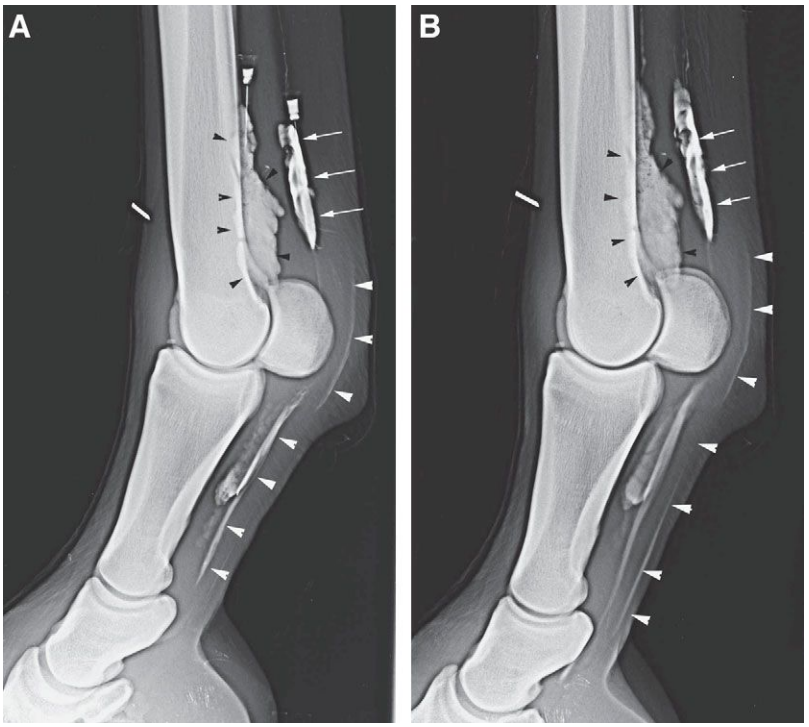


Fig. 7. Lateromedial radiographic views of a left forelimb of a live horse obtained 0 (A) and 10 (B) minutes after subcutaneous injection of 2 mL contrast medium over the medial palmar nerve at the junction of proximal 3/4 and distal 1/4 of the metacarpal region and over the medial palmar metacarpal nerve immediately distal to the distal aspect of the second metacarpal bone. The injections had been performed with the limb in a non weight-bearing position. A radiodense marker was placed dorsally at the junction of the proximal 3/4 and distal 1/4 of the metacarpal region. At the palmar injection site, the proximal aspect of the contrast patch has an elongated pattern and seems to be in the neurovascular bundle (arrows), but the contrast has also spread markedly distally, in a pattern that most likely represents injection or diffusion of the contrast medium into the digital flexor tendon sheath (DFTS) (white arrowheads). Note that in the radiograph obtained 10 minutes after injection (B), the dorsal wall in the distal aspect of the DFTS is better defined by the contrast medium. Also note that the proximal end of the palmar patch of contrast medium is more proximal on the second than on the first radiograph, indicating proximal diffusion with time. The contrast medium is diffusely distributed around the palmar metacarpal injection site (black arrowheads). (Reproduced from *Equine Vet J* 2010;42:512–518, with permission).

Intra-articular analgesia of the fetlock joint or intrathecal analgesia of the DFTS might be considered to be potentially more specific than perineural analgesia. However, there is the potential to influence structures in close proximity to these synovial structures resulting in false-positive results. Intra-articular analgesia of the fetlock joint may abolish pain associated with lesions of the suspensory ligament branches or the origin of the oblique or straight sesamoidean ligaments. Intrathecal analgesia of the DFTS can also alleviate pain associated with injuries of the straight or oblique sesamoidean ligaments or branches of the SDFT. Intra-articular analgesia of the fetlock joint can also result in false-negative results in the presence of subchondral or trabecular bone pain.

Clinical observations and cadaver studies have demonstrated the potential lack of specificity of a variety of techniques for analgesia of the proximal palmar aspect of the metacarpus.[16] This has been further highlighted by an in vivo study that used the radiodense contrast medium model.[17] After perineural injection of the medial and lateral palmar metacarpal nerves distal to the carpus from either the medial and lateral aspects, or from the lateral aspect alone, there was radiodense contrast medium in the carpometacarpal and middle carpal joints in 50% of limbs and 12.5% of limbs, respectively. Thus, it is important to evaluate both the proximal metacarpal region and the carpus with the use of diagnostic imaging after a positive response to subcarpal analgesia of the palmar metacarpal nerves. Although this lack of specificity is most important in racehorses, which have a high incidence of carpal disease, there is increasing recognition of the importance of carpal pain in sports horses as well.[18–20] With a lateral approach to the lateral palmar nerve, there is a small but significant risk of inadvertent penetration of the carpal sheath,[16,17] which may result in false-negative responses to diagnostic analgesia. With a medial approach to the lateral palmar nerve, there is a risk of proximal diffusion of local anesthetic solution into the distal one-third of the antebrachium and spread around the median nerve and the caudal branch of the ulnar nerve (Fig. 8).[17] This may result in desensitization



Fig. 8. Lateromedial radiographic image acquired immediately after injecting radiodense contrast medium using a medial approach to the lateral palmar nerve at the level of the accessory carpal bone. There is proximal spread of the contrast medium in a Y-shaped pattern in the distal third of the antebrachium, around the median nerve and the caudal branch of the ulnar nerve. Such spread of local anaesthetic solution could result in densensitisation of the palmar aspect of the carpus.

of the entire palmar carpal region and caudodistal aspect of the radius.

It is clear that no method of anesthesia of the proximal metacarpus is either entirely specific or reliable. It is therefore important to recognize the limitations of each technique, including the risks of synovial infection. Interpretation is further complicated by the clinical recognition that carpal and proximal metacarpal lesions may coexist.[21] Moreover, although proximal diffusion from a subcarpal injection site was not seen in the contrast-medium study, palmar and palmar metacarpal nerve blocks have abolished pain associated with a parasagittal fracture of the third carpal bone and rupture of the medial palmar intercarpal ligament[d] or lesions at the origin of the ALDDFT on the palmar aspect of the third carpal bone.[22]

The medial approach to the lateral palmar nerve is potentially perhaps the least specific technique for analgesia of the proximal palmar aspect of the metacarpus. Some difficult horses are easier to inject with the limb on the ground; a lateral approach to the lateral palmar nerve may be preferable in such a horse. This approach also is unlikely to interfere with subsequent ultrasonographic examination, whereas with the subcarpal techniques there is a risk of creating air artifacts. Other horses are more easily restrained with the limb to be injected maximally flexed; in such horses either approach to the palmar metacarpal nerves may be the preferred technique. Proficiency in all techniques and adaptability are desirable.

It is a common clinical observation that lameness associated with proximal suspensory desmopathy (PSD) in hindlimbs may be improved by intra-articular analgesia of the tarsometatarsal (TMT) joint, particularly if assessment of the block is delayed >10 minutes or if >3 mL of local anesthetic solution is injected. There is close proximity between the distal plantar outpunching's of the joint capsule and the suspensory ligament.[23] If >3 mL of mepivacaine is injected into the TMT joint, increased pressure within the joint is likely to encourage plantar leakage through the needle tract. Injection of radiodense contrast medium into the TMT joint resulted in spread to the centrodistal joint in 35% of limbs[23] and frequent diffusion (90% of limbs) around the insertions of tibias cranialis and fibularis tertius, as far proximally as the tarsocrural joint. There is therefore the potential for mepivacaine to influence the dorsal metatarsal nerves. There was also diffusion into the tarsal sheath, with traces present in 35% of limbs by 15 minutes after injection of the TMT joint. In one limb, contrast medium was distributed in the tarsocrural and talocalcaneal-centroquartal joints and not in the TMT joint. It is conceivable that the needle was inadvertently positioned proximal to the fourth tarsal bone.

After subtarsal injection of contrast medium around the plantar metatarsal nerves with/without the plantar nerves, proximal distribution of contrast medium within the tarsal sheath was seen in 40% of limbs; no proximal diffusion was identified outside the tarsal sheath. Contrast medium was only seen in one TMT joint, suggesting that the likelihood of subtarsal analgesia influencing the tarsus is relatively small. However, even with the use of the potentially more specific technique of perineural analgesia of the deep branch of the lateral plantar nerve (3 mL mepivacaine evaluated 10 minutes after injection), pain associated with the central tarsal (CT) bone has been substantially improved in two horses, one with abnormal mineralization of the CT bone and one with a fracture (Fig. 9),[24] highlighting the lack of specificity of diagnostic analgesia and confirming previous observations.[25]

We have previously documented false-negative responses to intra-articular analgesia of the tarsocrural joint associated with lesions in the subchondral bone[26] and false-negative responses to intra-articular analgesia of the CD and TMT joints in associated with advanced osteoarthritis (OA).[27] Perineural analgesia of the tibial and fibular nerves should abolish pain from the tarsus. However, local diffusion from the tibial injection site may result in resolution of lameness associated with gastrocnemius tendonitis.[28] Proximal diffusion may result in im-



Fig. 9. Dorsomedial-plantarolateral radiographic image of the tarsus and proximal metatarsus of a horse with grade 5/8 left hindlimb lameness, abolished within 10 minutes of perineural analgesia of the deep branch of the lateral plantar nerve. There is a complete, articular, non-displaced fracture of the central tarsal bone (arrow).

provement in lameness caused by a mid-diaphyseal stress fracture of the tibia.[e,f]

This limited overview highlights the limitations of a variety of local analgesic techniques but is not meant in any way to detract from the value and importance of diagnostic analgesia. Knowledge of the limitations of each technique helps in the interpretation of the response. Interpretation is further compounded by improvement in lameness rather than abolition of lameness and determining whether the degree of improvement is sufficient to be explained by a single source of pain or whether more proximal blocks are required to identify additional source(s) of pain contributing to lameness. There have been many publications describing specific lameness conditions in which a 50% improvement after diagnostic analgesia has been considered sufficient. In my experience, 50% improvement in the baseline lameness after perineural analgesia suggests that it is highly likely that there may be a more proximal source of pain contributing to lameness and additional nerve blocks are required. Solving lameness is like a jigsaw puzzle; all of the pieces of information must fit together correctly. This is the art and science of lameness diagnosis.

4. MRI and Lameness Diagnosis: What Have We Learned?

Magnetic resonance imaging has greatly enhanced our ability to make more specific diagnoses, most particularly in the foot, but also in the pastern, fetlock, metacarpus and metatarsus, carpus and tarsus, and stifle. However, as with any imaging modality, acquisition of high-quality images and accurate interpretation are of paramount importance. Selection of the correct region to examine is crucial; the previous discussion about the lack of specificity of local analgesic techniques highlights this. If lameness is abolished by palmar (plantar) nerve blocks performed at the base of the proximal sesamoid bones, it may be necessary to examine not only the foot and pastern but also the fetlock.

The use of MRI has also highlighted our lack of understanding about what may cause pain. A horse with unilateral lameness may show similar abnormalities of the navicular bone and deep digital flexor tendon bilaterally. Why did the horse not show bilateral lameness? Is the non-lame limb at an earlier subclinical phase? How likely is it that the horse will become lame on this limb? If a combination of structures is abnormal, which is most likely to be the current major source of pain? Does the presence of increased radiopharmaceutical uptake make it more likely that a structure is a source of pain? These and many other questions remain currently unanswered. The following discussion reviews some of our advances in knowledge related to a variety of conditions of the foot, including pertinent clinical features. It is crucial to recognize that not all horses require MRI. Careful clinical appraisal and acquisition of high-quality radiographs and ultrasonographic images, correctly interpreted, can often lead to an accurate diagnosis. Since the advent of MRI, I believe that we are in a better position to understand the clinical significance of some subtle radiological abnormalities.

Collateral Ligaments of the DIP Joint

Biomechanical Function

The collateral ligaments (CLs) of the DIP joint originate from depressions on the distal medial and lateral aspects of the middle phalanx and insert both in small depressions on the dorsomedial and dorsolateral aspects of the distal phalanx, close to the joint margins, and to the dorsal aspect of the medial and lateral ungular cartilages. Their function is to support the DIP joint in its movements in sagittal, frontal, and transverse planes.[29] Asymmetric foot placement, with the quarters at different heights, results in lateral or medial rotation and sliding of the distal phalanx relative to the middle phalanx (frontal movement). It also results in the middle phalanx rotating and the elevated side of the middle phalanx moving in a palmar direction (transverse movement). These movements are passive but place particular stress on the CLs of the DIP joint. Landing on the lateral aspect of the foot first, a consistent feature in Warmblood horses (although not necessarily detectable by gross observation),[30] may result in asymmetric forces in the CLs of the DIP joint. A recent in vitro study with the use of three-dimensional bone and ligament reconstruction

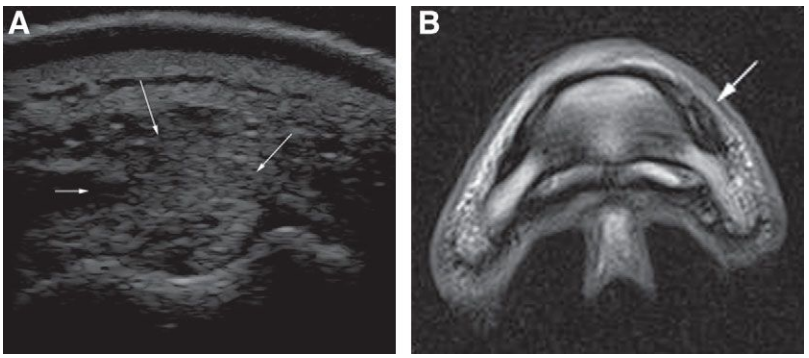


Fig. 10. A, Transverse ultrasonographic image of the medial collateral ligament (CL) of the distal interphalangeal (DIP) joint. The CL is enlarged, with poorly demarcated margins and heterogeneous echogenicity, consistent with desmitis. B, Transverse fast spin echo low-field magnetic resonance image of the same limb as Fig. 10A. Medial is to the right. The medial CL of the DIP is enlarged close to its insertion on the distal phalanx and has increased signal intensity. The lesion extended the entire length of the ligament and also had increased signal intensity in fat suppressed images.

demonstrated that strain in the CLs of the DIP joint was increased on sharp turns compared with straight lines; the highest strain increase was in the medial CL.[31] The medial CL is placed under particular strain by the outward rotation of the DIP joint created by the rapid deceleration of the lateral side of the foot as it lands first.[32]

CL Injury

CL injury has been recognized as an important cause of foot pain either alone[33–39] (Fig. 10) or in association with other osseous or soft tissue injuries.[33,39,40] There are frequently no localizing clinical signs, although occasionally there is mild swelling just proximal to the coronary band and/or heat, which may only be detectable after clipping preparatory to ultrasonography. To date, no conformational abnormalities of the digit have been associated with CL injury. Horses may be uncomfortable standing on a wedge that tilts the foot mediolaterally or standing on a board that elevates the toe. Lameness may be detectable at walk but be much less apparent at trot in straight lines. There is a longer stance phase at the walk than at the trot, so there is greater extension of the DIP joint and thus more stress on the CLs of the DIP joint. In a histological study, lesions at the insertion were worse toward the palmar aspect of the CL.[41] This concurs with both scintigraphic observations that increased radiopharmaceutical uptake (IRU) often extends palmar to the insertion site[33,34,42,43] and radiological observations that osseous cyst-like lesions (OCLLs) tend to occur at the palmar aspect of the insertion site.[44,45] Extension of the DIP joint results in increased tension in the palmar aspect of these ligaments. Characteristically lameness is often two grades or higher more severe at trot in circles compared with straight lines, especially on a firm surface when the foot does not sink into the ground.

Response to Diagnostic Analgesia

In 114 horses with primary CL desmopathy of the DIP joint and no other cause of lameness, 35.6% were sound after perineural analgesia of the palmar digital nerves; 44.5% improved ≥50%; in 30% there was no change or <50% improvement.[46] Lameness was abolished in all horses by palmar (at the base of the proximal sesamoid bones) nerve blocks. Sixty-eight percent of horses did not respond to intra-articular analgesia of the DIP joint within 5 minutes of injection; 30% improved ≥50%, and 2% were sound. Lesions at the distal aspect of the CLs identified histologically occurred principally adjacent to the synovium of the DIP joint.[41] This may have implications for pain associated with injury and the potential response to intra-articular analgesia. Local anesthetic solution may desensitize the superficial nerves in the synovium within seconds after intra-articular injection.[47] It was presumed that the horses that did respond to intra-articular analgesia may have secondary synovitis or OA caused by mild joint instability, the result of CL injury. However, it is possible that pain associated with a focal lesion on the axial, synovial side of the CL insertion could be alleviated, especially if the assessment of the response to intra-articular analgesia was delayed. We have observed abnormal accumulation of synovial fluid axial to an abnormal CL using MRI, and, in association with an OCLL there may in some horses be communication between the lesion and the joint.[44,45] None of 65 horses with primary CL injury of the DIP joint showed improvement in lameness after intrathecal analgesia of the navicular bursa. A uniaxial palmar block usually had no effect in horses with uniaxial lesions, perhaps because both CLs were painful but only one had structural change.

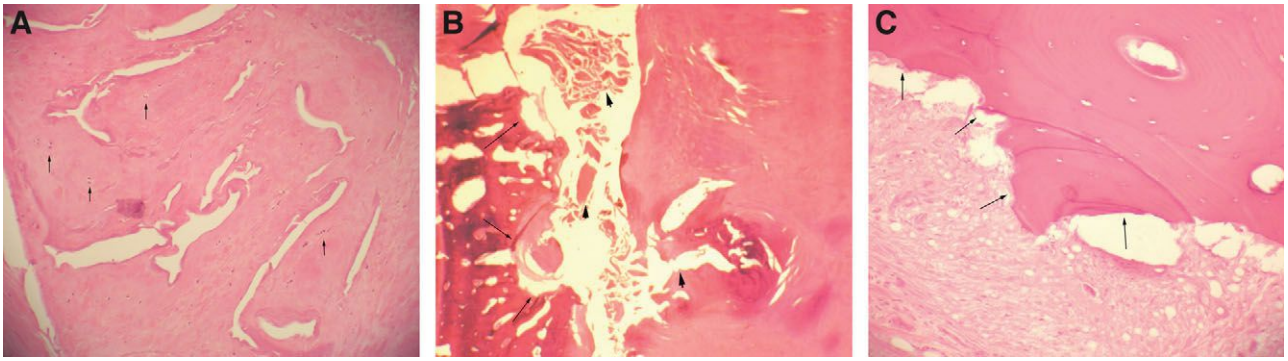


Fig. 11. A, Sagittal section through the distal aspect of a collateral ligament of the distal interphalangeal (DIP) joint with generalised chondroid metaplasia and widespread fissure development. There are chondrocyte clusters (black arrows). The fissures (white spaces) have smooth margins, whereas processing artefacts are usually irregular. There are areas of hyalinisation representing alteration to an amorphous-type acellular structure (black arrow heads). Pale areas (e.g., surrounding the chondrocyte clusters) represent proteoglycan deposition. (Haematoxylin & Eosin, × 40). B, Sagittal section through the distal aspect of a collateral ligament of the distal interphalangeal joint. The ligament is hyalinised with no normal ligamentous architecture. There is chondroid metaplasia. There is a naturally occurring cleft between the ligament and the distal phalanx within which are clumps of fissuring degenerate ligamentous tissue (arrow heads). The bone margin (arrows) is irregular reflecting resorption. (Haematoxylin & Eosin, × 10). C, Sagittal section through the solar aspect of a distal phalanx. The solar aspect faces distally (arrows) and has an irregular margin. Proximally there is normal lamellar cortical bone. There is an abrupt and irregular transition from bone to abnormal fibrovascular tissue containing osteoclasts. This section was obtained directly distal to the site of insertion of the injured ipsilateral collateral ligament of the DIP joint. It is proposed that injury may result in altered stability of the DIP joint, predisposing to abnormal loading of the ipsilateral aspect of the distal phalanx, and secondary osseous changes. (Haematoxylin & Eosin, × 40).

Diagnostic Imaging

Lesions may be identified ultrasonographically, but only a limited length of the ligament can be assessed. Ultrasonographic assessment revealed lesions consistent with desmitis characterized by enlargement of the ligament, and areas of reduced echogenicity were identified in 85 of 313 horses (27%), with lesions confirmed with the use of MRI.[39] On MRI, CL injury was defined as increased signal intensity in T1 and T2*-weighted (W) gradient echo (GRE) or fast spin echo (FSE) images in part or all of the ligament, with enlargement or change in shape, or altered definition of the margins, with or without alteration in signal intensity in the perligamentar tissues. Some horses also had increased signal intensity in fat-suppressed images.[33–38] In all studies, medial lesions were identified more frequently than lateral injuries; lesions were sometimes biaxial. Interpretation of MR images is potentially confounded by the magic angle effect (MAE) in images acquired in both low-field[48] and high-field magnets[49] related to the orientation of the fibers, especially at the origin; the lateral CL is especially susceptible in images acquired standing because of its more sloping orientation. Use of sequences with long echo times is useful but does not abolish the MAE. Concurrent osseous pathology is discussed below.

Verification of MRI by Comparison With Histology

To verify the interpretation of MR images, a comparative high-field MRI and histological study was performed in horses both with and without suspected CL injury.[41] It was hypothesized that abnormal signal intensity and tissue contour would represent change in tissue structure detected through histological examination. The aims were to compare results in horses free from lameness and those with chronic lameness and to describe possible progression of lesions. One or both feet from 12 horses free from lameness (Group N) and 25 horses with foot-related lameness (Group L) were examined with the use of MRI and by gross postmortem examination. The MR images were graded (0–3) by one analyst. Sagittal histological sections from the proximal and distal aspect of each CL were examined histologically and were assigned a histological grade: 0 = normal or the presence of transitional fibrocartilaginous metaplasia, considered to be within normal limits, near the origin and/or insertion; 1 = localized pallor; 2 = diffuse, extensive, or multifocal fibrocartilaginous metaplasia; 3 = extensive fissuring degeneration and any associated osseous pathology (Fig. 11A,B). The overall score assigned to each CL was the sum of the proximal and distal grades. Scintigraphic images from lame horses were also evaluated.

In horses from Group N, 25 CLs were graded normal on both MR images and histology. The majority of CLs had a histology score of 0. Two CLs were graded 1 on MR images but were histologically normal. Two CLs had MR abnormalities verified histologically. However, two CLs appeared normal on MR images but were histologically abnormal.

In Group L, in 72 of 89 CLs the results of MRI and histopathology concurred. Eighteen CLs were

deemed normal on both MR images and histology. Fifty-four CLs had MR abnormalities verified histologically. Of the 13 CLs from 10 horses that were graded as normal with the use of MRI but were abnormal histologically, the overall histology score ranged from 1 to 5 of 6 (mean, 3.33). Histological abnormalities of the CLs of the DIP joint were identified in 37 medial CLs and 30 lateral CLs. When the score was compared between medial and lateral, there was a significantly more severe histological score in the medial than lateral ligaments (P = 0.007). The grade of lesion was greater distally than proximally in 44 CLs, of equal grade in 16 CLs, and was less distally than proximally in seven CLs, with similar proximodistal distribution of lesions medially and laterally.

The distal insertion of the CLs was the region in which the most severe degenerative abnormalities were seen histologically, especially axially (ie, adjacent to the synovium of the DIP joint) and toward the palmar aspect. Early changes were characterized by linear areas of pallor and hyalinization of the collagen fibers, followed by transformation of fibroblasts into chondrocytes and the development of diffuse areas or focally dense areas of fibrocartilaginous metaplasia (Figs. 11A,B). More severe lesions showed a very typical pattern characterized by tortuous, intercommunicating fissures, with smooth edges within the degenerate, hyalinized collagen, containing chondrocytes and chondrones. The pattern was very distinct from ordinary splitting artifacts as the result of histological processing. Several horses showed evidence of lesion-associated blood vessel occlusion and attempted revascularization. Naturally occurring clefts were seen at the bone-ligament interface in association with severe ligament degeneration. Osseous changes were seen immediately distal and palmar to the insertion of the CL on the distal phalanx, with early changes characterized by bone loss on the abaxial cortical surface of the distal phalanx and replacement by fibrous tissue. Other horses had mild interstitial edema in the marrow fat. Lesions progressed to the formation of bone spaces and ultimately an OCLL.

In the majority of horses, histological lesions in both the medial and lateral CLs were similar (23.9%) or worse (65.7%), distally compared with proximally. We have previously suggested that the insertion site is a major stress point, irrespective of the site of injury within the ligament.[43] We have also documented a higher prevalence of osseous lesions at the insertion of the CLs on the distal phalanx, compared with at their origin on the middle phalanx.[39]

In several horses in which a block of the distal phalanx was examined, there was an irregular, spiculated appearance of the cortex of the solar aspect of the distal phalanx in the same frontal plane as the insertion of the ipsilateral CL of the DIP joint (Fig. 11C). This was due to bone loss and infilling by fibrovascular tissue, and in some sections there were scattered osteoclasts.

Nuclear scintigraphy was performed in 21 horses, and IRU was identified at the insertion of a CL in one (n = 7) or both (n = 2) limbs of nine horses, medially in 10 limbs, and laterally in one. All of these 11 limbs had associated MRI abnormalities of the CLs, although only nine had abnormalities verified histologically. There were no sections available of the bone ligament interface in the two limbs in which no histological abnormality was detected. There was no relationship between the presence of IRU and the presence of osseous abnormalities detected through the use of MRI. However, there was an association between IRU and a higher histological score.

The lesions that were identified in abnormal CLs were principally degenerative in nature and similar to those seen in human posterior tendon insufficiency,[50] with little evidence of inflammatory reaction. There was some evidence of attempts to create repair tissue, which was presumably inadequate to withstand load. Repair is probably compromised by lack of blood supply with some evidence of blood vessel occlusion and attempts at revascularization, as we have also seen in both the DDFT[51] and the proximal aspect of the suspensory ligament in hindlimbs.[52]

It was concluded that high-field MRI is reasonably reliable for detection of lesions of the CLs of the DIP joint but may underestimate their prevalence. Chronic CL injury appears to be a primary degenerative process, which may explain the poor response to conservative treatment and a need for promotion of regeneration.

Concurrent Osseous Pathology

The relationship between CL injury and osseous abnormalities was investigated[39] (Fig. 12). Focal or diffuse areas of IRU in the distal phalanx at the insertion of the CL had previously been identified scintigraphically,[33,34,43] and it had been suggested that this site is susceptible to significant biomechanical loading, irrespective of the site of injury within the ligament.[43] It was hypothesized that there would be (1) a higher incidence of osseous abnormality at the insertion of an injured CL than at the origin; and (2) a relationship between the presence of osseous abnormality and duration of lameness.

Magnetic resonance images of 313 feet of 289 horses with foot pain and a definitive diagnosis of collateral desmopathy of the DIP joint were retrospectively analyzed for presence and type of osseous abnormality in the middle and distal phalanges.[39] Scintigraphic images were examined and the presence of IRU in the middle or distal phalanges was recorded. Osseous abnormalities were detected in 45.7% of feet, 18.8% of which had osseous and CL injury alone; the remainder had CL-related osseous injury and multiple injuries within the hoof capsule.

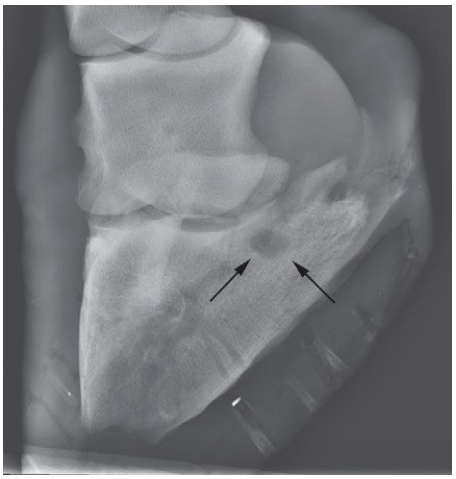


Fig. 12. Dorsomedial-palmarolateral flexed oblique radiographic image of a foot. There is a well-defined osseous cyst-like lesion in the distal phalanx (arrows) immediately palmar to the insertion of the medial collateral ligament (CL) of the distal interphalangeal (DIP) joint. There is also mild modelling at the origin of the lateral CL of the DIP joint on the middle phalanx.

Endosteal reaction and entheseous new bone were the most common types of osseous pathology associated with the ligament origin, accounting for 8.3% (n = 26) and 7.3% (n = 23) of feet, respectively. The most frequent types of insertional osseous pathology were entheseous new bone (36.4%, n = 114) and focal increased signal intensity on short tau inversion recovery (STIR) images (11.5%, n = 36). Osseous cyst-like lesions occurred in only 13 feet (4.1%), of which only two were associated with the ligament origin. Osseous cyst-like lesions in the distal phalanx were all situated distal and palmar to the site of CL insertion. Diffuse increased signal intensity on STIR images was the most frequent type of osseous injury located within the distal phalanx, occurring in 7.3% (n = 23) of feet. Twenty-eight feet (8.9%) had diffuse increased signal intensity on STIR images and concurrent reduced signal intensity on T1-weighted images in either the middle or distal phalanges.

Some degree of mineralization of a palmar process ipsilateral to CL injury, characterized by reduced signal intensity in both T1W and T2*W GRE images, occurred in 17.3% of feet (n = 54). Osseous abnormalities of the DIP joint were identified in 12 feet (3.8%), characterized by increased signal intensity in fat-suppressed images in the subchondral bone and/or in the trabecular bone distal to the subchondral bone plate, with/without localized increased thickness of the subchondral bone. These lesions probably reflect instability of the joint or acute overload of the subchondral bone. Subluxation of the DIP joint was evident in only four feet, all with severe CL injury. A fragment associated with the ligament origin or insertion was the least common type of osseous pathology seen, occurring in only two feet.

Moderate or intense focal IRU was seen either at the site of insertion of the CL (n = 80, 15%) or at the insertion and extending into the ipsilateral palmar process (n = 20, 4%). Increased RU associated with osseous abnormality related to CL injury of the DIP joint was more frequently observed in the distal phalanx (n = 123, 97%) than the middle phalanx (n = 4, 3%), coexistent with the higher incidence of insertional osseous pathology. Focal intense IRU in one or more palmar process was observed in 14% of feet. There was a higher incidence of osseous abnormalities medially than laterally and at the ligament insertion than at the origin. There was a significant association between presence of IRU and osseous injury (P = 0.001), but normal RU did not preclude significant osseous pathology associated with CL injury.

Increased RU was often situated just palmar to the site of ligament insertion most obvious on solar scintigraphic images, in accordance with other studies.[42,43] We previously reported a positive association between the presence of IRU at the site of insertion of a CL on the distal phalanx with severity of injury relative to histological grade of CL pathology.[41] A small proportion (4%) of feet in the current study had IRU at the site of the ligament insertion extending into the ipsilateral palmar process, which was often associated with abnormal mineralization identified by MRI, characterized by decreased signal intensity in T1W and T2*W GRE images, with or without increased signal intensity in fat-suppressed images. Thus, abnormalities at the CL insertion may be associated with injury to adjacent structures. Focal IRU in a palmar process, seen in 14% of feet, has been previously documented in association with a variety of other lesions detected with the use of MRI, causing lameness or as an incidental finding in the non-lame limb of a bilaterally lame horse.[53] In the previous study, mild to intense IRU was seen in the medial palmar process of 5.6% of 512 feet and 2.4% of lateral palmar processes. The higher frequency of IRU in a palmar process of the distal phalanx in the current study association with CL injury suggests that CL injury may increase the risk of trauma to the ipsilateral palmar process. Nuclear scintigraphic examination may therefore be useful for the detection of osseous pathology associated with CL injury, although a negative result does not preclude the presence of osseous injury.

There was a significant negative association between the presence of all CL-related osseous injury and duration of lameness (P = 0.036). It is well recognized that radiological abnormalities may predate the onset of pain and lameness associated with OA, and it is likely that the same is true for lesions detected with MRI. It is possible, however, that the presence of an osseous abnormality such as entheseous/endosteal reaction associated with the

ligament origin or insertion may afford some stabilization of the injured ligament, which may account for the negative association between the duration of lameness and presence of an osseous abnormality.

Relationship Between Ossification of the Ungular Cartilages and Injuries of the CLs of the DIP Joint

To determine the relationship between ossification of the ungular cartilages and CL injury, dorsopalmar (dorsoplantar) radiographs of one foot from each of 462 horses were examined and ossification of the cartilages of the foot graded with the use of a modification of a previously published scale.[54] The presence or absence of CL injury was recorded. There was left-right symmetry of ossification between feet and significant association between grades of each foot, with lateral greater than or equal to medial cartilages. Possibly significant ossification (PSO) (grade ≥3 + separate center of ossification [SCO]) occurred in the maximally ossified cartilage in 59 (12.8%) feet. There was a significantly higher frequency of PSO in cobs and cross-breeds compared with all other breeds (P = 0.0002). There was a significantly higher frequency of PSO of the maximally ossified cartilage in horses with CL desmopathy compared with horses without CL injury (P = 0.0179). There was a significant association between PSO of the maximally ossified cartilage and injury of the distal phalanx (P = 0.0391). There was no association between distal phalanx injury and marked asymmetry of the ossified cartilages of the foot.

The left-right symmetry and association between lateral and medial cartilages were similar to those reported previously.[55–58] The higher proportion of PSO in cobs compared with other breeds was in accordance with a previous study[55] and may be genetically determined. This could also be associated with the greater body weight to height ratios typical of this type of horse,[56,58,59] or the greater component of vertical movement in the stride of these horses,[59] compared with horses with a more extended gait, creating a greater amount of vertical force per unit area, with less damping through soft tissues and the distal limb joints.

It has been hypothesized that the force of impact with the ground compresses the digital cushion,[60] forcing the cartilages of the foot abaxially at their connection with the distal phalanx. It was also hypothesized that the force is transferred, and hence dissipated, through the venovenous anastomoses running through the cartilages.[60] Venovenous anastomoses are more common at the base of the cartilage,[60] and most ossification starts here. The base of the cartilages of the foot had greater RU uptake than more proximally,[61] indicating that it is a site of continued modeling throughout life, reflecting the focal concentration of force. Extensive ossification reduces the flexibility and capacity for energy dissipation of the cartilages, thus transferring a greater proportion of the force through other structures of the foot, possibly causing adaptive changes or injury in adjacent tissues.[56] The predilection for fractures of ossified cartilages at their base may be explained by maximum stress/or strain concentration at this point.[62]

There is a close anatomical relationship between the ungular cartilages and the CLs of the DIP joint, which is likely to be important in the association between PSO and CL injury. The axial surface of an extensively ossified or fractured cartilage may abrade the abaxial surface of the CLs during flexion and extension of the DIP joint.[29] The cartilages of the foot are connected to surrounding structures, such as the digital cushion, proximal, middle, and distal phalanges and navicular bone by small ligaments. Increased tension in these ligaments has been suggested to be partially responsible for the ossification process.[56,63] The chondrocoronal ligament runs from the axial aspect of each cartilage to the middle phalanx, and the chondroungular ligament connects the distal aspect of the cartilage with the ipsilateral palmar process of the distal phalanx.[56] These ligamentous connections may be responsible for energy transmission, which results in injury of the distal phalanx in association with extensive ossification. The chondroungular ligament in particular may be implicated because if ossification extends in a palmar (plantar) direction, the ligament may become completely enclosed within ossified tissue. In some horses, the dorsal aspect of the cartilage fuses with the ipsilateral CL,[56] allowing direct transmission of force from the cartilage to the CL, potentially resulting in injury. It has previously been suggested that forces mediated by ligamentous attachments to the cartilages may be transmitted differently through a rigid osseous structure compared with an unossified cartilage, possibly resulting in increased stress, modeling, and risk for bone trauma or fracture at the base of the extensively ossified cartilage.[43]

Response to Treatment of Horses With Injury of a CL of the DIP Joint

Magnetic resonance images from 313 feet of 289 horses with foot pain and a definitive diagnosis of collateral desmopathy of the DIP joint were retrospectively analyzed for presence of osseous abnormality associated with the ligament origin or insertion and the middle and distal phalanges.[64] Horses were assigned to groups according to the combination of their injuries. Type of treatment (box rest and controlled walking exercise; intra-articular medication of the DIP joint; corrective trimming to restore appropriate foot balance and shoeing according to foot conformation and to facilitate breakover; extracorporeal shockwave therapy [ECSWT] or radial pressure wave therapy [RPWT]) was recorded and follow-up information was ob-

tained. Thirty-two horses with additional sources of lameness were excluded from analysis of outcome. Follow-up data were available for 182 horses, 55 of which had follow-up information for up to 2 years after presentation. Forty-four percent of horses (n = 41) with CL injury alone and 43.2% of horses with CL related osseous injury (n = 55) returned to their previous athletic function. Prognosis for a combination of injuries to multiple soft tissue and osseous structures within the hoof capsule was substantially worse, with only 14.6% of 71 horses returning to their original level of work. There was no effect of ECSWT or RPWT on outcome, but this may reflect the low power of the study. The presence of mild to moderate CL-related osseous injury did not appear to influence prognosis compared with CL injury alone; however, horses with OCLL in the distal phalanx at the insertion of CL had a poor outcome. Paradoxically, there was association between an excellent outcome at 12 months and the presence of entheseous new bone at the ligament insertion (odds ratio = 6.8, 95% confidence interval = 1.7–27.1, P = 0.05), suggestive that horses with this type of osseous abnormality were more likely to become sound.

If the results of horses with increased signal intensity in fat-suppressed images in the injured CL were considered (n = 41), 51% returned to full athletic function for a minimum of 6 months after return to full function. These results are comparable with a previous study in which 12 of 20 horses (60%) with collateral desmopathy returned to previous level of athletic function with a minimum follow-up of 9 months after injury.[38] In the current study,[64] four horses that were unresponsive to conservative management underwent bilateral palmar digital neurectomy and returned to their previous function, remaining sound for a minimum of 12 months and up to 4 years. On the basis of these small numbers, it appears that palmar digital neurectomy is a safe procedure to perform with primary CL injury. Our previous correlative MRI, scintigraphic, and histopathological study demonstrated that IRU at the insertion of an injured CL of the DIP joint was associated with a more severe histological score of the injured CL; however, there was no relationship between the presence of IRU and osseous abnormalities detected with the use of MRI.[41] In the current study, there was no association between the presence of IRU at the insertion of a CL of the DIP joint and outcome at 6 or 12 months. The lack of significant association may reflect the low power of the study or that the presence of IRU does not relate to prognosis.

Follow-up data are now required for horses treated by the intralesional injection of mesenchymal progenitor (stem) cells and intra-articular medication with interleukin 1 receptor antagonist protein.

Other Injuries Associated With Ossification of the Ungular Cartilages (Cartilages of the Foot)

Functional Anatomy

The ungular cartilages provide support to the palmar (plantar) aspect of the foot, dissipate forces of the foot's impact with the ground, and are involved in venous return from the digit.[60] Ossification of the ungular cartilages (sidebone) normally starts at the base of the cartilage and extends proximally or can originate from an SCO.[65] Lateral ossification is frequently more extensive than medial,[37,56–58] although heritability of ossification in Finnhorses is similar for both lateral and medial cartilages.[57] Marked mediolateral asymmetry of ossification is unusual[57,58] but has been linked with injuries,[43,61] including fractures of the ossified cartilage,[59] trauma to the base of the ossified cartilage, and injury of the distal phalanx.[43,59,66–68] Ossification reduces the energy dissipating capacity of the cartilages of the foot.

Injuries of the Ungular Cartilages and Related Structures

Ossification of the ungular cartilages has long been recognized and historically was considered of clinical importance especially in draft breeds,[69] but, in more recent years, the clinical significance of injuries of the ungular cartilages has largely gone unrecognized. However, there is a growing body of evidence to suggest that the ossified cartilages can sustain primary injury[59,63,64,66] or be associated with other injuries in the foot, notably the distal phalanx and the CLs of the DIP joint.[37,40,54,59,67,68]

Primary injuries of unossified cartilages with/without related ligaments (chondrocoronal and chondrosesamoidean) of the foot are unusual but have been seen in a small number of horses[68] and have probably been previously overlooked. Diagnosis was dependent on MRI. In three horses with low collapsed heels and a large body size relative to foot size and in one horse with markedly asymmetrical heel bulbs, the affected cartilage(s) were markedly thickened, had irregular contours (especially abaxially), were hypervascular, and had diffuse increased signal intensity in fat-suppressed images, which extended into the ipsilateral aspect of the distal phalanx.

One horse with grade 0 ossification of the medial ungular cartilage had reduced signal intensity at the base of the cartilage and throughout the medial palmar process of the distal phalanx in T1W and T2*W GRE images and increased signal intensity in fat-suppressed images, consistent with mineralization and bone trauma. There was marked enlargement of the medial chondrocoronal ligament with increased signal intensity in fat-suppressed images.

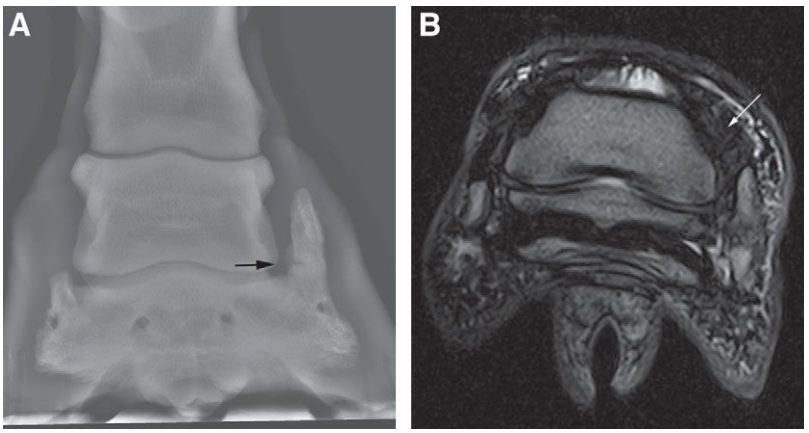


Fig. 13. A, Dorsopalmar radiographic image of a foot. Medial is to the left. There is extensive ossification of the lateral ungular cartilage. There is a transverse incomplete fracture at the base of the ossified cartilage with periosteal callus axially (arrow) and generalised increased opacity across the ossified cartilage. Note the asymmetrical shape of the pastern and hoof capsule; there is more pastern and foot lateral to a line bisecting the proximal and middle phalanges, compared with medially. B, Transverse fast spin echo low-field magnetic resonance image of the same foot as Fig. 13A. Lateral is to the right. The lateral chondrocoronal ligament is enlarged and has increased signal intensity (arrow). Note also that the lateral ungular cartilage is thicker than medially.

Mineralization of Ossified Ungular Cartilages, Trauma at the Junction Between Separate Centers of Ossification, and Trauma or Fracture at the Base of an Ossified Ungular Cartilage

Injury of the junction between SCsO, fractures of an ossified cartilage, usually at the base, and more generalized trauma of an ossified cartilage have been described as primary causes of lameness seen in association with an ossification grade of ≥3.[62,66] These are relatively uncommon causes of lameness; trauma or fracture of an ossified cartilage was identified as primary injury in 24 of approximately 4500 horses (0.53%) undergoing lameness investigation over 9 years (2001–2009).[66] The diagnosis was based on radiological and scintigraphic findings in 12 horses; the other 12 horses also underwent MRI. Fifteen of 32 horses (46.9%) with collateral desmopathy of the DIP joint in association with grade ≥3 ossification of the cartilages of the foot also had evidence of trauma of one or both ossified cartilages, characterized by focal or diffuse regions of increased signal intensity in fat-suppressed images. Seven of 22 horses (31.8%) with other causes of foot pain determined using MRI in association with grade ≥3 ossification of one or both cartilages of the foot also had evidence of trauma of an ossified cartilage of the foot.[66] Horses with at least one ossified cartilage grade ≥4 predominated.

A clear radiolucent line at the base of an ossified cartilage surrounded by osseous modeling was indicative of a fracture (Fig. 13A). Diagnosis was substantiated by nuclear scintigraphy demonstrating focal intense IRU correlating anatomically with the fracture site. It was not always possible to differentiate between a fracture or trauma to the junction between SCsO. Moreover, not all fractures were detected radiologically and MRI was required, although at the base of the ossified cartilage clear differentiation between trauma to the junction between SCsO and a fracture was again not always possible. Modeling at the base of an ossified cartilage combined with focal IRU, supported by increased signal intensity in fat-suppressed images, was defined as trauma to the base of an ossified cartilage. Osseous modeling around the junction between SCsO in the mid shaft of an ossified cartilage detected radiologically, associated with IRU reflected probable instability or trauma. Trauma was supported by MRI findings, including increased signal intensity in fat-suppressed images at the opposing ends of the SCsO. Other radiological findings included mediolateral thickening of an ossified cartilage, irregular cortical contours, heterogeneous radiopacity, or focal or diffuse areas of increased opacity especially distally, and ill-defined transverse radiolucent lines, especially at the base of an ossified cartilage. Careful evaluation of all radiographic projections was crucial because some fractures were only evident in one projection. Other MRI findings included focal or diffuse areas of increased signal intensity in the injured ossified cartilage in fat-suppressed images.

In horses examined with the use of MRI, there were focal or diffuse areas of decreased signal intensity in both T1W and T2*W GRE images in the injured ossified cartilage, consistent with mineralization in 50% of limbs with either primary injury of an ossified cartilage (6/12) or collateral desmopathy of the DIP joint (16/32) in association with grade ≥3 ossification of the cartilages of the foot. Whether such mineralization reflects a response to chronic trauma or is a physiological response is not known. It may further stiffen an ossified cartilage and alter force transmission through the ossified cartilage and

possibly increase the risk of injuries of the cartilages themselves or closely related osseous and soft tissue structures. The predilection for fractures of ossified cartilages at their base may be explained by maximum stress/or strain concentration at this point. The junction between SCsO is a potential weak link.

Trauma of the Distal Phalanx Associated With Ossification of the Ungular Cartilages

Alterations in signal intensity in the ipsilateral aspect of the distal phalanx were identified in seven of 12 horses (58.3%) with a primary injury of an ossified ungular cartilage.[66] This was characterized either by diffuse areas of hypointense signal in T1W and T2*W GRE images consistent with mineralization or areas of hyperintense signal in fat-suppressed images, consistent with bone trauma. One horse had an incomplete fracture of the axial aspect of the distal phalanx. Thirteen of 32 horses (40.6%) with collateral desmopathy of the DIP joint in association with grade ≥3 ossification of one or both ungular cartilages also had evidence of abnormal mineralization or bone trauma of the distal phalanx. In both groups, the presence of active bone modeling was generally supported by IRU in the corresponding anatomical location. These osseous changes may be related to altered force distribution associated with ossification of the ungular cartilages.

Injuries of Associated Ligaments

Injuries of the chondrocoronal and/or chondrosesamoidean ligaments were characterized by increased size (Fig. 13B), loss of demarcation of margins and increased signal intensity in fat-suppressed images, or evidence of entheseous reaction at their origin on the ungular cartilages seen as increased signal intensity in fat-suppressed images or irregularities of the axial cortex.[66] Endosteal and periosteal reactions were also seen at the insertion of the chondrocoronal ligaments on the middle phalanx. Such injuries may be seen with or without ossification of the ungular cartilages.

It is suggested that on the basis of our recent advances in knowledge, the identification of extensive ossification of the ungular cartilages either uniaxially or biaxially at a prepurchase examination should be documented and the potential clinical significance discussed with the purchaser. Although extensive ossification may be a normal variant in some breeds, it is not usual in most sports horse breeds and may be a risk factor for future lameness.

Injuries of the DDFT

Functional Anatomy

Within the digit, the DDFT induces axial compression of the articular surfaces of the PIP and DIP joints.[70,71] It has an important role in stabilizing the DIP joint. The anatomical arrangement of the collateral sesamoidean ligaments (CSLs) facilitates compression of the articular surfaces of the navicular bone into those of the middle and distal phalanges.[70] The DDFT has a dorsal fibrocartilaginous pad that supports pressure of the *tuberositas flexoria*, the transverse prominence on the proximopalmar aspect of the middle phalanx.[71]

The relationship of the DDFT to the navicular bone varies with the phase of the stride. During the full weight-bearing stance phase of the stride, the DDFT is only in contact with the distal aspect of the bone, whereas in the propulsion phase the DDFT bends over the middle scutum (the fibrocartilaginous insertion of the straight sesamoidean ligament on the middle phalanx) and comes into full contact with the navicular bone. Tension in the DDFT is maximal, and active muscle contraction and the elasticity in the tendon and in its accessory ligament result in extension of the DIP joint.[71] At the beginning of the swing phase of the stride, the tension in the DDFT contributes passively to induce flexion of the interphalangeal joints. During extension of the DIP joint, which is maximum at the propulsion phase of the stride, pull on the DDFT creates a shear force between the DDFT and the distal sesamoidean impar ligament (DSIL).[70]

Injuries of the DDFT

Several lesion types have been described, including core lesions, focal or diffuse dorsal border lesions and sagittal plane splits (Fig. 14).[72–74] A comparative study of MR images of horses with and without DDFT injuries demonstrated that there was strong left forelimb and right forelimb symmetry in the cross-sectional area (CSA) of the DDFT, and within limbs there was strong medial lateral symmetry.[75] There was a positive correlation between bodyweight and CSA. Cross-sectional area increased in association with core lesions of the DDFT but not in association with other types of lesions.

A second study compared horses with foot pain (n = 34) and age-matched control horses (n = 25).[76] Lesions of the DDFT were graded in severity as absent, mild, moderate, or severe. In the majority of sound horses, lesions of the DDFT were absent (85%) or graded mild (15%), whereas in the lame group moderate (41%) and severe (35%) lesions predominated.

Lesions of the DDFT are common, occurring in 82.6% of limbs of 264 horses with foot-related lameness examined using MRI; however, some of these were isolated sagittal plane splits or minor dorsal irregularities of questionable clinical significance.[77] Lesions occurred most commonly at the level of the CSL (59.4%) and the navicular bone (59.0%). At the level of the proximal phalanx core, lesions predominated (90.3%), whereas at the level of the CSL and navicular bone sagittal plane splits, dorsal abrasions and focal core lesions were most common.

Primary lesions of the DDFT are defined as lesions that are the principle cause of lameness and usually comprise core lesions proximal or distal to

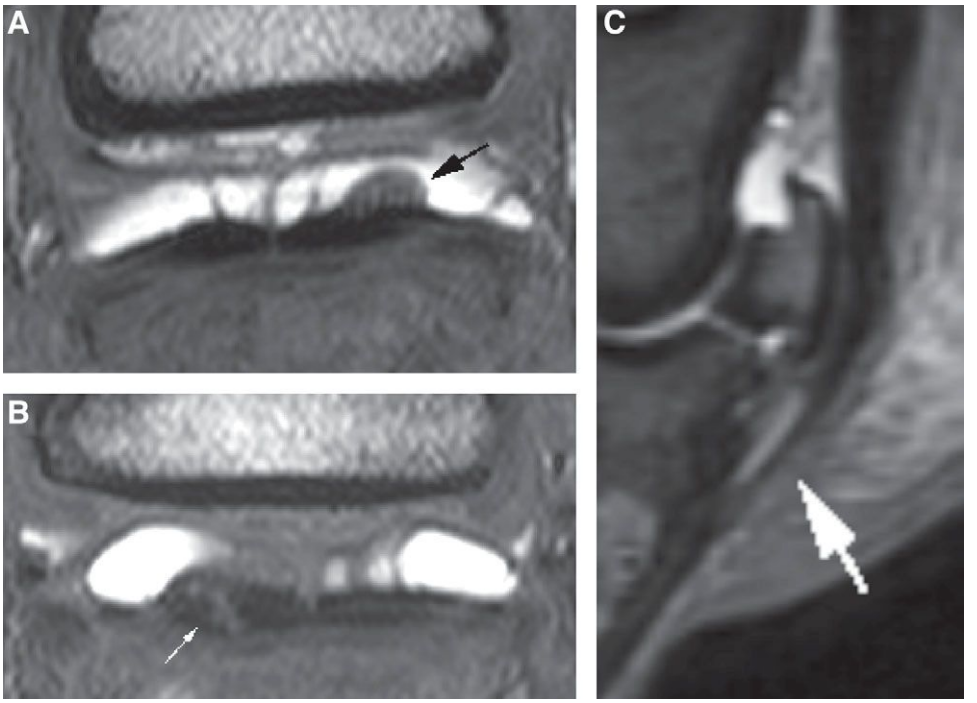


Fig. 14. A, Transverse T2* weighted (W) gradient echo (GRE) high-field magnetic resonance image at the level of the middle phalanx. Dorsal is to the top and lateral to the right. The lateral lobe of the deep digital flexor tendon (DDFT) has an irregular dorsal border with contiguous tissue of intermediate signal intensity protruding into the navicular bursa (arrow), consistent with granulation tissue. The navicular bursa is somewhat distended. B, Transverse T2* W GRE high-field magnetic resonance image at the level of the middle phalanx. Dorsal is to the top and lateral to the right. The medial lobe of the DDFT is enlarged and there is an oblique core lesion of intermediate signal intensity (arrow). This lesion, which extended proximally to the distal aspect of the proximal phalanx, also had increased signal intensity in fat suppressed images. The navicular bursa is distended. There is tissue of intermediate signal intensity on the dorsal aspect of the DDFT axially traversing the bursa. C, Sagittal short tau inversion recovery high-field magnetic resonance image. Dorsal is to the left. There is diffuse increased signal intensity in the distal aspect of the deep digital flexor tendon, which is enlarged.

the navicular bone. Primary lesions of the DDFT often involve principally one lobe and extend a variable distance proximodistally, anywhere from the proximal phalanx to the tendon's insertion on the distal phalanx.[72,73,77] Occasionally lesions extended proximal to the metacarpophalangeal joint, but not all limbs were examined this far proximal. Acute inflammatory or necrotic lesions were seen on T1W and T2*W GRE images and fat-suppressed images, whereas more chronic lesions with fibroplasia may only be seen in T1W and T2*W GRE and FSE images. Lesions identified only in T1W images may be chronic or degenerative. Lesions confined to the insertion were often best identified in fat-suppressed images. Core lesions were often associated with swelling of the affected lobe, resulting in loss of the normal mediolateral symmetry.[75] There was often associated distension of the DFTS and/or the navicular bursa, with or without soft tissue proliferation within the bursa. Severe lesions involving the dorsal aspect of the tendon may be associated with adhesion formation between the DDFT and either the CSL and/or the DSIL. Definitive diagnosis of adhesion formation can be challenging[78] without fluid distension of the navicular bursa.[79] Core lesions of the DDFT have also occurred in conjunction with other soft tissue injuries contributing to lameness.[80,81] These other lesions were often on the ipsilateral side of the foot, suggesting that similar biomechanical forces contributed to injury.

Histopathology of core lesions has revealed no evidence of inflammatory reaction but extensive core necrosis in horses with lameness of less than 6 months' duration[82] (Fig. 15), whereas in horses with more chronic lameness, the core lesions were predominantly fibroplasia and/or fibrocartilaginous metaplasia. Core necrosis was characterized by vacuoles in the fascicles, which tended to coalesce with breakdown material in the vacuoles, and some "floating" chondrocytes and fibroblasts within the vacuoles. There was some evidence of revascularization toward necrotic core lesions, especially those extending into the pastern.

Other DDFT lesion types identified with the use of MRI include dorsal border irregularity and sagittal plane splits. Such lesions occurred with greatest frequency at the level of the CSL and the navicular bone.[77] In some horses, different lesion types are seen at different levels of the tendon. Horses with

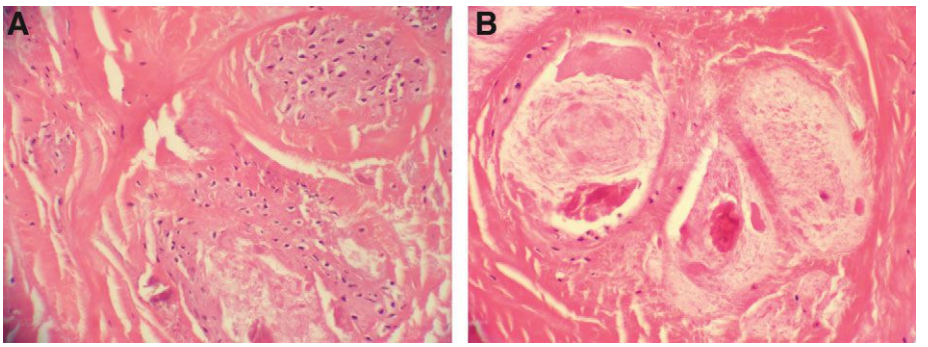


Fig. 15. A, Transverse section of a deep digital flexor tendon showing early core necrosis. Some fascicles undergoing degeneration contain spindle-shaped and rounded cells, resembling fibroblasts and tenochondrocytes, which are believed to have migrated into the fascicle from the surrounding interstitium. At the bottom of the image centrally there is a focal area of collagen dissolution. Haematoxylin and eosin. Magnification × 100. B, Transverse section of a deep digital flexor tendon showing core necrosis. There is a central area with loss of normal collagen structure, basophilia and increased cellularity, comprising numerous fibroblasts and differentiating chondrocytes. The striated appearance of the surrounding tendon tissue is a processing artefact, the result of separation of collagen fibres. Haematoxylin and eosin. Magnification × 100. (Reproduced from *Equine Vet J* 2009;41:25–33, with permission).

lesions of both the navicular bone and the DDFT often had multifocal lesions involving the medial and lateral lobes of the DDFT, especially from the level of the proximal aspect of the navicular bursa distally. Lesions of the navicular bone were often in the same sagittal plane as the DDFT lesions. Defects in the palmar compact bone of the navicular bone often had focal adhesions to the DDFT. Small focal adhesions may be more difficult to identify in low-field images because of the thicker slice thickness compared with high-field images. These DDFT lesions appear to be degenerative with vascular compromise, especially in the septae, and matrix changes, characterized by increased proteoglycan deposition and changes in tenocyte morphology, and fibrocartilaginous metaplasia.[51,82,83] There was no evidence of any acute inflammatory response.

Clinical Features of Primary Injuries of the DDFT

A previous study demonstrated that horses that jump had a higher frequency of occurrence of primary DDFT injuries than horses from other disciplines.[72] A more recent study indicated that elite show jumpers were particularly at risk.[84] Horses 10 to 15 years of age had an increased risk of primary DDFT lesions than horses <six years old (odds ratio, 3.30).[85] A theoretical model indicated that a 1° decrease in the angle of the solar surface of the distal phalanx would result in a 4% increase in force of the DDFT on the navicular bone at the end of stance.[86] However, in a recent study of 300 horses with foot pain, there was no significant association between injury type and angles of the distal phalanx, although there was a trend for the angle of the dorsal aspect of the distal phalanx with the horizontal to be smaller in horses with injuries of the podotrochlear apparatus or the DDFT compared with other groups.[87] In a small radiological study comparing 20 horses with DDFT lesions and 20 control horses, the angle at which the DDFT passed over the palmar aspect of the navicular bone was more acute than in the control horses.[88]

Primary lesions of the DDFT as the principal cause of lameness were seen in 80 horses between January 2001 and March 2005.[89] These horses had closed core lesions or dorsal or less commonly palmar abrasions or long full-thickness splits; horses with short isolated parasagittal plane splits were not included. Horses with increased signal intensity in the palmar third of the navicular bone in fat-suppressed images were included, but horses with other forms of navicular pathology or other lesions potentially contributing to pain and lameness were excluded. Horses presented with either unilateral (n = 48) or bilateral (n = 30) forelimb lameness or unilateral hindlimb lameness (n = 2). Lameness often improved with rest but was exacerbated by work. Lameness varied considerably in degree (3 to 7 of 8; the most frequent lameness score was 4) but was usually worse on a circle on a hard surface. There were usually no localizing clinical signs, although some horses pointed at rest. Palmar digital analgesia rendered 25 horses (31%) sound; improved lameness >50% in a further 33 (41%), but produced no change in 22 (27%). Lameness was abolished in all horses by palmar nerve blocks at the base of the proximal sesamoid bones. Intra-articular analgesia of the DIP joint (5 mL mepivacaine; lameness assessed at 5 minutes after injection) improved or abolished lameness in 41 of 75 horses (55%) but produced little change in 34 (45%). Intrathecal analgesia of the navicular bursa resulted in improvement in lameness in 25 of 37 horses (68%). Intrathecal analgesia of the DFTS improved lameness in three of 12 horses (25%).

Nineteen of 76 horses (25%) had IRU in the region of the DDFT in lateral pool phase scintigraphic im-

ages. Twenty-four horses (32%) had IRU in bone phase images in the region of insertion of the DDFT on the distal phalanx. Horses with lameness of <3 months' duration were more likely to have IRU than horses with a longer duration of lameness. Only 10 of 75 (13.3%) horses had detectable ultrasonographic abnormalities of the DDFT, although many lesions did extend into the pastern when evaluated with the use of MRI.

Treatment and Outcome of DDFT Lesions

Treatment varied according to the site and severity of the lesion(s). Focal lesions restricted to the navicular bursa were surgically debrided. Most other lesions have been treated conservatively by rest and controlled exercise, combined with trimming and shoeing according to the individual's foot conformation, with or without shock wave therapy.

Follow-up information for 76 horses examined before September 2004 was available.[80] Three horses were humanely destroyed for unrelated causes, and two horses were lost to follow-up. Twenty-three of 71 horses (32%) returned to full athletic function for a minimum of 6 months and a maximum of 3 years; eight horses (11%) were sound and in light work. Forty horses (56%) had persistent or recurrent lameness.

In a study of 92 horses that underwent endoscopic evaluation of the navicular bursa, 37 of 89 horses (42%) returned to full function for an unspecified period, after an unspecified period of rest.[90] The results for treatment with biological preparations require critical assessment.

A New Form of Navicular Disease

Since the introduction of MRI, our understanding of navicular disease has greatly increased. It is now known that navicular bone pathology in isolation is comparatively rare and frequently occurs in association with injuries of the DDFT, the CSL, and/or the DSIL.[36,40,80,81,91–95] It is clear that there are a variety of forms of navicular bone pathology[81,94] such as thickening of the palmar compact bone, or degenerative lesions of the palmar compact bone, including deep erosions involving both the fibrocartilage and subchondral bone.[93] There are lesions restricted to the spongiosa characterized by diffuse increased signal intensity in fat-suppressed images in the spongiosa; linear increased signal intensity between the attachments of the CSL and DSIL; or OCLLs in the distal half of the bone. In addition, there are abnormalities restricted to the distal border including marked increase in number and size of synovial invaginations, entheseous new bone, and distal border fragments.[96,97] Potentially reversible lesions consistent with acute trauma of the navicular bone may also occur.

A recent correlative MRI and postmortem study demonstrated that diffuse increased signal intensity in the spongiosa of the navicular bone in fat-suppressed images was associated with alterations of the marrow fat which was either focal or diffuse, with loss of clear cytoplasmic boundaries and increased vascularization of the interstitium, with frequent small capillaries (Fig. 16).[98] There was fibroplasia and enlarged intertrabecular bone spaces. There was accompanying bone loss with thinning of trabeculae and irregularly spiculated borders. Osteoclasts were seen occasionally. In some bones there was multifocal accumulation of pale to acidophilic material in the interstitium of the marrow fat.

It has previously been suggested that degenerative change of the spongiosa is generally only seen dorsal to extensive fibrocartilage damage.[99] However in the Dyson et al study,[98] abnormalities of the spongiosa could be seen in isolation. In a previous study of horses with chronic palmar foot pain (although one inclusion criterion was lameness of >2 months' duration, all horses had been lame for considerably longer), mild or moderate focal or generalized increased signal intensity in fat-suppressed images was associated with trabecular thinning and widened intertrabecular spaces.[76,100] High signal intensity in fat-suppressed images, associated with decreased signal intensity in T1W gradient echo images and mixed signal intensity in T2*W GRE images, was associated with generalized osteonecrosis and fibrosis, with irregular trabeculae, adjacent adipose tissue edema, and prominent capillary infiltration.

A recent postmortem study of feet with advanced radiological abnormalities of the navicular bone demonstrated that increased signal intensity in fat-suppressed images correlated with areas of degenerate adipose tissues, with hemorrhage or replacement by fibrocollagenous material, or fluid-filled cystic spaces.[91] Marrow space fibrosis was also previously reported in horses with advanced navicular disease.[99,101]

In the Dyson et al study,[98] of horses with more recent onset lameness without any radiological abnormalities, we identified fat atrophy, with loss of clear definition of lipocyte cytoplasmic borders, accompanied by an interstitial capillary proliferation, perivascular or interstitial edema, fibroplasia, enlarged spongiosa bone spaces, and thinned bone trabeculae, showing loss of bone with irregularly spiculated edges of moth-eaten appearance (Fig. 16). These changes are similar to those seen in emaciated horses with generalized serous atrophy of bone marrow fat in the medulla of the distal limb bones,[102] although in those horses there was more widespread distribution of edema and accumulations of macrophages. Macrophages were not seen in the current study.

Our recent study[98] was not a longitudinal study, so we do not know how changes in the spongiosa may progress. Increased signal intensity in fat-suppressed images parallel and dorsal to the palmar cortex seen in association with primary DDFT lesions may resolve over time, or occasionally more

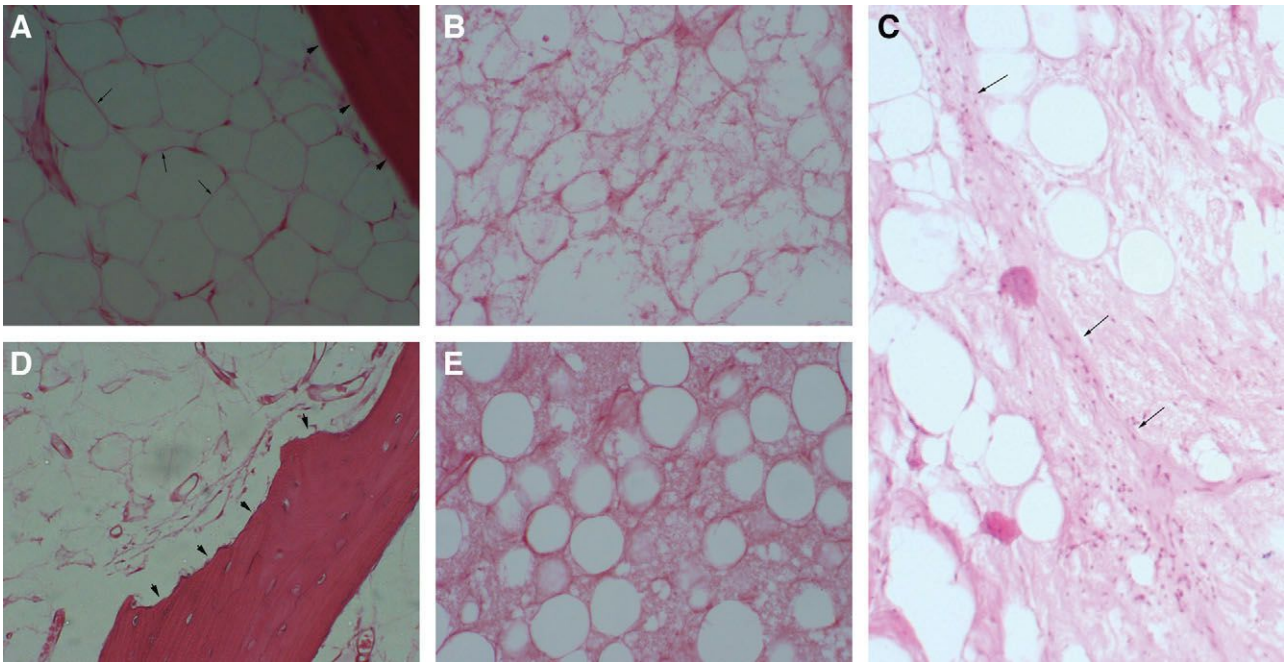


Fig. 16. A, Sagittal histological section of the spongiosa of a navicular bone with normal architecture. The cytoplasmic membranes of the lipocytes are clearly defined (arrows) and the trabecular margins are smooth (arrow heads). Haematoxylin and eosin, × 200. B, Sagittal histological section of the spongiosa of a navicular bone with fat atrophy. The cytoplasmic membranes of the lipocytes are poorly defined. There is dispersed acidophilic material consistent with cellular disruption and oedema. Haematoxylin and eosin, × 200. C, Sagittal histological section of the spongiosa of a navicular bone showing some disruption of the lipocytes architecture and fibroplasia (arrows). Haematoxylin and eosin, × 100. D, Sagittal histological section of the spongiosa of a navicular bone showing marked disruption of lipocyte architecture, increased vascularisation of the marrow fat and a very irregular trabecular margin (arrow heads). Haematoxylin and eosin, × 200. E, Sagittal histological section of the spongiosa of a navicular bone. The lipocytes are reasonably well defined, but there is extensive acidophilic material between lipocytes consistent with interstitial oedema. Haematoxylin and eosin, × 200. (Reproduced from *Equine Vet J* 2012;44:692–698, with permission).

severe navicular pathology develops.[72,98g] However, diffuse increased signal intensity in fat-suppressed images in the spongiosa of the navicular bone associated with primary navicular pathology has often persisted unchanged at follow-up examinations in association with persistent lameness,[98h] although in horses with an acute-onset trauma of the navicular bone, both lameness and high signal intensity may resolve. In other osseous anatomical locations, increased signal intensity in fat-suppressed MR images may persist, despite resolution of pain and lameness, whereas in other horses pain also continues.[13,98,103,104] It is possible that the difference between these clinical groups relates to what histological features the high signal intensity on fat-suppressed images represents and, therefore, whether these are likely to be reversible changes. The potential clinical importance of increased signal intensity in fat-suppressed MR images of the navicular bone was highlighted in a study of horses with lameness of <6 months' duration, with no detectable radiological abnormalities of the navicular bone.[95] Focal or diffuse increased signal intensity was seen in the navicular bone of 108 limbs (75%); unfortunately, its distribution was not described in detail.

Previous studies with the use of histomorphometry,[105] tetracycline labeling of bone,[106] and scintigraphic studies[43,107] have indicated that there is evidence of increased bone turnover in association with some forms of navicular disease, even in the absence of radiological abnormalities of the bone. Increased RU predominantly reflects increased osteoblastic activity but is not synonymous with either pain or lameness[108] and may reflect a functional adaptation to foot conformation and the biomechanical forces on the navicular bone or preclinical disease. Comparison between scintigraphy and MRI has demonstrated that many horses with focal moderate or intense IRU have abnormalities of the navicular bone detectable using MRI.[43] However, scintigraphy can also produce false-negative results, indicating that pathological abnormalities of the navicular bone are not always associated with increased osteoblastic activity. Radiopharmaceutical uptake was normal in the navicular bone of seven of 11 limbs in which the principle abnormality on MR images was diffuse increased signal intensity in fat-suppressed images and hypointense signal in T1W GRE images, and there was mild IRU in the remaining four bones.[77] In contrast, in three horses with

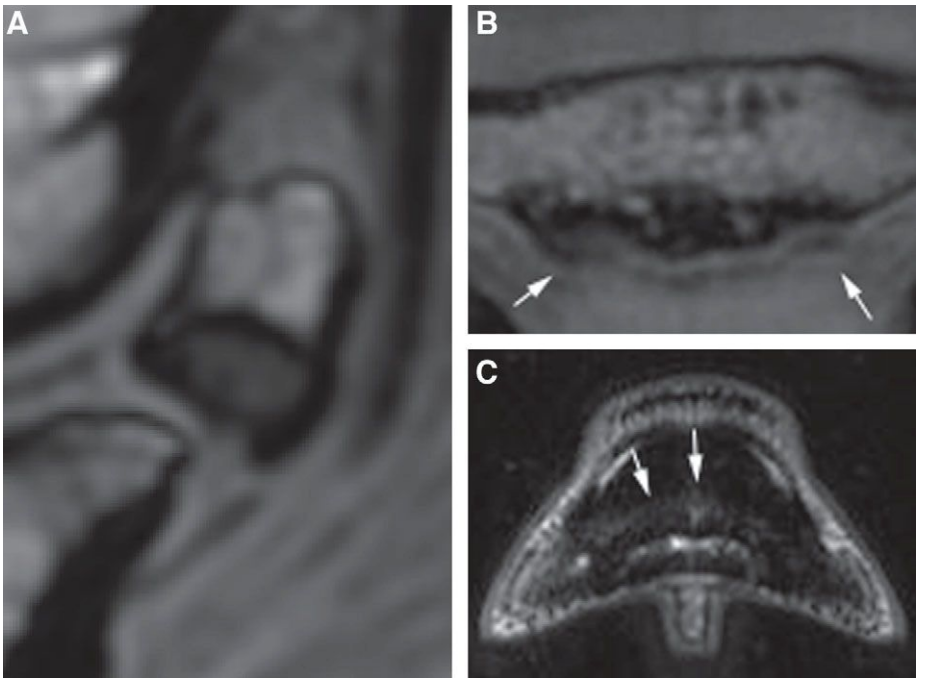


Fig. 17. Sagittal (A) and dorsal (B) spoiled gradient echo and transverse short tau inversion recovery (C) high-field magnetic resonance images illustrating features that showed a positive correlation with histological abnormalities of the distal sesamoidean impar ligament (DSIL). (A) An osseous cyst-like lesion (OCLL) in the distal third of a navicular bone. (B) Distal border fragments at the medial and lateral angles of the bone (arrows), with concave defects in the parent bone and associated osseous reaction. Increased signal intensity in the distal phalanx at the site of insertion of the DSIL (arrows).

acute-onset, severe, unilateral lameness associated with increased signal intensity in the spongiosa of the navicular bone in fat-suppressed images, which was believed to be traumatically induced, there was intense IRU.[98] Lameness in these horses resolved with rest and time. If increased signal intensity in the spongiosa was the principle pathological change in horses with foot pain, without IRU, it was speculated that treatment with the bisphosphonate, tiludronate, may be of benefit, particularly in light of osteoclasts identified in the current study, but negligible improvement was seen in 12 of 12 horses.[98]

Injuries of the Distal Sesamoidean Impar Ligament

The DSIL is an integral part of the podotrochlear apparatus, and we have demonstrated a significant association between the severity of grade of IRU in the navicular bone of lame horses, reflecting abnormal bone modeling, and combined lesions of the CSL and DSIL detected with the use of MRI.[77] The DSIL consists of bundles of longitudinally orientated collagen fibers, interspersed by synovial invaginations from the DIP joint and the navicular bursa, and penetrating blood vessels.[100,109] As a result, a normal DSIL has a heterogeneous appearance on MR images, and differentiation between anatomical variants and pathological changes is not always straightforward.[110] Nonetheless, in a comparison of lame horses and age-matched control horses, high-field MR abnormalities were consistently more severe in horses with foot pain,[36] characterized by osseous fragments or focal mineralization near the origin, irregularity of fiber pattern, swelling of the ligament, and adhesions to the DDFT. However, comparison of MRI findings with histopathology in the same group of horses[76] revealed only fair agreement; thus, there is a significant risk of false-positive results.

To further characterize lesions of the DSIL and to determine relationships between the MRI appearance of the navicular bone and distal phalanx, 50 limbs from 28 horses were examined with the use of high-field MRI and histopathology.[111] Magnetic resonance abnormalities of the DSIL, its origin on the navicular bone and its insertion on the distal phalanx were graded. Sections of the axial third of the DSIL were examined histologically and graded according to fiber orientation, integrity of fibroblasts, collagen architecture, and vascularity.

There were significant correlations between the presence of an OCLL in the distal one-third of the navicular bone, or a distal border fragment, or increased signal intensity in fat-suppressed images at the insertion of the DSIL on the distal phalanx and the histological grade of the body of the DSIL (Fig. 17). There were significant associations between an OCLL in the distal one-third of the navicular bone and the presence of either a distal border fragment, entheseous new bone at the insertion of the

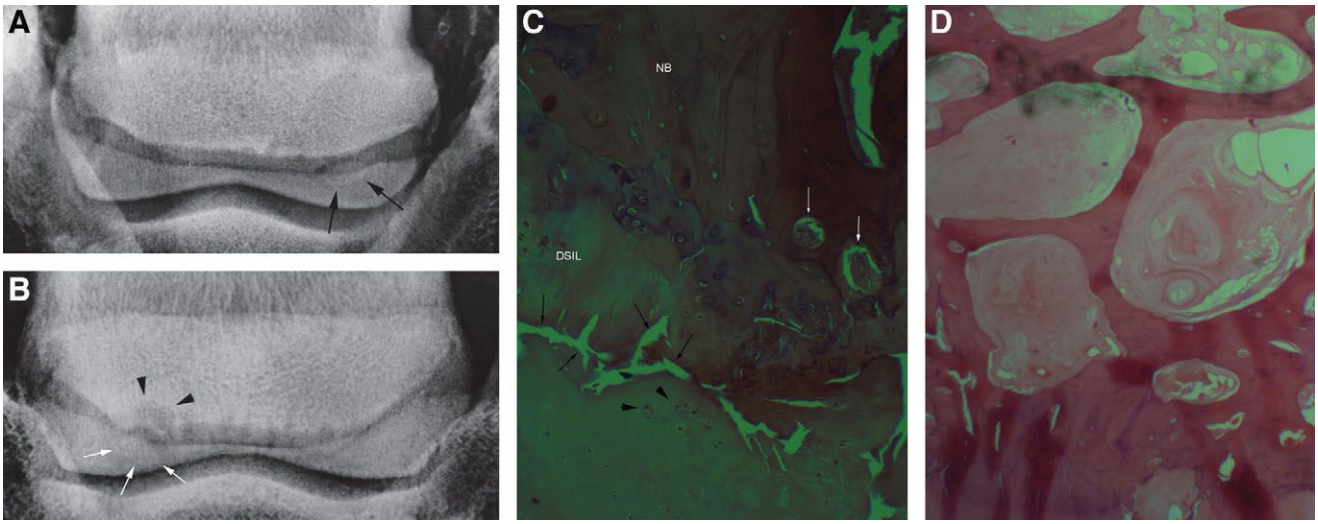


Fig. 18. Dorsoproximal-palmarodistal oblique radiographic images exposed for the navicular bone; lateral is to the right. A, There is a distal border fragment at the junction between the horizontal and lateral sloping borders of the navicular bone (arrows). The adjacent distal border has mild concavity and normal radiopacity. B, There is a distal border fragment at the junction between the horizontal and medial sloping borders of the navicular bone (white arrows). There is a defect in the contour of the distal cortex of the navicular bone, and there is a large radiolucent area in the bone proximal to the fragment (black arrowheads). There are also 7, variably shaped and sized, synovial invaginations along the distal horizontal border of the navicular bone. C, Sagittal histological section of a navicular bone (NB) and the distal sesamoidean impar ligament (DSIL) obtained at the level of an osseous cyst-like lesion at the junction of the distal horizontal and sloping borders, distal to which was a fragment. Proximal is to the top, and palmar is to the left. There is a transverse fissure in the DSIL near the distal cortex of the bone (black arrows); the lesion is surrounded by large chondrones (arrowheads). The distal border cortex is very irregular with loss of calcified zone and focal bone loss (white arrows) (Haematoxylin & eosin, × 10). D, The same bone as Fig. 18C, just proximal to the origin of the DSIL. There are several enlarged bone spaces containing proliferative fibrovascular tissue. This lesion corresponded to an OCLL seen on magnetic resonance images (Haematoxylin & eosin, × 2).

DSIL, swelling of the DSIL, and increased signal intensity in the DSIL in fat-suppressed images. There was a significant association between distal elongation of the palmar border of the navicular bone and the presence of one or more distal border fragments. There were also significant associations between swelling of the body of the DSIL and irregularity of its palmar border or increased signal intensity in fat-suppressed images in the DSIL. Bone resorption at the insertion of the DSIL and/or DDFT on the distal phalanx has also been seen radiologically and/or with the use of MRI in association with lesions of either or both the DSIL and DDFT.[i112] Such lesions have been characterized histologically by bone necrosis.

The combination of swelling of the DSIL, irregularity of its palmar border and increased signal intensity in fat-suppressed images is more likely to reflect genuine injury than any one of these features alone. The results indicated that alteration in size, number, or symmetry of the synovial invaginations in the DSIL is not a reliable feature of injury.

Lesions of the DSIL are rarely seen in isolation, usually occurring in conjunction with other injuries of the podotrochlear apparatus. However, we recently documented a horse in which a distal border fragment of the navicular bone, an OCLL in the distal third of the navicular bone and focal distal sesamoidean impar desmitis were identified as the most likely causes of pain and lameness.[113] No other lesions were identified on MR images. The OCLL was characterized histologically by enlarged bone lacunae containing proliferative fibrovascular tissue.

Distal Border Fragments and Navicular Disease

There is considerable controversy concerning the potential clinical significance of distal border fragments of the navicular bone. In a radiological study comparing 55 sound horses and 377 lame horses, we observed fragments in 3.7% and 8.7% of sound and lame horses, respectively. In lame horses, distal border fragments were present in 24.1% of horses with a diagnosis of primary navicular pathology and in 12.9% of horses with navicular pathology and other associated lesions.[114] There was an association between fragments and the overall navicular bone grade ($P = 0.0013$), radiolucent areas at the angles of the distal border of the navicular bone ($P < 0.001$), and the number and size of the synovial invaginations along the distal border of the navicular bone ($P < 0.001$). It was concluded that fragments may be part of navicular disease. Further evidence was provided for this by a high-field MRI study of 427 horses.[97] Fragments were classified as small, medium, or large on the basis of

their MRI appearance and were graded 1 to 5 on the basis of changes in the adjacent distal border of the navicular bone. Large fragments were usually grade 4 or 5, whereas small fragments were usually grade ≤3. There was a significant association between the presence of a fragment and the total navicular bone grade ($P < 0.001$), OCLLs ($P < 0.001$), increased number and size of the synovial invaginations of the distal border ($P < 0.0067$), increased signal intensity on fat-suppressed images in the distal half of the navicular bone ($P < 0.001$), and size of distal border entheseophytes ($P < 0.0067$). There was an association between grade of the DSIL and grade 4 or 5 navicular bone fragments ($P < 0.0086$). The majority of fragments were associated with proximal extension of abnormal signal intensity in the adjacent navicular bone.

We also investigated the correlation between the presence of distal border fragments detected using high-field MRI and their radiological detection in 427 horses.[96] Medium and large fragments were most likely to be detected radiologically especially if grade 4 or 5, but up to 43% of large fragments were missed. There were significant associations between the presence of a fragment on radiography and the total MRI grade of the navicular bone ($P < 0.0001$); the presence of a radiolucency at the junction between the distal horizontal and sloping borders of the navicular bone and both a fragment observed on MR images ($P < 0.0001$) and OCLLs on MR images ($P < 0.0001$). There was also an association between an increased number and size of the synovial invaginations along the distal border on radiographs and the presence of both a distal border fragment on MR images ($P < 0.0001$) and OCLLs on MR images ($P < 0.0001$).

On histology, partially detached bone islands were separated from the distal border by areas of fibroplasia or fibrocartilaginous metaplasia and were associated with focal osteonecrosis.[76,100] Thus, increased signal intensity on STIR images in the distal border of the navicular bone associated with some fragments may reflect bone necrosis. There may be transverse fissures at the origin of the DSIL (Fig. 18). It has been suggested that movement of a fragment relative to the navicular bone may be a potential cause of pain.[110] In addition, lesions of the DSIL observed in association with high-grade fragments could be painful because of the rich sensory innervation of the DSIL, particularly at its insertion.[109]

5. Conclusions

Accurate lameness diagnosis remains challenging. As knowledge advances, more questions arise. Currently our diagnostic capabilities are superior to our ability to treat lameness successfully. Progress will be made by careful clinical observations, combined with accurate interpretation of diagnostic imaging and evidence-based studies documenting the responses to treatment in sufficiently large numbers of horses.

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How to Perform an Alternative Method to Anesthetize the Palmar/Plantar Digital Nerves

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1. Introduction

The palmar digital nerve (PDN) block is the most common diagnostic analgesic procedure performed for perineural analgesia in the forelimb. The plantar digital nerve (PDN) block is not used as commonly because the palmar digital nerve block; however, it is important in the isolation of pain in some rear-limb lameness.[1]

The referenced site for the injection of the local anesthetic agent for a palmar/plantar digital nerve block ranges from the proximal margin of the ungulate cartilages to mid pastern over the medial and lateral neurovascular bundles.[1–5] However, one reference describes the block as being performed by inserting the needle on the palmar midline, placing the line of the local anesthesia solution in a proximal-dorsal direction to the sites of the medial and lateral palmar/plantar digital nerves.[1]

Recent literature indicates that the PDN block, by use of the commonly prescribed sites, anesthetizes several structures of the foot, the proximal interphalangeal joint (PIP) and other related structures.[1–5] A recent publication described metacarpophalangeal joint lesions identified on magnetic resonance imaging with lameness that resolved through the use of palmar digital nerve analgesia.[6] However, the precise site of the PDN block was not described. The recent published information underscores why the procedure should be done as distal as is possible and practical to achieve anesthesia of the PDNs and limit the proximal and dorsal anesthesia of the digit and fetlock.

The following procedure was first explored by the author as a result of discussion during the Internationales Symposium (Strahlbeinlahmheiten), International Symposium on Podotroclosis, Dortmund, Germany, 1993. The attending Swiss delegation stated that to maximize the diagnostic value of the PDN block, it must be performed much more distal than the sites commonly used. Since 1993, the author has used the following technique to achieve analgesia of the foot. It has provided more consistent information than that found when the block was administered in and around the neurovascular bundle at the proximal margin of the ungulate cartilages. Careful clinical assessment of this block has been that painful lesions proximal to the mid palmar/plantar pastern are not anesthetized. Lesions proximal from the dorsal coronet band are not anesthetized. On several occasions, after incomplete resolution of the lameness from a PDN, intra-articular anesthesia of the coffin joint eliminated the lameness. Lameness from lesions involving the PIP joint will improve but will not be eliminated by

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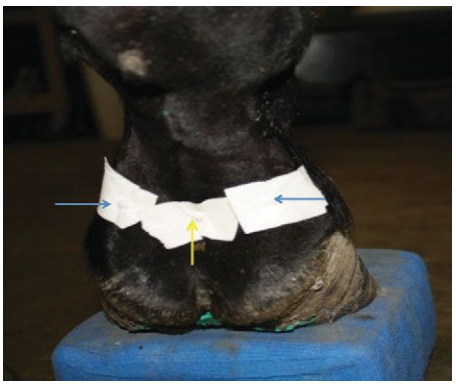


Fig. 1. Dorsal margin of the ungulate cartilages and the injection site are identified with radio-opaque markers taped and superglued to the skin.

the described method. Consequently, a pastern ring block eliminated the lameness.

Before a PDN block is administered, a thorough clinical examination of the lame leg must be performed. A careful, thorough, and methodical application of hoof testers is of paramount importance as a part of the clinical examination.

Application of the PDN block by this technique is very useful and reproducible because (1) the anesthetic can be administered more distal than the level of the proximal border of the ungulate cartilages, (2) only one painful event is experienced by the horse, (3) seldom is restraint necessary (ie, twitching), (4) there is less risk in breaking off a needle, (5) the PDNs can be displaced axially during administration, facilitating the placement of the anesthetic agent on or near the axial surface of the nerves, reducing the spread of the agent dorsally and proximally, (6) inadvertent synoviocentesis of the distal synovial sheath is not likely because the position of the distal digital annular ligament is deep to the site[3] and the palmar distal limit of the distal synovial sheath is proximal to the site of the injection,[7–9] (7) the results are more predictable in one's hands than the commonly recommended site at the level of the proximal border of the ungulate cartilage, (8) the technique eliminates the sensation occasionally found in the region of the coronet band at the midline over the central sulcus of the frog and 1 to 2 cm abaxial to the midline when the anesthetic is deposited over the commonly recommended site (it is suspected that an aberrant nerve branch being more palmar than the neurovascular bundles is responsible for this persistent sensation), (9) the technique significantly reduces the possibility of administration of the anesthetic solution intravascularly in branches of either the digital artery or vein, and (10) the technique eliminates the use of anesthesia of the coffin joint as a method to anesthetize the PDNs.[4]

2. Materials and Methods

A 1150-lb, 12-year-old Thoroughbred gelding scheduled for euthanasia because of atrial fibrillation was used to demonstrate the procedure and investigate proximal dorsal migration of injected agents from the injection site. The first block was done on the left front with a local anesthetic. The second was done on the right front with contrast media. Three days later, the right front digital synovial sheath was injected with contrast media, the right rear PDN site was injected with a 50/50 solution of contrast media and local anesthetic, and the right front PDN site was injected with dye. Before the procedure was performed on the right front and right rear, plantar/palmar nerve blocks were performed proximal to the fetlocks to alleviate discomfort from the procedures.

The site for the initial skin bleb is between the bulbs of the heels 2 to 4 cm proximal to the coronet band on the midline directly above the middle sulcus of the frog, where the dermis is flat over the distal digital annular ligament and the deep digital flexor tendon. The dorsal margin of the ungulate cartilages and the injection site were identified with radio-opaque markers taped and superglued to the

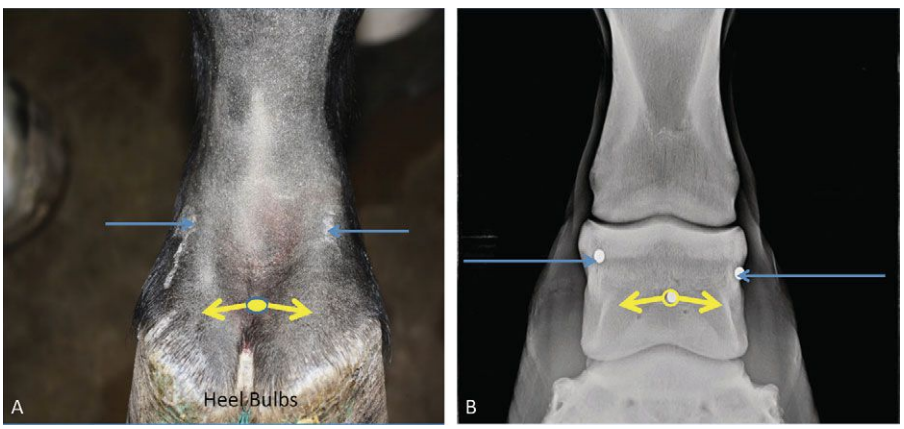


Fig. 2. Tape is removed and the site is radiographed.

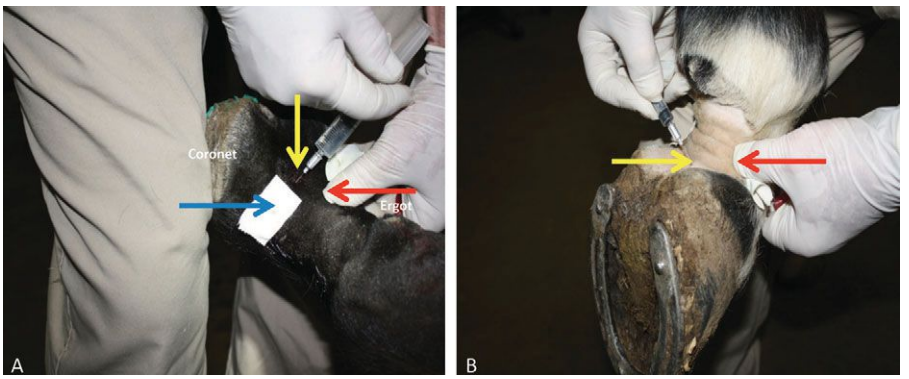


Fig. 3. To administer the palmar digital nerve block, the front limb is picked up and held between the administrator's knees (A). For the plantar digital nerve block, the rear limb is picked up, extended caudally, and held on the examiner's thigh (B).

skin (Fig. 1). The tape was removed and the site was radiographed (Fig. 2). Preparation of the site is accomplished by scrubbing with an antiseptic soap[a] followed by a thorough application of chlorhexadine gluconate[b]/alcohol solution on 4 × 4 sponges. Note: Excess hair is clipped away if the neurovascular bundles cannot be easily palpated, the initial site of injection cannot be well observed, or the evidence of subcutaneous distention from the anesthetic agent cannot be observed and/or palpated.

To administer the palmar digital nerve block, the front limb is picked up and held between the administrator's knees (Fig. 3A). For the plantar digital nerve block, the rear limb is picked up, extended caudally, and held on the examiner's thigh (Fig. 3B); 6 mL of 2% mepivacaine[c] in a disposable 6-mL syringe with a 25-gauge × 5/8-inch (1.6 cm) needle attached is routinely used (0.5–1 mL for the bleb and 2.75–2.5 mL directed from the bleb subcutaneously toward the nerve). The initial bleb, in the majority of cases, can be performed without restraint by applying pressure with movement with the use of the thumb or a finger of the free hand, just proximal to the site (Fig. 3A). Recently, lidocaine/prilocaine topical cream[d] has been applied to the site before the initial injection on a hypersensitive horse. Also, the use of a 3-mL syringe (Fig. 3B) may facilitate the placement of the bleb in the rear limb. From the bleb, the needle is directed abaxially toward the palmar/plantar digital nerve (medial or lateral) in a slightly distal dorsal direction, injecting a small amount subcutaneously as the needle is being advanced toward the nerve the full length of the needle (Fig. 4). The needle is withdrawn and redirected toward the opposite PDN. Often this can be performed without removing the needle completely from the skin. If there is significant resistance to the flow of the agent, the needle should be slightly redirected until there is less resistance and the slight subcutaneous enlargement can be seen. As the agent is being injected, the thumb or a finger of the free hand displaces the neurovascular bundle toward the midline just proximal to the injection site (Fig. 4). This facilitates depositing the anesthetic near the axial surface of the nerve, avoiding intravascular injection, and assists in limiting the proximal and dorsal migration of the anesthetic (Fig. 4).

After the injection, the horse can be allowed to stand quietly until the effectiveness of the block is

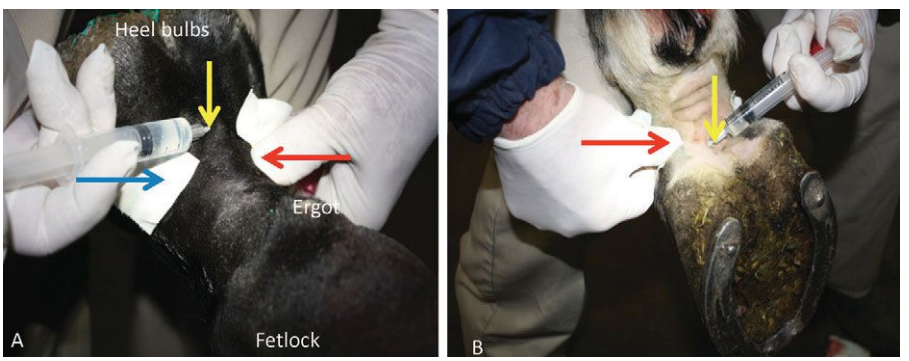


Fig. 4. From the bleb, the needle is directed abaxially toward the palmar/plantar digital nerve (medial or lateral) in a slightly distal dorsal direction, injecting a small amount subcutaneously as the needle is being advanced toward the nerve the full length of the needle.

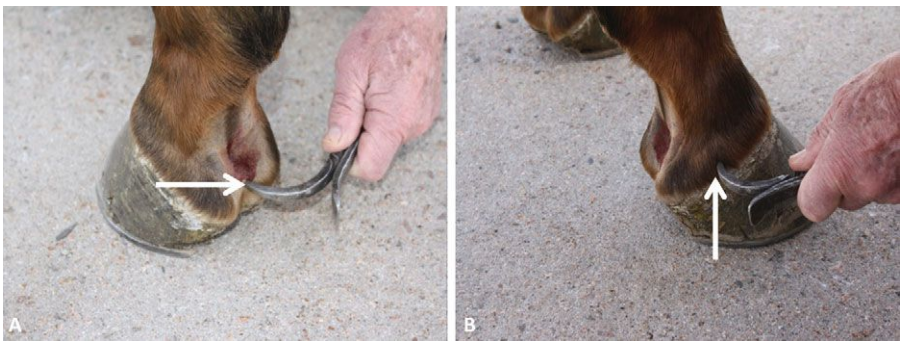


Fig. 5. Assessment begins at the coronet band directly above the middle sulcus of the frog, progressing dorsally toward the toe both medially and laterally at 1-cm intervals until pain response is produced.

evaluated 15 minutes after injection. The loss of skin sensation distal to the block is evaluated by applying pressure to the coronet band with a small, blunt instrument directed dorsally. It is important to start the assessment beginning at the coronet band directly above the middle sulcus of the frog, progressing dorsally toward the toe both medially and laterally at 1-cm intervals until a pain response is produced (Fig. 5). This is followed by the application of hoof testers to evaluate the loss of a positive reaction present before the block is given (Fig. 6). The next step is re-evaluation of lameness to determine whether pain is eliminated, or, if not, to what degree.

Initially, the PDN site of the right front was injected with 6 mL of iodinated contrast media,[e] as outlined for the PDN block to evaluate migration of the media. Serial radiographs were taken: the first was 2 minutes after injection and the last was 1 hour after injection. Note the contrast media in the afferent lymph channels (Fig. 7).

Three days later, in the right rear limb, a plantar digital nerve block was performed with 3 mL of mepivicaine and 3 mL of iodinated contrast media to evaluate migration of diluted media. Radiographs were taken within 5 minutes (Fig. 8). There is evidence of more lymph drainage in the rear limb than in the front limb.

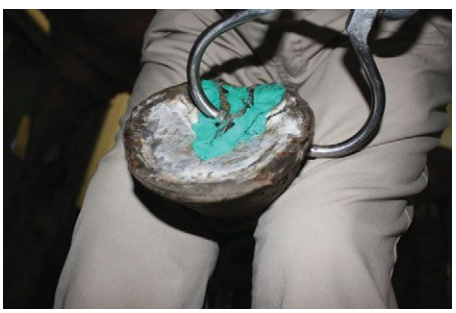


Fig. 6. Assessment is followed by the application of hoof testers to evaluate the loss of a positive reaction present before the block is given.

Three days after injection of the right front PDN site with contrast media, the right front digital synovial sheath was injected with 8 mL of iodinated contrast media. The lateral approach between the palmar annular ligament of the fetlock and proximal digital annular ligament was used. Radiographs at 2 and 30 minutes after injection demonstrated the palmar distal limit of the sheath to be at the level of the proximal border of the ungulate cartilage (Fig. 9).

After the tendon sheath study, 6 mL of methylene blue[f] was injected at the described site of the PDN block and in the same manner. Fifteen minutes later, the horse was euthanized and exsanguinated. The right front leg was immediately disarticulated at the carpometacarpal joint. The skin was later removed from the carpus to the coronet band to evaluate the migration of the dye. The bulk of the dye remained subcutaneously at the injection site (Fig. 10A). The remainder diffused abaxially to and around the palmar digital nerve and digital artery. Very little dye was evident around the digital vein or the dorsal branch of the digital nerve (Fig. 10B). However, the dye was evident in the afferent lymph channel from the injection site to the transection site (Fig. 11). There was minimal dye deep to the superficial fascia of the digit (Fig. 12A) and no evidence of the dye in the distal synovial sheath or around the deep digital flexor tendon (Fig. 12B).

3. Results

This procedure has produced consistent results in eliminating pain causing lameness in the foot. It has been effective in differentiating sites of pain proximal to the foot, including the dorsal aspect of both the coffin and the PIP joints and the fetlock. This technique has eliminated occasional pain from an aberrant nerve between the palmar digital nerves supplying sensation to the skin between the bulbs of the heels and deep tissues of the caudal portion of the foot (eg, hoof tester response).

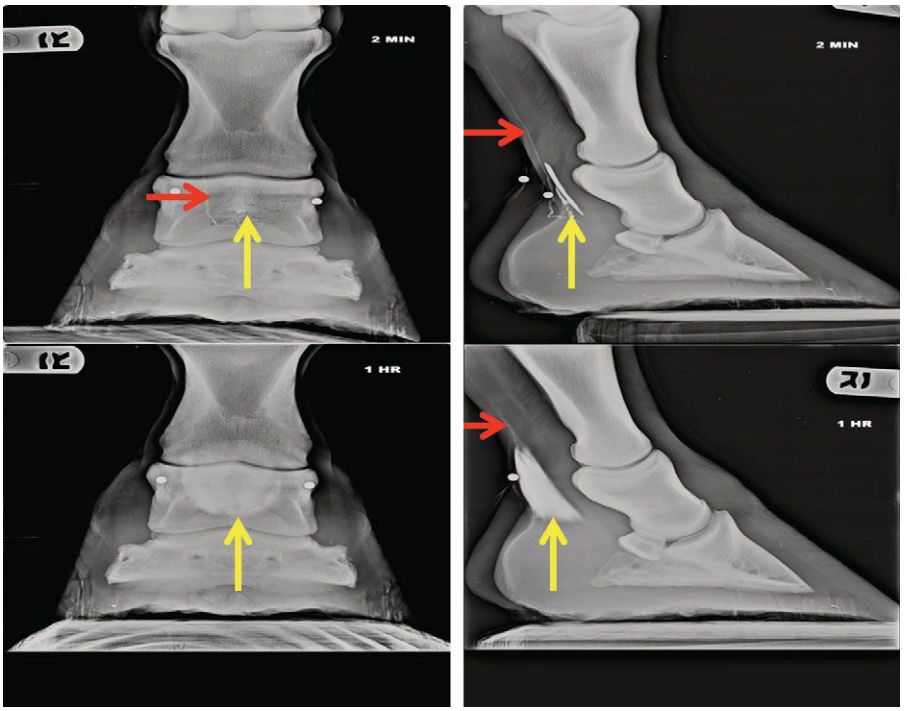


Fig. 7. Contrast media in the afferent lymph channels.

Since the information obtained from the International Symposium on Navicular Disease, Dortmund, Germany, 1993, this procedure has evolved in the author's hands. The number of cases in which the described procedure has been used during the 19-year time span of use is difficult to identify. However, a recently adopted record system[g] tabulated the number of PDN blocks performed by the author from February 2010 to February 2013 to be 179. With the use of this information, it could be extrapolated that over the past 19 years, the author performed approximately 1000 PDN blocks.

4. Discussion

The procedure facilitates a consistent site for the administration of a local anesthetic agent to the medial and lateral palmar/plantar digital nerves. It is easily performed, and the results are highly predictable. With the described technique, in the majority of cases, the level of anesthesia, as determined by the lack of response to pressure at the coronet band from a blunt instrument, will be from the middle sulcus area of the coronet band dorsally to one fourth to one third of the distance to the toe.

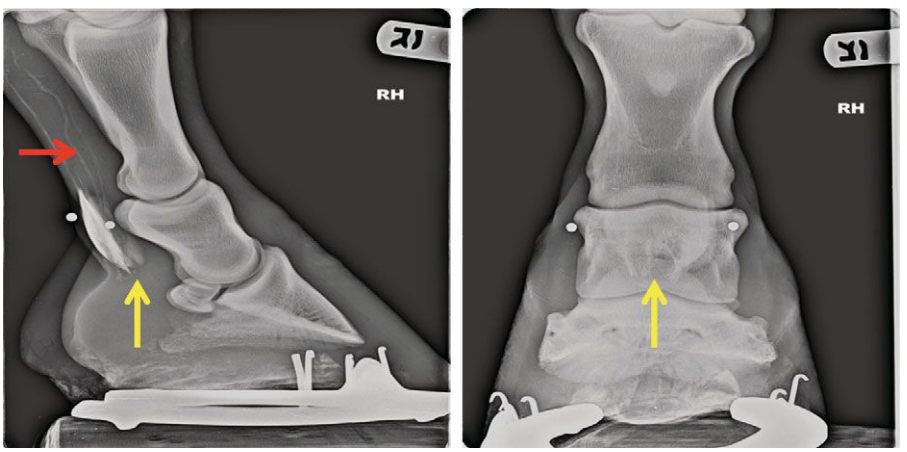


Fig. 8. Plantar digital nerve block performed with 3 mL of mepivicaine and 3 mL of iodinated contrast media to evaluate migration of diluted media. Radiographs were taken within 5 minutes.

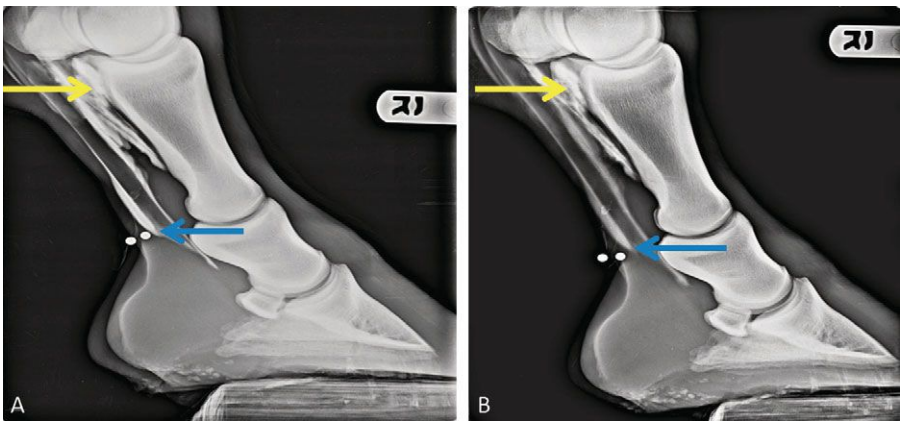


Fig. 9. Radiographs at 2 and 30 minutes after injection demonstrate the palmar distal limit of the sheath to be at the level of the proximal border of the ungulate cartilage.

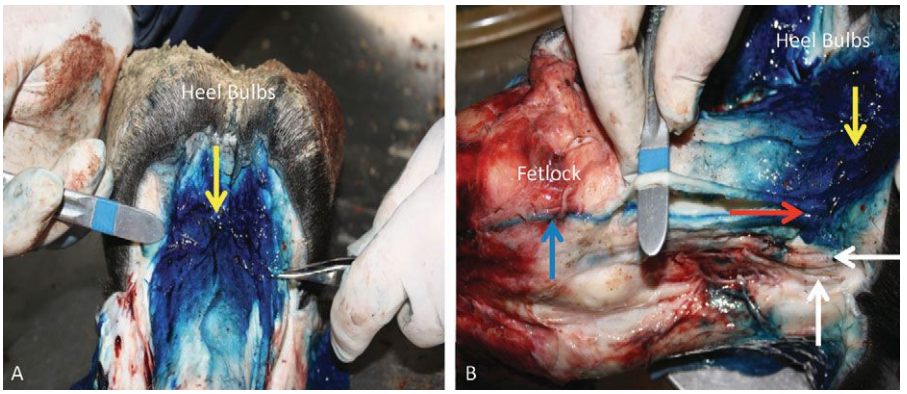


Fig. 10. Bulk of dye remained subcutaneously at the injection site (A). The remainder diffused abaxially to and around the palmar digital nerve and digital artery. Very little dye was evident around the digital vein or the dorsal branch of the digital nerve (B).

However, occasionally the anesthesia extends to the toe portion of the coronet band. In the author's opinion, this does not materially affect the interpretation of the block, but it does support the statement made by G. Kent Carter, "Diagnostic anesthesia has limitations; although the majority of horses have a reliable response variations are occasionally encountered, and confusing or inaccurate results are obtained."[4] A positive response to the hoof testers after the PDN block is evidence that the block failed and must be repeated. The procedure eliminates sensation from the occasional aberrant nerve in the palmar portion of the pastern between the PDNs. Failure to eliminate sensation from such an aberrant nerve will compromise the interpretation of the PDN block and misdirect the focus on the site of pain producing lameness.

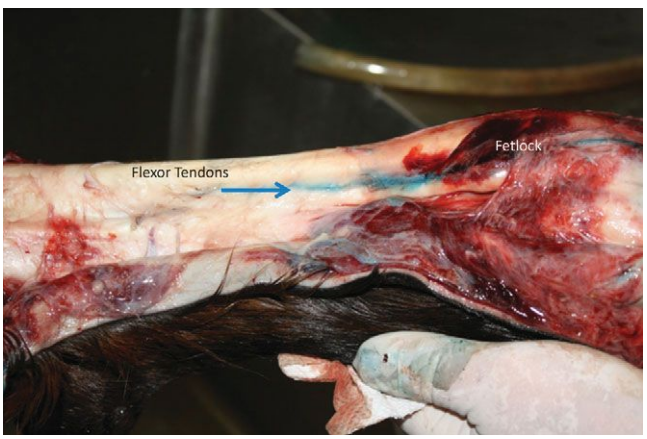


Fig. 11. Dye is evident in the afferent lymph channel from the injection site to the transection site.

When lesions involving the dorsal aspect of the PIP have been identified by clinical examination, radiographs, and/or magnetic resonance imaging, the described technique will improve but not eliminate the degree of lameness, nor will a positive response to the flexion of the phalangeal joints be eliminated. Consequently, when a ring block of the pastern eliminates the lameness and the positive response to flexion of the phalangeal joints, the pain is likely to be from the PIP joint and/or adjacent soft

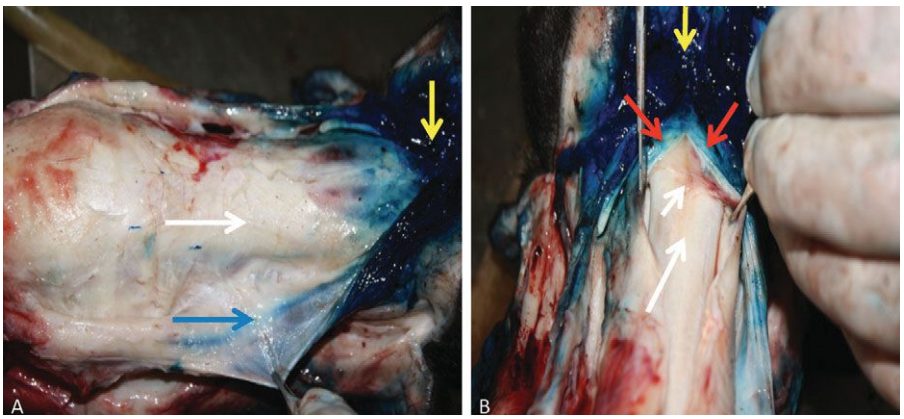


Fig. 12. Minimal dye deep to the superficial fascia of the digit (A) and no evidence of the dye in the distal synovial sheath or around the deep digital flexor tendon (B).

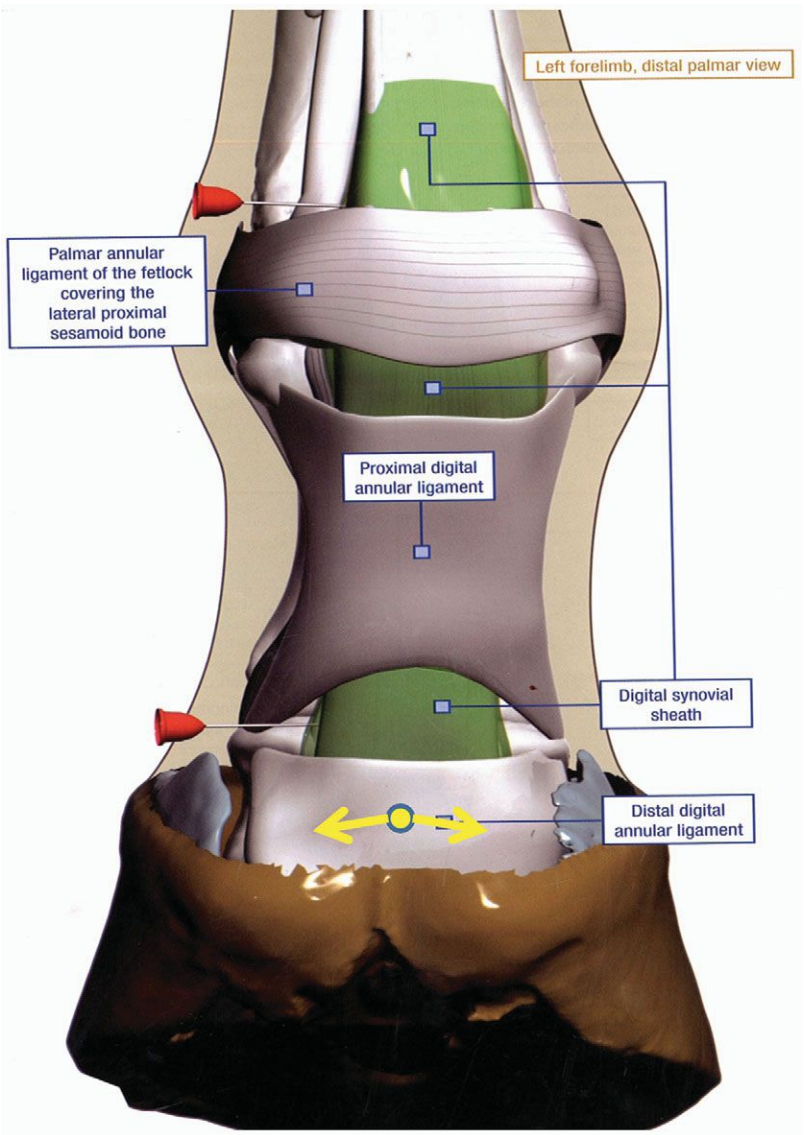


Fig. 13. Distal palmar limit of the digital synovial sheath is proximal to the site; the distal digital annular ligament is a barrier.

tissue structures. If there is little evidence of pathology in the PIP and the described PDN block improved the lameness but does not eliminate the lameness, intra-articular anesthesia of the coffin joint is the next step in an attempt to determine the site of pain. Often the lameness is eliminated. These findings indicate that the technique described does not routinely anesthetize the dorsal aspect of the distal interphalangeal joint or PIP joints, nor the fetlock region. This observation is similar to that of Bassage and Ross.[1]

The proximal dorsal migration of the contrast media and dye was less than anticipated. The immediate uptake by the afferent lymph vessels was an unexpected finding. Clinically, the amount of anesthetic in the lymph vessels appears not to materially effect anesthesia of sites proximal to the PDN block.

Because the described technique will eliminate sensation from an aberrant nerve between the palmar digital nerves, it should not be used to predict the effective analgesia of a PDN neurectomy. The PDN block for this purpose should be done at the anticipated site of the neurectomy.

Care should be taken not to enter the distal portion of the digital synovial sheath of the flexor tendons. This has not been a problem when the type of needle described is used and the anesthetic agent is deposited subcutaneously. Also, the distal palmar limit of the digital synovial sheath is proximal to the site.[7–9] In addition, the distal digital annular ligament is a barrier (Fig. 13). The possibility of breaking off a needle is reduced by the direction of the needle insertion being horizontal rather than being directed distally and parallel to the plane the horse's leg will travel if jerked forward. The introduction of a contaminate is reduced with the use of the small needle and proper preparation.[10]

Even though the specificity of the palmar/plantar digital nerve block has come into question, its use in the diagnosis of lameness remains a very important procedure to initiate the isolation of pain causing lameness. Also, the information is beneficial in determining the need for additional diagnostic modalities, selection of therapy, and providing a prognosis for the problem. The analgesic effect of the anesthetic migrating proximal in the afferent lymph channels poses a question that adds to the confusion as to what structures are anesthetized proximal to the PDN block. Clinically, this does not appear to be a problem because painful sites proximal to the PDN block routinely are eliminated by blocking sites proximally (ie, pastern ring block, four-point block, high suspensory block).

In summary, the technique described is relatively easy to perform and can be performed with minimal restraint; there is only one painful experience for the horse—the anesthetic is deposited on the axial margin of the PDNs; the invasion of the vascular portion of the neurovascular bundle is very unlikely; and the anesthetic agent is easily deposited below the level of the proximal limit of the ungulate cartilages. If aberrant nerves are present axial to the PDN, they will be anesthetized. The problem of anesthetizing sites of pain proximal to the PDN site is minimized. The results are reproducible.

Acknowledgments

The author thanks Katie Thomason, CVT; DeeAnn Wilfong, CVT, VTS-EVN; Carrie Turner, Secretary; Jairo Paucart, MV; and Kara Waldmeier, IT.

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[a]Prepodyne Scrub, West Argo, 11100 N. Congress Avenue, Kansas City, MO 64153.
[b]Chlorhexidine, MWI, Boise, ID 83705.
[c]Carbocaine, V, Pharmacia & Upjohn Company, Division of Pfizer Inc, New York, NY 10017.
[d]Lidocaine 2.5% and Prilocaine 2.5% cream, Hi-tech Pharmacal Co, Inc, Amityville, NY 11701.
[e]Omnipaque, 240 mg/mL, Distributed by GE Healthcare Inc, Princeton, NJ 08540.
[f]Methysorb, Elanco Products Co, a division of Eli Lilly and Company, Indianapolis, IN 80120.
[g]Hospital and Veterinarian Management System (HVMS), Business Infusions, Suite 317, 612–500 County Hills Bend NF, Calgary, AB, Canada T3K 5K3.

Veterinarians Addressing Local, State, and Federal Challenges in Man-Made or Natural Disasters

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Veterinarians must assume an increased role in the planning, management, response, and recovery in the face of disasters. Given virtually any type of disaster, animals will be involved. In a short period of time, since the start of the century, our country has experienced numerous horrific events such as the Twin Towers, hurricanes Katrina, Rita, Ike, and, most recently, Sandy; Joplin, Tuscaloosa, Texas, and Oklahoma massive tornadoes; and wildfires. The result is extensive human and animal suffering, prolonged recovery (many are ongoing), and extensive casualties. During this same period of time, society has ramped up its collective reverence for the importance of companion animals and livestock. The expectation of significant and effective leadership and participation from the veterinary profession has increased greatly. The manner and mechanism of such participation by our profession in a more organized and efficient manner should progress beyond no response, volunteer activation after the fact, or dependence on outside entities. No single group of individuals, other than licensed veterinarians, have the knowledge base, experience, and compassion to address the many and varied needs that affect the animal population in a disaster; thus, we should do no less than our abilities enable us. This report briefly states the why and the how such participation can evolve. Authors' address: Large Animal Clinical Sciences, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station, TX 77843; e-mail:wmoyer@cvm.tamu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

This presentation is designed to better understand the rapidly increasing need for veterinary expertise in a disaster as well as to inform practitioners as to the various ways and means in which one can participate. Disasters, both man-made and naturally occurring, are an unfortunate fact of life. Clearly, the type (hurricane, tornado, wild fire, explosion, earthquake, infectious disease outbreak, terrorist initiated event, etc); the size and location (geographic area/terrain, population density); the presence of complicating factors (associated chemical spills, storm surge, and salt toxicity, etc); and available resources collectively determine the level of needed response and the likelihood of reasonable and timely recovery. In virtually all instances, animals are victims (deceased, injured, ill, and with or without owners). Clearly and appropriately, assistance and preservation of human lives is the priority of a response and recovery; however, it is important

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to note that our nation's concern for its animal population continues to grow. For example, the Robert T. Stafford Disaster and Emergency Assistance Act (summarized) was amended and mandates that preparedness operation plans address the needs of household pets and service animals before, during, and after disasters. The USDA has also mandated that Emergency Disaster Preparedness plans be in place at every USDA licensed animal research facility by mid 2013. Texas HB-88 amended the government code to require the Texas Division of Emergency Management to assist political subdivisions in developing plans for the humane evacuation, transport, and temporary sheltering of service animals and household pets in a disaster. The point is that most plans are non-existent or are in their infancy and furthermore they require expertise that best originates from the veterinary medical profession.[1–5]

The degree, size, and lasting effects of disasters are often only of public concern for as long as it is media-worthy. Two examples experienced by these authors lend credence to the devastating effect of some disasters as well as the relative lack of planning for animal populations. Hurricane Ike (2008) was in Louisiana, Texas, and northern Mexico at the same time. The 25-foot storm surge went inland Texas as far as 42 miles. Thus, much of the surface water became contaminated and made brackish. It is estimated that 30,000 to 40,000 cattle were lost along with a resultant estimated 80% loss of subsequent calf crop. An estimated 100 horses died on one location, presumably from salt toxicity. The 2011 Bastrop Complex Fires, Bastrop, Texas, destroyed between 1600 and 1700 households and affected 30,000 acres. The county was not prepared because there was no effective county animal issues plan. Two veterinary hospitals (operational during the fires) in the county were quickly overrun with animals requiring treatment for burns and injuries, along with owners requesting sheltering as the human population was forced to evacuate. One practice made the decision to assist without charges; the other made an attempt to recover a fraction of the costs. In both instances, these practices suffered significant and lasting economic damage.

A volunteer may wish/desire and have the means to respond to an event as a result of a public call for help (hurricanes Katrina and Rita, for example). Acceptance, however, should be based on a number of known factors:

- Having a reasonably precise/accurate situational report describing risks and hazards.
- Accurate knowledge of what are the skill sets that are being requested to be sure of a good fit; often meaning an individual capable of handling a myriad of situations with a clear mission of stabilizing patients, thus allowing for safe and humane transport to a shelter.
- What human needs will be provided for the volunteers at the staging or working area.
- What are the requirements for credentialing for entry in the event site and/or other potentially required identification.

Volunteers, regardless of their willingness and even their expertise, can either be part of the solution or an additional problem that must be tended.

2. Participation From the Veterinary Profession

There are a variety of ways in which licensed veterinarians and the veterinary community at large can participate and be of value to their respective community, region, state, nation, or the world. Prior planning, the initial response, and facilitating recovery from a devastating event have multiple pieces—any one of which, parts of, or all—are greatly enhanced through veterinary expertise and participation. The following succinctly describes those pieces and how one can participate or at the very least help to diminish personal or practice loss.

- The development of a personal evacuation plan to protect the individual, one's family, pets/animals, property, and financial stability is essential. The following website (http://vetmed.tamu.edu/vet) provides such a template along with instruction and is self-explanatory. A willing individual can only be of value if he or she is assured that his or her personal and family needs are being met.
- The careful development of a practice evacuation plan is a very key business element and is usually an ignored item. Such a plan should be developed as an all-hazards plan that allows for the worst-case scenario (facility is closed, badly damaged, or destroyed). Key elements include meeting employee needs; preservation of medical and financial records; up-to-date accurate inventory accounting; management of controlled substances; how, when, and where to discharge hospitalized animals in an evacuation; and the list goes on. The same website provides a template along with instructions that can be easily modified or amended to fit specific practice needs.
- The most useful means of providing significant help, in the opinion of the authors, is to become an active member of the State VMA and/or County (or other jurisdictions) Animal Issues Committee allowing for and helping to create a local response. Who better knows the needs and the sources of resources than those who live there? This involves developing plans in concert with the jurisdiction that address the animal needs. Unfortunately, such plans are invariably absent or are in an outline without substance. Counties and regions with a history of repetitive events are more likely to have such plans. A well-developed all-hazards plan

provides for the material, facility, medical/surgical, shelter, and personnel needs before the event, thus facilitating a much more rapid recovery and a more likely means of saving lives.
- Additional opportunities include local, state, regional, and national Veterinary Medical Associations, such as: AAEP Emergency Guidelines, http://www.aaep.org/emergency_prep.htm; AVMA VMAT, https://www.avma.org/kb/resources/reference/disaster/pages/default.aspx; AWIC/USDA, http://awic.nal.usda.gov/companion-animals/emergencies-and-disaster-planning; APHIS/USDA–join NAEHRC, http://www.usda.gov/wps/portal/usda/usdahome?contentidonly=true&contentid=Emergency_Preparedness_and_Response.html.

3. Development of an Animal Issues Plan

The major components of such a plan include the following:

- Development of a Memorandum of Understanding with the jurisdiction.
- Understanding and using the Incident Command System (provided by the county/jurisdiction's Emergency Management office) to understand how information, requests for resources, ability to work with associated responders, authority management, and ultimately reimbursement is managed.
- Development of a communication system to alert and inform animal owners before, during, and after the event.
- Development of an animal evacuation plan (where and when to congregate, assisting evacuations for those unable, routes, what to bring, previously agreed upon sites, etc).
- Development of a temporary Animal Shelter (3–5 days) for those animals left behind that emerge or are rescued.
- Development of a temporary (3–5 days) Veterinary Medical Operations facility (making the assumption that existing practices are unable) to include assessing and managing large animals that were not evacuated.
- Development of a plan for carcass removal, disposal, and public health concerns.
- Development of a plan to ultimately reunite owners and animals.

It is important to point out that veterinarians are essential in developing the plans but that does not imply that those veterinarians are necessarily those who implement. Thus, the need for volunteers, animal expertise, and commitment from a variety of individuals from the community is a requirement.

A Memorandum of Understanding is developed and agreed on with the jurisdiction to provide and have in place the required elements (resources) and to ensure that the cost of materials, and so forth, is borne by the appropriate jurisdiction. Thus, such a memorandum states clearly that the jurisdiction provides a variety of resources (drugs, generators, tents, buildings, the necessary non-veterinary personnel and associated human needs); the absence of such an agreement means there will be no remuneration.

A clear understanding of how the Incident Command System works can be provided through multiple sources, the easiest of which to obtain would be through the local Emergency Operation Office for the jurisdiction. The system is based on past experiences in combination with best business practices and is logical, useful, and a means to track resources and communications.

A carefully constructed communications plan/network is a necessity. This should involve expertise from the local media (TV, radio, newspapers), social media, and list serves (client lists, for example). It involves knowing the local population and thus how best to communicate information to a variety of ethnic and cultural groups who otherwise would not have access. Many of the animals left behind are likely to be owned by those who are unaware.

An Animal Evacuation plan is developed to assist three major groups of the animal owning population: those individuals needing assistance (lack of transportation, people with special needs, etc.); those who have the means but need accurate information as to how and perhaps where to evacuate to; and those who own/manage animals that are likely to not be evacuated, which often includes large animals (livestock and horses). Specifically, the last group requires information about how to "shelter in place" in the face of an impending event (this might mean getting them to high ground with probable flooding, cutting fences in the face of a prairie fire, closing questionable housing structures that might fail in high winds, etc). This plan would include defining a means of identifying animals that are likely to be displaced and separated from their respective owners.

A temporary Animal Shelter Plan is developed to manage animals that were left behind but are not in need of significant medical care; a good plan calls for such a facility to be in place for 3 to 5 days, after which the permanent facilities/operations (such as local/regional humane associations) would take the lead through prior arrangement and assume the burden of reuniting or adopting out the remaining animals.

A temporary (3–5 days) Veterinary Medical Operation (VMO) plan is developed, making the assumption that the local veterinary operations are unable to operate and assist with the mission of managing the animal victims within the practical parameters defined by the participating veterinarians. Thus, it will require developing triage protocols and defining limits of care (basically to stabilize individuals so they can be successfully and humanely transported to animal shelters or facilities that are functioning), on the basis of available resources. Euthanasia,

unfortunately, will be a viable option in most disasters. Animal and human needs (shelter, food, water, and power) must be in place before such an operation becomes operational. This would require in the planning the selection of a safe location or locations; defining the necessary personnel; necessary resources (facility, equipment, supplies to support animal and man); medical record-keeping; animal identification; and a description of how injured/ill animals will be identified, treated, and tracked and be moved up the chain and returned to their respective owners. Such a plan should include honoring requests for field assessments to define the needs of animals left behind but not presently able to be transported (livestock, for example).

It should be evident that such an undertaking requires significant commitment in time, expertise, experience, leadership, and cooperation. The easy aspect is treating the animals; the difficult part is developing a plan that recognizes the considerable resource needs and the eventual implementation of that plan. The "art form" with which equine and large animal practitioners are uniquely gifted, is how to handle problems and situations that are not in the plan.

"Plans are useless, planning is essential" (D.D. Eisenhower).

4. Summary

The animal-owning public has ever-increasing expectations from our profession; one of which is to show up in a crisis. The "lessons learned" through massive tragedies are clear: disasters will happen, rapid recovery is dependent on "plans in place" to provide the resources (materials and people) with a need for expertise at the site (boots on the ground). Dependence on out-of-region help and expertise only delays (sometimes for considerable time) the response and requires a significant "learning curve" for those who venture in. It is apparent that a well thought-out, in advance of the event, plan, on the basis of local expertise, knowledge, and commitment greatly enhances the likelihood of lessening the damage and speeding up the recovery. This is our collective responsibility. This presentation is a brief introduction into why and how. Active and meaningful involvement begins with the local jurisdiction emergency office.

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A Simple Method of Splinting the Equine Carpus

Robert D. Lewis, DVM

Immobilization of the equine carpus is a necessary component in the management of a variety of injuries. This report describes a simple way to accomplish this with a splint using readily obtainable materials, which can be accomplished relatively easily in the standing patient. Author's address: Elgin Veterinary Hospital, Inc., PO Box 629, Elgin, TX 78621; e-mail: RLewis7752@aol.com. © 2013 AAEP.

1. Introduction

Immobilizing the equine carpus in an extended position may be indicated in the management of radial nerve paralysis and selected cases of ulnar or olecranon fractures and to aid in healing of some soft tissue injuries of the carpus, particularly on the dorsal aspect. Immobilization is an important component of emergency management of cases that have sustained multiple carpal bone fractures (crushed carpus) resulting in carpal instability. This also applies to some fractures of the distal aspect of the radius, as a means of stabilization of the limb before surgical repair or cast application.

2. Materials and Methods

Materials described in this article will be suitable for the average adult horse, weighing in the 400- to 600-kg range. Adjustments will be necessary in the size of some of the components for smaller patients. Materials needed are as follows:

(1) 24-inch section of 6-inch-diameter schedule 40 PVC pipe

(2) 24-inch section of 6-inch-diameter C900 PVC pipe

(3) five rolls of 12-inch wide cotton combine or 12-inch-wide roll cotton

(4) five rolls of 4-inch cohesive bandage

(5) three rolls of 4-inch elastic adhesive tape or one large roll of duct tape

The PVC pipe, both the schedule 40 and C900, are cut lengthwise to create two identical halves. One end of each of the two C900 pipe halves is tapered on one corner, such that when they are applied, one half is applied to the distal limb medially and the other half laterally. The distal corners should be tapered to prevent abrasions on the dorsal aspect of the pastern. It is recommended to also slightly round the corners of the dorsal end of one of the halves of the schedule 40 PVC pipes. Cotton combine roll is applied to the limb beginning at the foot, progressing up the limb with subsequent rolls as far dorsally as possible. This material is layered as necessary to achieve a relatively tubular appearance to the final bandage. The intent of the cotton bandaging is to create a snug fit when the PVC splints are applied. The author prefers a 4-inch cohesive bandage to bind these sequential layers as they are placed. When the configuration of the bandage is satisfactory, the C900 pipe halves are applied to the distal limb, one half applied medially and the other half laterally. They are taped together tightly at the distal end and the midbody, with either duct

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tape or elastic adhesive tape, so the opposing cut edges are in contact. One of the schedule 40 pipe halves is then placed on the caudal aspect of the limb, the dorsal end being positioned just below the upper limit of the bandage. Pressure is placed on the dorsal carpus to force it into the splint while the upper splint is pulled cranially. This should gain full extension of the carpus. The leg and splint are held in this manner while an assistant binds this splint with tape.

This splint, when properly applied, should be very rigid and fit snugly on the limb. The distal end should rest on or slightly above the ground caudal to the heel of the foot, and the other end should be as far proximal as can be obtained with the bandage.

3. Discussion and Conclusions

The described splint technique has been used by the author successfully on dozens of the various described cases. This technique was developed out of frustration in the use of other more conventional splinting methods. A distinct advantage of this splint is that it can be easily applied in most standing patients, reducing the need for full limb cast application under general anesthesia. Compared with casting, the ability to remove the splint and change the underlying bandage materials may be more desirable in cases of soft-tissue trauma. Another distinct advantage is the inherent ability to vary the length of the splint to achieve an optimum fit. The main disadvantage is the necessity to achieve the necessary configuration of the underlying bandage to facilitate a uniformly snug fit inside the splints. Also, this splint will not provide immobilization as rigid as does a thinly padded, full limb cast.

The author has found this splint to be particularly useful in cases of temporary radial nerve paralysis from trauma to the lateral aspect of the humeral region.[1] Although some cases are relatively transient in nature, requiring 2 to 3 weeks before sufficient nerve function is regained, this splint has been used to manage cases lasting as long as 4 months. It has also been used in several cases of temporary iatrogenic paralysis of the extensor branches of the radial nerve caused by infiltrate of local anesthetic in the humeroradial joint.

Splinting of the carpus is indicated in the management of some cases of ulnar or olecranon fractures.[1] Relatively non-displaced physeal fractures of the olecranon process in young horses can be managed quite well with splinting of the carpus and stall confinement. Splinting is also an aid in management of ulna and olecranon fractures when economic considerations preclude surgical repair with internal fixation.

Cases of multiple carpal bone fractures resulting in instability of the carpus typically are best managed with some form of surgical intervention combined with external coaptation such as a cast. However, many acute severe orthopedic injuries require immediate stabilization at the time of injury to assist in transport to a surgical facility.[2] This is also done to facilitate some weight bearing on the injured limb in an effort to reduce the risk of contralateral limb laminitis developing.

The author has used this splint technique in several incomplete fractures of the distal radius that presented with open wounds from external trauma. This splinting system will allow management of the wounds while protecting the distal radius during the time required for adequate fracture healing. Additionally, this splinting technique has been used to protect wound repairs of several cases of severe cranial carpal lacerations with open joints that were surgically repaired under general anesthesia.

In conclusion, the described splinting system can be an effective aid in managing many conditions that benefit from carpal immobilization and support. This system utilizes readily obtainable materials and is relatively simple to apply to a standing patient.

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Implications of Melanoma in the Pre-Purchase Examination

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The biologic behavior of equine melanoma can range from localized, small, and non-invasive to metastatic, proliferative, and catastrophically invasive. Therefore, it is important during the pre-purchase examination to note and accurately record the presence of any tumor suspected as melanoma, capture its current status photographically, and clearly communicate to the potential buyer that its future implications, although unpredictable, can be serious. Author's address: Werner Equine LLC, 20 Godard Road, Box 5, North Granby, CT 06060–1400; e-mail: hwwvmd@wernerequine.com. © 2013 AAEP.

1. Introduction

The following case report regards an adult gray gelding seen for pre-purchase examination. A single, small melanoma was identified on the sheath (Fig. 1) and suspected as the cause of a swelling on the head. Two years later, the horse was euthanized as a consequence of terminal metastasis of melanoma. The initial recording and explanation of the implications of equine melanoma were recalled favorably by the owners at the time of euthanasia and were appreciated as helpful guidelines to them at the time of purchase.

2. Materials and Methods

A single case report with follow-up of 2 years after purchase is described. The pre-purchase examination was performed in a thorough, systematic manner with accurate recording, supportive imaging, and reporting to and interpretation for the buyer.

3. Case Reports

A 12-year-old gray Thoroughbred gelding ("Chaz") was examined before purchase. At that time, the horse was in his eighth year of a successful career as a children's show hunter. The examination findings were within normal limits, except for the following:

(1) The presence of a 1-cm dermal swelling on the left sheath, typical in appearance of equine melanoma.

(2) The presence of a several centimeter firm, subdermal mass at the left temporal region.

Neither lesion had any apparent functional significance at that time.

The potential buyers were informed that both lesions were probably melanomas and that although many melanomas will exist in status quo or very slow progression for years,[1,2] three important facts must be realized:

(1) The future biologic behavior of these melanomas was not predictable.

(2) The potential existed for local multiplication and invasion, cosmetic deformity, and life-threatening metastasis.

(3) No currently available medical or surgical therapy could be expected to be successful if treatment was attempted.[1,2]

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Fig. 1. Melanoma on sheath (1 cm).

The written report to the buyers contained the following statements:

(1) "Chaz was examined at rest and in motion in hand, on longe, and under saddle on the flat and over fences."

(2) "Chaz did not exhibit lameness, ataxia, abnormal respiratory sounds, or fatigue."

(3) "From examination of those organ systems made available to me for evaluation, I note the following:

- A firm swelling (4 cm), deep to the skin, rostroventral to the base of the left ear.
- A small melanoma (1 cm) at the left sheath."

(4) "I interpret these observations as follows:

- The swelling below the left ear base does not involve the external ear canal and has no inflammatory component or external tract; it is clinically insignificant at this time.
- Given the presence of an obvious melanoma on the sheath, it is reasonable to suspect the left facial swelling may be a melanoma as well."

(5) "While the biologic behavior of melanomas in horses can be limited to cosmetic blemish, a number of individuals will suffer extensive local tissue invasion and/or metastasis to vital structures."

(6) "It is not possible to predict the future behavior of this facial swelling."



Fig. 2. Suspected melanoma at left temple.

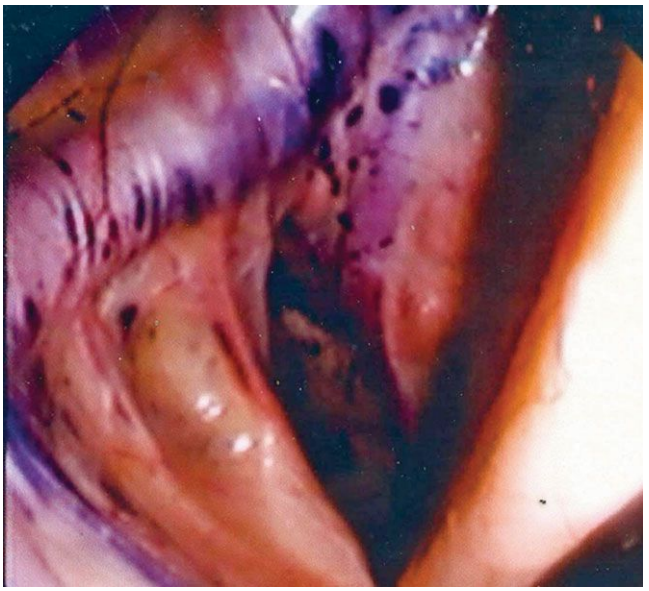


Fig. 3. Melanoma infiltration of guttural pouch.

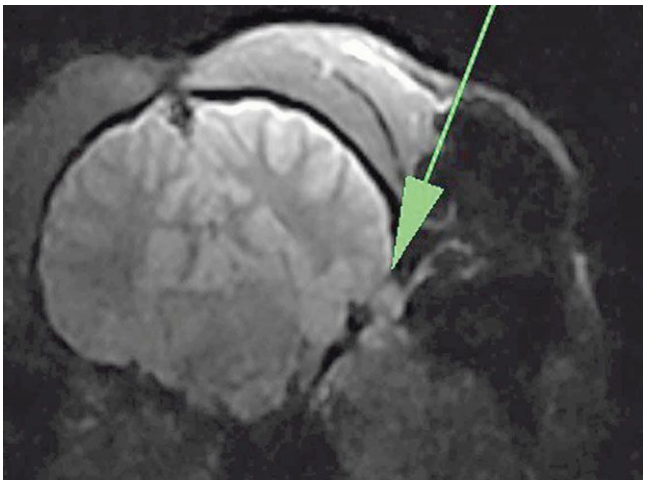


Fig. 4. Invasion of melanoma through calvarium.

The buyers proceeded with the purchase, and the horse continued his successful career uneventfully for 2 more years. At that time, he began to exhibit intermittent, brief episodes of panic behavior. These would usually occur in the quiet of the barn aisle while in cross-ties and, less often, when walking in hand or under saddle. There were never any obvious inciting causes, and he returned quickly to normal when returned to his stall.

Although the external size and profile of the temporal mass had not changed since the pre-purchase examination (Fig. 2), diagnostic imaging was performed in an attempt to determine what role it might be playing in behavior episodes. Radiography proved inconclusive, but endoscopic examination of the guttural pouches revealed extensive infiltration of melanoma (Fig. 3). Finally, magnetic resonance imaging of the skull clearly showed infiltration of the temporal mass through the calvarium (Fig. 4) into the left temporal lobe of the brain (Fig. 5) as well as into the left middle ear. The owners elected to euthanize the horse before recovery from general anesthesia.

4. Discussion

This case emphasizes the importance of timely and thorough examination, documentation, and communication at the time of the pre-purchase examination as well as recognition of the troubling and unpredictable course that equine melanoma may take in the future.

In this case, both foci of melanoma were identified, archived photographically, documented, and reported to the potential buyer, and the nature of equine melanoma was explained—all before purchase. What the reaction of this buyer would have been 2 years later without such steps having been taken is unknown. However, given his daughter's and her family's strong emotional attachment to the horse and its considerable purchase price, the stage could easily have been set for an angry and shocked client. Such a circumstance frequently is damaging to an equine practitioner's reputation as well as a risk factor for legal recourse.

5. Conclusions

Regarding the pre-purchase examination, a thorough and systematic approach, accurate and detailed recording, and understandable and relevant reporting



Fig. 5. Invasion of melanoma through temporal lobe.

and explanation to the potential buyer are essential to proper delivery of this professional service.[3–5]

Furthermore, recognize that equine melanoma is always a potential metastatic malignancy that will behave unpredictably. Avoid predicting its future or taking it for granted. Although there may be a tendency to dismiss one or a few solitary small melanomas at the time of the pre-purchase examination as of minimal or no long-term significance, especially in an otherwise excellent prospect, such an approach is both risky and medically untrue. This casual assessment can lead to serious repercussions in the future.

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Application of the Fetlock Support Shoe for Suspensory Desmopathy

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Suspensory ligament injuries are a significant cause of lameness in performance horses and racehorses. Although treatments to decrease inflammation and stimulate ligament healing are used to treat most injuries, stress on these structures is unchanged as the result of normal weight-bearing required during rest. Use of a fetlock support shoe can facilitate healing by preventing excess stress on the suspensory ligament. This technique is particularly helpful in supporting the rear fetlock during treatment of suspensory ligament desmopathy. Authors' address: Marion duPont Scott Equine Medical Center, Virginia Tech, PO Box 1938, Leesburg, VA 20177; nawhite2@vt.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The fetlock support shoe or brace (also known as the "Roberts Shoe") is depicted in books published in the early 1900s for treatment of tendon or suspensory rupture (Fig. 1).[1,2] In more recent texts, the various modifications of the support shoe are described. Although there are numerous descriptions in textbooks, there are few reports in the literature describing success or failure with different types of injuries.[3,4] A support shoe used for support of tendon lacerations was reported to be successful in restoring use in 60% of horses.[5]

Dorsal support of the fetlock can be provided by splints such as the Kimzey Splint[a] or in a cast, however, these are normally used during the initial phase of support but do not allow the partial loading of the suspensory ligament needed for healing over the long term. Attempts to attach a splint on the dorsal aspect of the limb have been used by the authors, but these splints do not allow normal flexion of the fetlock, and tension applied by the bandage behind the metacarpus/metatarsus and fetlock can result in pressure sores (Fig. 2).

Treatment for proximal rear suspensory ligament desmopathy (injury at the origin) is reported to be less successful in horses with straight rear limbs and excessive dorsiflexion of the fetlock, causing constant stress on the suspensory ligament (Fig. 3).[6]

To facilitate healing in cases of suspensory desmopathy, a fetlock support shoe was used by the authors to limit fetlock dorsiflexion and relieve stress on the suspensory ligament(s) during healing.

2. Materials and Methods

Chronically lame horses with rear-limb suspensory ligament lesions at the origin or in the suspensory branches (diagnosed by ultrasonography) were treated with a fetlock support shoe. All the horses had rest and surgical or regenerative medical treatment of the hypoechoic lesions. Selected horses with excessive fetlock dorsiflexion associated with proximal suspensory desmopathy were fitted with a

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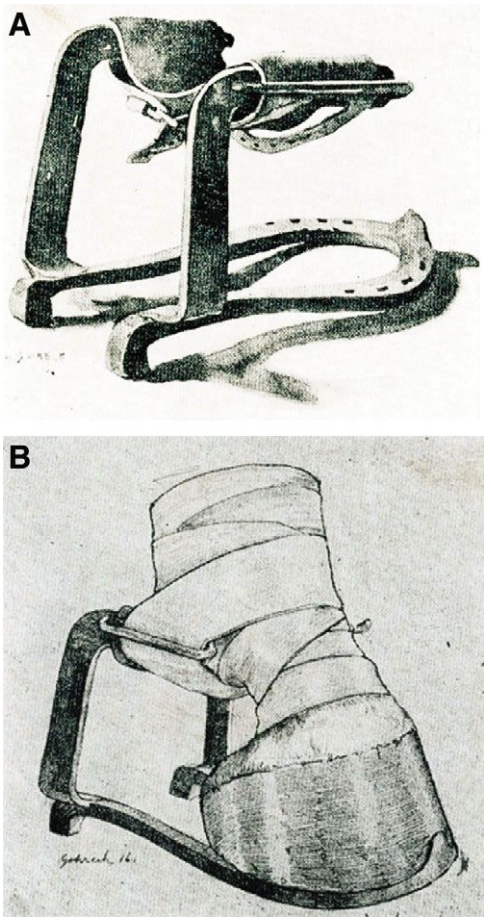


Fig. 1. Horseshoe with extended heels and a support brace (A) is designed to have a leather strap between the bars to support the fetlock as it descends in extension (B).[1]

fetlock support horseshoe for 1 to 2 months during a recommended period of stall rest.

The fetlock support shoe, which requires experience and skill for proper construction, was designed as a bar shoe with toe clips, an extended heel (3–5 cm), and two hex nuts welded to the heel extension. Two support rods were designed and measured with a length to reach just past the level of the fetlock for each horse. The rods were welded together near the shoe and inserted into the hex nuts. A setscrew in the side of the hex nut was used to keep rods from moving. This allows the rods to be removed for management of the bandage, which is needed for padding (Fig. 4). A support wrap with several layers of sheet cotton was applied from the coronary band to the proximal metacarpus/metatarsus. An elastic adhesive bandage[b] was wrapped from side to side around the support rods and positioned tightly to support the fetlock when the horse bears weight on the limb. An alternative is the use of rubber tubing around the supports to provide more flexibility during weight-bearing. The brace can be left open or covered with a light bandage placed to cover the support rods, which allow the fetlock to flex during walking without any pressure on the limb. Adequate padding on the limb is required to prevent pressure sores on the back of the fetlock.

3. Results

Seven horses with excessive rear fetlock dorsiflexion and chronic suspensory ligament desmopathy were treated with a fetlock support shoe and rest. Three of six horses with proximal suspensory ligament desmopathy were treated with desmoplasty/fas-



Fig. 2. Dorsal splint (A) was successful in increasing the pastern angle during full weight-bearing but resulted in pressure sores on the back of the fetlock (B).



Fig. 3. Horse with a bilateral rear proximal suspensory ligament desmopathy. The prognosis for resolution of the desmopathy in horses with straight leg conformation and excessive extension of the fetlock is poor.[5]

ciotomy, two with platelet rich plasma (PRP) injection, and one with desmoplasty/fasciotomy, PRP, and stem cells.[7] One horse with chronic suspensory branch desmopathy had a fetlock support shoe applied and had no treatment other than rest (Fig. 5). All horses had absolute stall rest while wearing the support shoe. All seven horses had improved ultrasonographic evidence of healing and subjectively had improved conformation (less abnormal fetlock dorsiflexion) after wearing the shoe for approximately 8 weeks. All hypoechoic regions in the suspensory ligaments had increased echogenicity 2 months after the treatments and rest. The horse with chronic suspensory branch injury was scheduled to have PRP injection in the core lesions, but after 2 months of support, intralesional treatment was not required. In the six horses for which short-term follow-up was available, lameness was resolved in six horses at the time of the last recheck either by examination or communication with the owner. Long-term follow-up included resolution of the lameness in four horses: one horse used for very light riding, one for dressage, one sound in turn-out and about to be returned to work, and one retired (sound in turn-out). Three horses were lost to long-term follow-up.

4. Discussion

Recommended treatments for suspensory desmopathy include rest, support bandages, anti-inflammatory therapy, and a slow return to controlled exercise. Stem cells and PRP injection also anecdotally help to stimulate healing. Although resolution of lameness and return to exercise after lateral plantar neurectomy with fasciotomy or ultrasound-guided suspensory desmoplasty is approximately 70% to 80%, horses with "dropped fetlocks," either at rest or at the walk, are unlikely to return to any type of exercise under saddle because of failure of healing or re-injury.[6–8]

Horses with an abnormal rear-limb conformation (straight angle in the hock with excess dorsiflexion

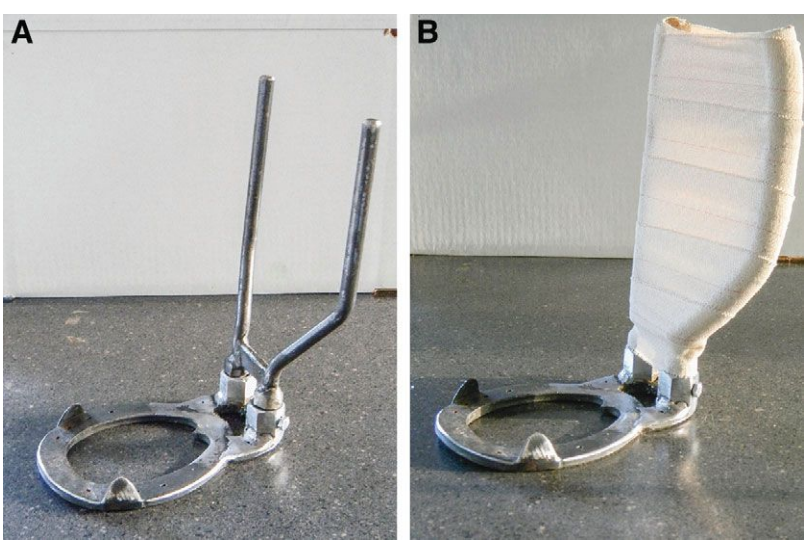


Fig. 4. Shoe is designed with hex nuts attached to the extended heel and locking screws inserted through the hex nuts to allow removal of the support rods for adjustment and bandaging (A). Elastic adhesive bandage is used to create a flexible sling to reduce fetlock extension (B).



Fig. 5. Arabian mare presented for a chronic right rear medial suspensory branch desmitis, which had not responded to PRP injection and subsequent rest for 9 months. The fetlock support shoe was applied for 2 months: rear view (A), lateral view (B). After 2 months, increased echogenicity was present on ultrasonographic examination and the previously abnormal fetlock extension (dropped fetlock) was improved.

of the fetlock, as shown in Fig. 3) appear to place excess tension on the suspensory ligament, which increases the risk for injury or resistance to healing. The authors' experience suggests that injury to the proximal suspensory ligament can lead to this altered rear-limb conformation, but it has not been determined if one problem precedes the other. In these cases, use of the fetlock support shoe has helped to resolve the desmopathy and subjectively improved rear-limb conformation in seven horses during the treatment period. Because all except one horse had multiple treatments, it is possible that the other treatments may have been successful without the fetlock support. However, the clinical response in these cases suggests that fetlock support is an aid in the successful treatment of rear-limb suspensory desmopathy associated with abnormal fetlock conformation. Critical assessment of this technique is needed to determine if it is successful in returning horses to their previous level of work.[6,7]

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[a]Kimzey Leg Saver Splint, Kimzey Metal Products, 164 Kentucky Avenue, Woodland, CA 95695.

[b]Elastikon, Johnson and Johnson, One Johnson & Johnson Plaza, New Brunswick, NJ 08933.

How to Perform a Thorough Equine Eye Exam in the Field

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1. Introduction

Complete ophthalmic examinations are imperative for any horse with an ocular complaint (squinting, tearing, cloudy eye, vision loss, etc). It has been reported that more than 50% of horses have observable ophthalmic lesions, whereas 5% to 10% have potential vision-threatening ophthalmic abnormalities[1]; therefore knowledge and ability to perform ocular examinations is a very important veterinary skill. Fortunately, multiple equine ophthalmology resources are available with pertinent information and color images to aid veterinarians.[2–4] Routine performance of ophthalmic examinations will help to maintain evaluation skills, improve assessment of subtle lesions, and allow one to see the many variations of normal. Last, it is important to teach owners that horse eye problems can deteriorate very quickly; therefore early veterinary evaluation is key to maintain or restore ocular health and vision potential.

2. Materials and Methods

To investigate an ocular problem, it is important to first gather historical information, asking when ocular signs were initially noted, whether they have changed over time, whether empiric treatment has been attempted, and, if so, what response was observed, while also inquiring about possible systemic issues and perform a complete physical exam if indicated. Even if ophthalmic signs are not present, because many systemic diseases have ocular manifestations, a complete eye examination may provide valuable information in any ill animal.

Ocular examinations should optimally be performed in a quiet, protected area that can be darkened to minimize glare and maximize lesion assessment. In the field setting, a barn or lean-to–type shed is optimal, though a large sheet or blanket can be extended over the examiner and animals' heads to achieve a more suitable dark setting if no enclosed structure is available. Cooperative patients can be examined without sedation, but uncooperative or painful horses may require mild sedation and/or a periocular nerve block. The most common nerve block performed is the auriculopalpebral nerve block with 1 to 2 milliliters of lidocaine or another local anesthetic injected subcutaneously over the zygomatic arch, where the palpebral branch of the auriculopalpebral nerve courses, with the use of a 25-gauge five-eighths–inch needle. This blocks motor function to the orbicularis oculi muscle and facilitates ocular examination while minimizing the

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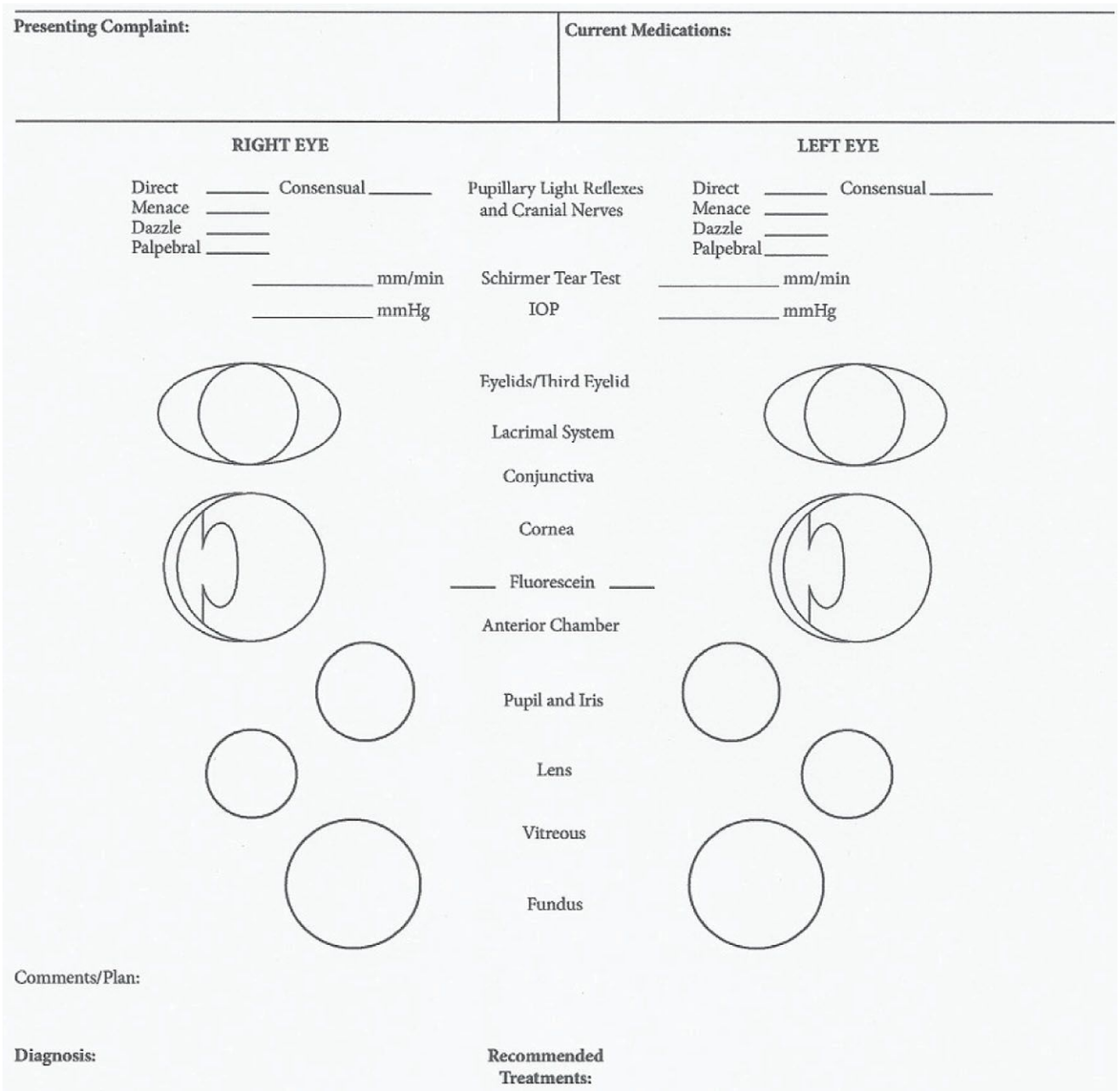


Fig. 1. Eye exam sheet.

risk of compromising a fragile eye during forced eyelid opening (eg, if a deep corneal ulcer or globe penetrating injury may be present). Other periocular nerve blocks may be used for additional ophthalmic procedures or periocular surgery.[4]

Materials needed for equine eye exams can be kept in a tack box or tool box for convenience and ease of transport. A preprinted ophthalmic examination sheet (Fig. 1) is helpful to guide the examination, to serve as a checklist, and to enable drawing of pictures to document ophthalmic lesions and allow for evaluating progress over time. Instruments needed for ocular examination include a bright focal light source (eg, Finoff transilluminator), a condensing lens (eg, 20 diopter), and a direct ophthalmoscope. Additional supplies include fluorescein stain for corneal assessment and nasolacrimal patency evaluation, 1% tropicamide for pupil dilation, topical ophthalmic anesthetic (eg, 0.5% proparacaine or tetracaine) for ocular diagnostic procedures, culturettes for infectious disease testing, cytology equipment for sample acquisition, and eyewash or saline for ocular flushing. A tonometer is advantageous to assess intraocular pressure, with Tono-Pen and TonoVet instruments both easily used in horses. Mean intraocular pressure values for horses are

slightly higher than small-animal patients and have been found to generally range between 15 and 30 mm Hg.[5,6] If a tonometer is not available, digital tonometry can be performed by placing both index fingers over the eyelid when covering the eye, and gentle, alternating digital pressure can be used to palpate for normal mild globe indentability. Alternatively, a soft, blunt device (eg, a cotton-tipped applicator) can be used to attempt to gently indent the cornea after application of topical anesthesia. These methods give an approximation of eye pressure through subjective indentation and may help to identify soft or firm eyes consistent with uveitis or glaucoma, respectively, if a tonometer is not available.

Though tear film deficiency is not commonly reported in horses, a Schirmer tear test may be performed in animals suspected of having keratoconjunctivitis sicca, such as those with a dull corneal surface, unexplained corneal pathology, or those with facial nerve dysfunction. The tear test strip is folded at the notch while still in its plastic packaging and is then gently placed over the lower eyelid margin so that the folded tip sits within the conjunctival fornix. After 1 minute, the strip is removed and the reading is immediately recorded where moisture has traversed the test strip. Normal tear values in horses are highly variable, with results in one study never below 10 mm/min and sometimes exceeding 35 mm/min.[7]

When beginning an eye exam general facial and ocular symmetry should first be assessed from a distance in addition to looking for any signs of blepharospasm or ocular discharge. Cranial nerve evaluation can then be performed with a palpebral reflex (evaluates trigeminal and facial nerves), menace response (evaluates optic and facial nerves), and pupil light reflex testing (evaluates optic and oculomotor nerves), although pupil light reflexes are commonly slow and incomplete in horses. Animals that do not menace because of impediments in the ocular media (eg, profound corneal edema, hyphema, cataract, etc) can have a dazzle reflex assessed by rapidly shining a very bright light at the eye and observing for a blink response or head jerk to indicate retinal and optic nerve functioning. Ocular motility can also be consciously evaluated because both eyes should be able to move in all directions and to do so concurrently (evaluates oculomotor, trochlear, and abducens nerves). Periocular palpation can be used to assess around the orbital rim. Globe retropulsion allows for further orbital examination by attempting to caudally displace the globe through digital pressure on a partially closed upper eyelid. Retropulsion also allows easier evaluation of the third eyelid because of its passive elevation with posterior globe movement.

Retroillumination is a technique in which light is shone toward the eyes at arm's length (~2–3 feet from the cornea) and the fundic reflection is visible through the pupil. It allows for easy assessment of pupil size and symmetry and draws immediate attention to any impediments in the reflection (eg, focal corneal scar, cataract, etc). Detailed examination of the ocular structures should proceed in a systemic manner with both direct and transillumination, in which light is directed with the line of gaze, and at alternating perpendicular angles to the line of gaze so that the eye is truly assessed in all three dimensions.

The individual anatomical structures that should be consciously examined during a complete ocular evaluation include the eyelids, third eyelid, conjunctiva, sclera, cornea, anterior chamber, iris, lens, vitreous, and fundus. Anterior segment examination requires only a light source and, ideally, a means for magnification. Inexpensive head loops or the otoscope head on a Welch Allyn examination set serve as good magnification tool options. Complete lens and posterior segment examination should be performed after pharmacologic dilation with 1% tropicamide (acts within 20–30 minutes), though adequate cursory evaluation is commonly possible in a darkened environment with the rheostat on the light source dimmed down slightly to minimize the pupil light reflex. In addition to fundus evaluation, the direct ophthalmoscope can also be used to assess for aqueous flare, an indicator of anterior segment inflammation, by selecting the smallest focal circular beam of light and holding the instrument 5 to 10 mm in front of the cornea while viewing from the side (45–90° angle) in a very dark exam setting. Normal eyes will show the light beam hitting the cornea, a void in the anterior chamber, then light hitting the anterior lens capsule and coursing through the lens to end at the posterior lens capsule. An eye for which the light beam continues through the anterior chamber (like a head light beam in the fog) to connect the cornea and lens has aqueous flare, which is common with intraocular inflammation or uveitis.

Fundic examination can be performed with the direct ophthalmoscope focused at 0 to −2 diopters (red numbers) to give a highly magnified direct upright image. This method is good for detailed optic disc or fundic lesion assessment but is difficult for general examination, given the very limited field of view (<2% of the fundus). The Welch Allyn Pan-Optic attachment is also a direct ophthalmoscope that gives a 5-times-larger field of view with less magnification. Indirect ophthalmoscopy with the use of a light source (eg, Finoff transilluminator) and handheld condensing lens (eg, 20 diopter) gives the greatest field of view to more easily assess the entirety of the fundus and is the author's preferred initial assessment tool. The view with this technique is inverted and reversed; therefore observed anatomy is effectively 180° off.

Fluorescein stain should be performed on every horse with an ocular complaint to evaluate corneal health and nasolacrimal duct patency. Stain can be applied to the ocular surface either by direct

application to the conjunctiva or by spraying fluorescein liquid (0.5 mL of eyewash added to fluorescein strip tip in a syringe) onto the corneal surface through a needle hub (needle should be gently broken off) held a few millimeters from the eye, with care taken not to contact the globe with the hub of the needle. Eyewash rinse is rarely needed because ample tearing in horses naturally flushes excessive fluorescein from the eye. Intact corneal epithelium will not retain fluorescein dye, whereas areas of corneal epithelial loss stain green as the result of fluorescein adhering to the hydrophilic exposed corneal stroma and are diagnostic for corneal ulceration. Very deep corneal ulcers that extend down to the innermost layer of the cornea (Descemet membrane) may have a clear, dark, nonstaining center as the result of complete corneal stromal absence and are termed descemetoceles. Corneal lacerations or potentially ruptured corneal ulcers can also be evaluated for leakage after concentrated fluorescein stain application (Seidel testing) because escaping aqueous humor will dilute concentrated fluorescein in the tear film and result in a darker stream flowing down from the rupture site.

After ocular fluorescein application, the nares should be examined for stain appearance (Jones testing). This is especially helpful in patients with epiphora or mucopurulent ocular discharge and no obvious ophthalmic cause to investigate nasolacrimal system patency. If fluorescein does not passively appear within the nostrils within a few minutes, active nasolacrimal duct flushing can be performed as a diagnostic test or treatment to remove occluding debris. Topical anesthetic should be applied to the ocular surface and the lacrimal puncta located just inside the inner eyelid margin dorsally and ventrally approximately 5 millimeters from the medial canthus. A lacrimal cannula or soft catheter (eg, 3.5F tomcat catheter or 20-gauge intravenous catheter with stylet removed) attached to a syringe with eyewash can be gently advanced into one lacrimal punctum, and flushing is then performed. Fluid should be seen exiting the opposite lacrimal punctum, and digital pressure can then be applied to both the upper and lower eyelids at the level of both puncta to allow flushing down the nasolacrimal duct and out the nasal punctum. If material emerges from the puncta, it can be collected for culture and cytology. If anterograde flushing is unsuccessful or not possible because of patient noncompliance, retrograde flushing can be performed by introducing a longer soft catheter into the nasal opening of the nasolacrimal duct. After topical nasal punctum anesthesia (eg, lidocaine liquid or gel) is given, a catheter is introduced and gentle digital pressure is applied over the opening to prevent backflow while flushing with eyewash and monitoring for flow at the level of the eye.

3. Results

A systematic ophthalmic examination approach and the use of appropriate diagnostic equipment and techniques will afford optimal results and prevent missed lesions or lacking information. Ophthalmic examinations performed in an appropriate environment will be more successful than if attempted out in the elements with possible wind, glare, and corneal reflection aberrations, to name a few problems. Complete findings will help allow accurate diagnosis and appropriate therapy, allow for serial monitoring of ophthalmic conditions, and will result in identification of lesions early enough to avoid vision-threatening complications. Thorough exams will also display the value of services to clients so that they more readily call when ophthalmic problems are noted.

4. Discussion

Eyes are special structures that require a few extra considerations and additional equipment for clinical evaluation. Even in barn and field settings, complete eye exams can be performed with the right knowledge, equipment, and diagnostic tests. A systematic approach will help to avoid missed steps and facilitate arriving at the correct diagnosis for optimal management. Fluorescein staining, retroillumination, and aqueous fare assessment are just a few examples of critical steps that should always be implemented during equine ophthalmic examinations. Abbreviated ocular evaluations are also warranted during general physical examinations and prepurchase examinations and may be of particular benefit for systemically ill patients.

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How to Repair Eyelid Lacerations

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1. Introduction

Eyelid lacerations are common in horses and should be regarded as surgical emergencies to prevent undesirable tissue devitalization, infection, scarring, corneal desiccation, and corneal ulceration. Lacerations usually can be repaired in the standing, heavily sedated horse. General anesthesia may be required when there is extensive trauma or with an unruly horse. Even when horses are not evaluated immediately after the trauma and infection may have begun, repair should always be attempted immediately and with minimal debridement to prevent further tissue damage. Although a complete ophthalmic examination should be performed in all horses with eyelid lacerations, ocular examination usually reveals a normal globe; however, it is not unusual to have two lacerations, with one being much smaller than the other. Lacerations usually are caused by nails and hooks protruding from boards in the barn or stall, from fence wire or tree branches, or from sharp metal protuberances in trailers. Unfortunately, even with extensive study of the horse's surroundings, the cause of the laceration is rarely determined.

An understanding of eyelid anatomy and physiology is imperative for proper surgical repair because of the importance of perfect alignment of the eyelid margin and the close proximity of sutures to the cornea. A normal eyelid margin is crucial for globe health. The eyelid margin prevents hair from contacting the cornea and allows the lids to have perfect contact with the corneal surface. This normal eyelid contact during blinking removes precorneal debris and spreads the tear film over the cornea to prevent desiccation. Under normal conditions, there are no hairs or cilia on the eyelid margin. Meibomian gland openings form a row of tiny spots along the lid margins. These glandular openings serve as an important landmark for the figure-eight suture.[1] The haired eyelid skin is very thin, with no redundancy. The palpebral conjunctiva lines the bulbar side of the eyelid and is also very thin, approximately 10 cell layers thick. The tissue between the skin and the palpebral conjunctiva from superficial to deep contains the orbicularis oculi muscle, the stroma (which contains the levator palpebral superioris muscle in the upper lid), and thin, fibrous tissue referred to as the tarsus. For the purpose of this report the layers of the eyelid will be referred to as the skin, stroma, and conjunctiva. Whereas the muscles that open and close the eyelids are important physiologically, they are not easily visible when the eyelid is lacerated and do not require special attention during the repair.

Knowledge of eyelid innervation is also important for laceration repair. A branch of the facial nerve (auriculopalpebral nerve) controls the orbicularis oculi muscle, which closes the eyelids. This nerve

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must be blocked to assist in ocular examination and eyelid repair. Local anesthesia of the auriculopalpebral branch of the facial nerve only suppresses motor function to the eyelids and does not block sensation. Sensation to the eyelids and corneal surface is provided by branches of the trigeminal nerve, which must also be anesthetized to facilitate laceration closure.

Eyelid laceration repair is relatively easy as long as several key points are followed. The purpose of this report is to provide step-by-step instructions on eyelid laceration repair that will leave the horse with anatomically and physiologically normal eyelids.

2. Materials and Methods

Initial Ocular Examination

The first part of the examination, including external examination and reflexes, should be performed before sedation and nerve blocks are administered. An external examination of the head is necessary for any horse that has had trauma. Symmetry of the orbits and sinuses and globe position should be evaluated. Palpation of the orbital rim may reveal fractures. Eyelid swelling is common with eyelid lacerations, but more diffuse severe swelling may indicate blunt trauma in addition to the sharp trauma that caused the laceration.

The menace response and palpebral reflexes are evaluated next. The menace response involves making a sudden hand motion near the eye being tested and is confirmed with closure of the eyelid or shying of the head away from the motioning hand. It is usually obvious in adult horses, but foals may not have a fully developed menace response until 9 days of age. Some foals may have a partial response a day before showing a complete response, or they may have an asymmetrical response initially.[2]

Sedation and Local Anesthesia

After the initial ocular exam and physical exam, sedation should be used to facilitate the remainder of the ocular examination. Sedation is imperative in almost all horses with a painful eye. Detomidine (10–20 μg/kg IV) is very effective because it provides analgesia as well as sedation. Xylazine (0.5–0.75 mg/kg IV) is effective in horses that only require sedation. If analgesia becomes necessary, butorphanol (0.01–0.02 mg/kg IV) can be administered in conjunction with the xylazine. These sedatives generally act for the duration necessary to do a complete ophthalmic exam. Additional sedation probably will be necessary if eyelid repair is performed after the examination.

The auriculopalpebral nerve block is generally the only injectable local anesthesia needed to assist with the ocular examination. Akinesia of the auriculopalpebral nerve prevents active closure of the palpebral fissure, thus facilitating ocular examination. The nerve block is performed by injection of 1 to 2 mL of 2% lidocaine subcutaneously near the branch of the auriculopalpebral nerve. The nerve branch is located by running the index finger over the anterior aspect of the zygomatic arch. The nerve can be strummed where it runs perpendicularly across the zygomatic process of the temporal bone. A 25-gauge needle is inserted superficial to and parallel to the nerve. The syringe is attached, and lidocaine is injected. Gently rubbing the area after removing the needle will facilitate diffusion of the lidocaine into the tissue. Avoid the artery and vein that run with the nerve. If the block is successful, the lid movement and strength will be impaired.



Fig. 1. Examination for eyelid laceration. Special attention is paid to eyelid margins, with evaluation for multiple lacerations and the presence of foreign bodies.

Complete Ocular Examination

In addition to sedation and the auriculopalpebral nerve block, a good light source, darkened examination area, and magnification source are necessary for complete ophthalmic examination. A direct ophthalmoscope and Finoff transilluminator are excellent rechargeable light sources. Bright penlights can also be used. The direct and consensual pupillary light reflexes are evaluated with the use of the strong focal light source. When a horse is seen for an eyelid laceration, special attention is paid to the eyelid margins, with evaluation for multiple lacerations and the presence of foreign bodies (Fig. 1). If a foreign body is present, Graefe fixation forceps or thumb forceps may be used to grasp the object after administration of topical anesthetic. The normal conjunctiva is glistening, very thin, translucent, and pale pink. When the eyelid is lacerated, the conjunctiva often becomes chemotic, hyperemic, or hemorrhagic. The conjunctiva should also be evaluated for foreign bodies or extensive trauma.

Fluorescein staining should be performed on all eyes that have received an eyelid laceration. Whereas large ulcers are easily visualized with white light, small corneal ulcers are more easily visualized with

the use of magnification and a cobalt blue filter. Depth of any ulcers or corneal lacerations should be determined. If a full-thickness corneal laceration is present, fibrin, blood, or iris usually comes from the wound. Some cases with evidence of severe trauma will also have orbital fractures.

Generally if the cornea does not have signs of trauma, the anterior chamber, lens and posterior segment will be spared as well. However, at least a brief examination of the anterior chamber, iris, and lens should be performed with the use of a focal light source. The anterior chamber is evaluated for aqueous flare, blood, fibrin, and hypopyon. Occasionally, miosis will be present with no other anterior chamber signs; this is still indicative of mild uveitis. With the great majority of eyelid lacerations, the eye itself is completely normal because the eyelids have successfully protected the globe and absorbed the brunt of the trauma.

Preparation for Surgery

After sedation and akinesia of the auriculopalpebral nerve, the next step in preparing for laceration repair is the surgical preparation and analgesia of the eyelids and cornea by anesthetizing the trigeminal nerve. The affected area should be prepped with dilute betadine solution and saline. Betadine scrub and alcohol should not be used because they are toxic to the corneal epithelium and can cause corneal ulceration. After surgical preparation, the palpebral branch of the trigeminal nerve is blocked.[3,4] The easiest way to block the palpebral branch of the trigeminal nerve is to perform a line block with 2% lidocaine and a 25-gauge needle in the skin just peripheral to the affected part of the eyelid. This ensures that anesthesia of the lacerated portion of the eyelid that will be sutured. The bulbar conjunctiva and cornea should also be anesthetized with the use of 0.5% proparacaine hydrochloride ophthalmic solution to diminish responses if the globe is accidently contacted with instruments or sutures during the repair. This is best applied by spraying the solution from a tuberculin syringe in which the tip of a 25-gauge needle has been broken off. Always keep this syringe at least 6 inches from the cornea because the broken needle tip is still sharp.

In most cases, the eyelid is only lacerated, and no tissue has been ripped away. Severe swelling and congestion in the lacerated tissue can alter its appearance and give the illusion that tissue is missing, but the lid should be closed as a simple apposition until one is sure that tissue is missing. Unfortunately, the horse has very little extra eyelid skin, which makes it difficult to perform blepharoplastic procedures. Keeping this in mind, preparation of the surgical bed should be minimal. If there is question as to the viability of the lacerated tissue, it can be scarified with a dry, sterile 4×4 gauze pad or a scalpel blade until bleeding is observed. Avoid excising tissue that has any potential for viability. Because the eyelid is so well vascularized, the tissue may recannalate. The edges of the laceration are often jagged. This should be pieced together with suture rather than being excised to make a clean edge. An eyelid pedicle created by a laceration should never be simply excised because exposure keratitis and ulceration probably will occur. If the nasal canthus is involved, the nasolacrimal system should be evaluated for patency. If the nasolacrimal system has been damaged, general anesthesia may be required for the repair.

Special Surgical Considerations

The eyelids are extremely vascular, and hemorrhage from incisions may be profuse. However, cautery should never be used because excessive scarring can lead to conformational changes of the eyelid. Suture choices that would be considered contraindicated at other locations are routinely used for the eyelids because of the copious blood supply. For example, polyglactin 910[a] is the preferred suture for eyelid lacerations. Characteristics of polyglactin 910 that make it favorable for eyelid margin apposition include excellent knot security and handling and the fact that it becomes soft when wet. Additionally, because they are absorbable, polyglactin 910 sutures do not need to be removed; this is a desirable feature when removing very small sutures in the equine eyelid. Some texts mention burying the suture knots or suturing the conjunctiva. Although it is important to close gaping distances between the lacerated conjunctival edges, one must avoid closing the conjunctiva in a way that the suture might contact the cornea and cause ulceration. The conjunctiva heals very quickly, even when it is not in perfect apposition.

Simple Two-Layer Closure

The instruments needed for eyelid laceration repair include Bishop-Harmon forceps or other fine-toothed forceps, Derf or Castroviejo needle holders, and Stevens tenotomy scissors. The preferred suture is 5–0 polyglactin 910 with a spatula or cutting needle. First, the stroma, which is the tissue between the conjunctiva and skin that contains the tarsus and orbicularis oculi muscle, is apposed with simple continuous or simple interrupted sutures such that the deep aspect of the suture does not protrude through the conjunctiva and contact the cornea. The knot of this suture must be oriented away from the conjunctiva and toward the skin. This step can be skipped with very small lacerations and may even hinder perfect apposition in these situations. Next, the eyelid margin is apposed with a figure-eight suture (Fig. 2A).[1] Bites should be small (approximately 2 mm), and bites on one side of the laceration should mirror those on the opposite side. If the eyelid margin is not squarely apposed, the figure-eight suture should be redone. Suture tags are left long and pulled away from the eye by incorporating them into the simple interrupted su-

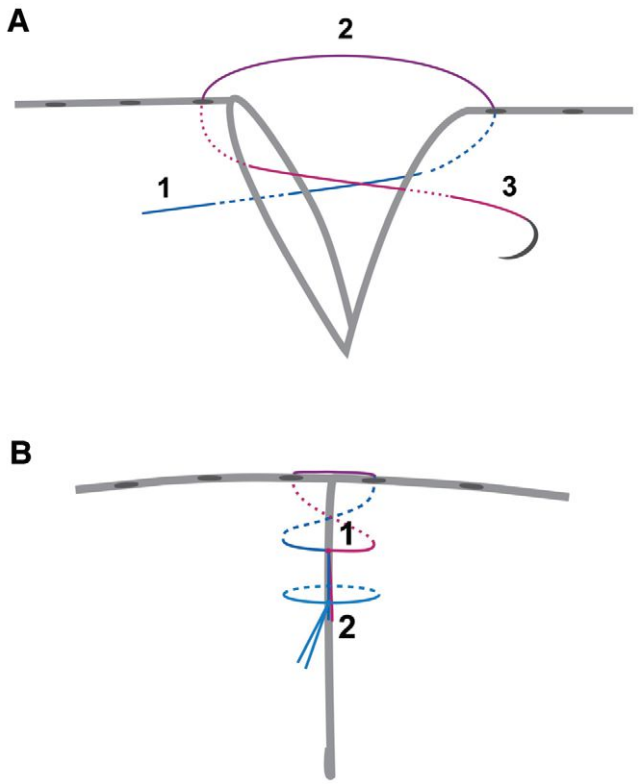


Fig. 2. A, Placement of the figure-eight suture pattern. The needle enters the skin just behind the eyelid margin (1, blue suture), exits the stroma of the eyelid, and enters the stroma on the opposite side. The suture then exits on the eyelid margin at the line of meibomian gland openings and is passed externally (2, purple) to enter the meibomian gland opening on the opposite side. For the third pass, the suture is passed out of the eyelid stroma, across to the stroma on the opposite side, and exits the skin behind the eyelid margin (3, magenta). B, Complete figure-eight suture is shown. Tags from the figure-eight suture (1) are laid across the knot from the first simple interrupted suture knot (2) to prevent contact with the cornea.

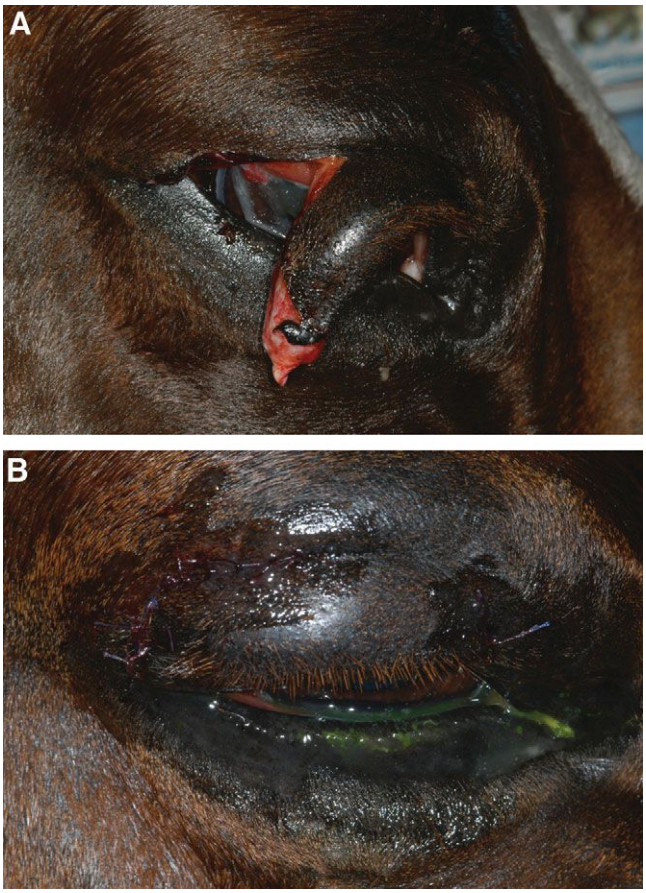


Fig. 3. A, This eyelid laceration extended from the lateral canthus to the midpoint of the upper eyelid. Swelling was severe. B, The same horse immediately after repair. Note the suture located medial to the main repair. This horse had a smaller laceration not evident in the picture of the laceration.

ture that is used to appose the skin distal to the eyelid margin (Fig. 2B). The tails of the figure-eight suture must be situated on top of one square knot and below at least one additional square knot. If the lacerated margin affects the canthus, a horizontal mattress suture or simple interrupted suture may be placed in the margin, still incorporating the tags into the first simple interrupted skin suture to avoid corneal contact by the knot. The remainder of the lacerated tissue is apposed with simple interrupted sutures. The pieces often must be "quilted" together because the edges may be jagged or narrow (Figs. 3 and 4). Excision of any skin should be avoided if possible.

Complicated Lacerations

When a portion of a lid or an entire upper or lower lid has been torn away, there are few options. If the lower eyelid is affected rather than the upper eyelid, the cornea is more likely to remain healthy because the upper eyelid more actively protects the eye. Even portions of the temporal or nasal upper eyelid can be absent and the cornea remains healthy. After the laceration edge heals, removing skin hairs that contact the cornea may be beneficial if the horse is painful or has keratitis (Fig. 5). Cryotherapy of offending hairs can be attempted but may not be effective. Electrolysis may be necessary. Although it may be tempting to perform a modified Hotz-Celsus procedure to remove a crescent of tissue to roll the haired skin away from the cornea, this generally is not beneficial and allows for more corneal exposure. Blepharoplastic techniques such as H-plasty can be attempted, but contracture of the surgery site tends to occur.

If the nasal canthus is involved in the laceration, the nasolacrimal duct may be damaged. In these cases, general anesthesia probably will be necessary to repair the nasolacrimal duct or to place a stent.[4] Damage to the nictitating membrane can be repaired with the use of a simple interrupted pattern of 6–0 polyglactin 910, once again, taking care to avoid suture contact with the cornea.

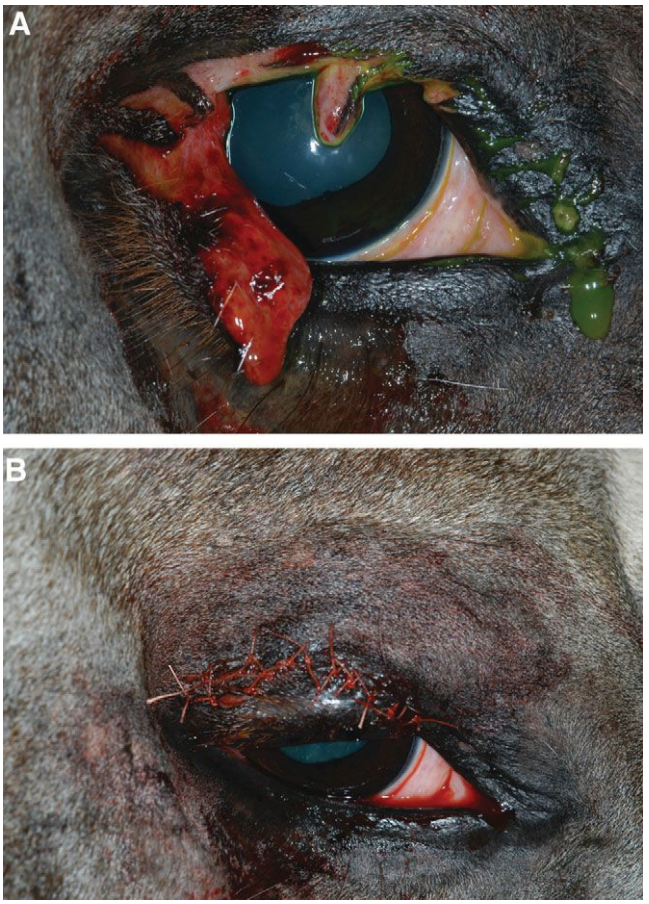


Fig. 4. A, This horse had a laceration of the upper eyelid with multiple tears in the skin. No skin was excised from this eyelid before repair. Pedicles of skin were incorporated into the sutures. B, The same horse immediately after repair. Repair was successful in restoring eyelid integrity and function. Swelling was resolving the following day.

If the surgical repair dehisces within a day or two of repair, it can often be repaired again with freshening of the laceration edges. If the dehiscence is not treated immediately, the eyelid will be disfigured and a wedge resection may be necessary (Fig. 6). Alternatively, small areas of dehiscence may heal with granulation tissue.



Fig. 6. This horse's eyelid was lacerated and repaired 2 weeks before evaluation. Repair had been attempted but appeared to have dehisced. A wedge resection to remove the tissue from the last visible meibomian gland in the lower eyelid to the lateral canthus with a two-layer figure-eight closure was recommended, but the owners declined. No keratitis is present, but an immature cataract and evidence of previous posterior synechia is seen.

Postoperative Care

Postoperative care for eyelid lacerations often includes a protective hood with a hard cup[b] over the eye to minimize self-trauma. Additionally, a topical broad-spectrum ophthalmic antibiotic ointment is used to lubricate the cornea and prevent secondary infection of the repaired laceration. Most commonly, triple antibiotic ophthalmic ointment (neomycin, polymyxin, and bacitracin) is applied to the laceration site and the cornea. Flunixin meglumine, or phenylbutazone should be administered every 12 hours for 2 to 5 days, depending on the degree of swelling and severity of the laceration.



Fig. 5. This horse lost the temporal aspect of its upper eyelid. Trichiasis is present, but the cornea appears normal.

If the horse received tetanus toxoid 6 months or longer before injury, it should be revaccinated. Treatment with systemic trimethoprim sulfamethoxazole is indicated when infection is present.

If any other ocular trauma is found, it should be treated appropriately. A superficial ulcer should be treated with triple antibiotic ophthalmic ointment every 6 to 8 hours, and ophthalmic atropine every 12 to 24 hours and the already prescribed nonsteroidal anti-inflammatory drug (NSAID) should be given. Mild anterior uveitis probably will respond to the systemic NSAID and ophthalmic atropine given every 12 to 24 hours. For treatment of more severe ocular diseases such as corneal lacerations, ophthalmic texts should be consulted.

3. Results

With just a few key techniques as described above, the vast majority of eyelid lacerations can be easily and successfully repaired. There are no retrospective studies discussing equine eyelid laceration repair in the peer-reviewed literature; however, years of experience have shown that use of the described technique produces excellent results. The figure-eight suture has been published in multiple texts, and procedures similar to those described here have been published.[1,5–7] With the use of the described technique, complications are rare. Occasionally, the tip of the lacerated eyelid margin will become necrotic. If the area is located at the lateral canthus, it may heal with granulation tissue without further complication. Alternatively, the necrotic tissue may need to be excised and the figure-eight suture replaced. Several horses have been seen that were not repaired in the described manner, or veterinary care had not been sought. Some were seen in which conjunctival sutures had been "buried," with the knot toward the cornea rather than toward the skin side of the eyelid. In these cases, the cornea was ulcerated, and the horses were very painful. In some cases, it was possible to remove only the offending sutures, but in others, the entire primary surgical repair had to be removed to access the offending sutures.

4. Discussion

Eyelid laceration repair according to this described technique is effective for several reasons. First, apposition of the eyelid margin with the figure-eight suture ensures that there will be no defects in the eyelid margin that would allow for corneal trauma or desiccation. Second, the palpebral side of the eyelid is closely apposed with the placement of more superficial stromal sutures rather than conjunctival sutures, which allows for quick healing by migration of conjunctival epithelial cells without the danger of corneal ulceration from conjunctival sutures. Last, the use of a small needle and fine sutures allows for precise suture placement without excessive damage to the tissue. The only pitfalls to this procedure are the need for good lighting, getting accustomed to the smaller suture, and the learning curve associated with placing a proper figure-eight suture. The lighting can be provided with a surgery light or headlamp. Handling the small suture with a small needle and placing the figure-eight suture can be frustrating initially, but, with practice, good lighting, and heavy sedation, the apposition that can be obtained is well worth the effort.

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[a]Vicryl, Ethicon Inc, Somerville, NJ 08807.
[b]Eye Saver, Jorgensen Laboratories, Loveland, CO 80538.

How to Manage Excessive Tearing in the Horse

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1. Introduction

Tearing is a common sign of ocular disease and is frequently misdiagnosed by equine practitioners as obstruction of the nasolacrimal system. This report will focus on the anatomy of the lacrimal system, the differential diagnoses of excessive tearing, and treatment of excessive tearing in the horse.

2. Anatomy and Physiology of the Nasolacrimal System

The primary function of the lacrimal system is to produce and drain the tear film from the surface of the eye. The tear film is composed of three integrated layers: the aqueous, mucus, and lipid layers. The aqueous layer is produced by the orbital lacrimal gland and gland of the third eyelid and secreted onto the ocular surface through ductules that traverse the conjunctiva. The mucus layer is produced primarily by conjunctival goblet cells and secreted directly onto the ocular surface. The lipid layer is produced by the meibomian glands present at the eyelid margin and is also secreted directly onto the ocular surface. Tear film plays an essential role in corneal health; it provides oxygen and nutrients to the corneal epithelium while removing debris and waste products. Tear film also keeps the ocular surface lubricated and plays an important part in the optics of the eye, allowing light to pass uninterrupted from the external environment into the eye. Immunologic properties of the tear film are important for ocular surface defense and health. The old adage, "no foot, no horse," is certainly true; its ocular equivalent is "no tear film, no eye"!

The nasolacrimal drainage system is responsible for removing the tear film from the ocular surface. The structures involved in the nasolacrimal drainage system include the ocular (proximal) puncta, the canaliculi, the lacrimal sac, the nasolacrimal duct, and the nasal (distal) puncta (Fig. 1). The ocular puncta are located in the medial canthus, with one punctum located just inside the eyelid margin of the upper eyelid and one punctum located just inside the eyelid margin of the lower eyelid. The ocular puncta are difficult to visualize without magnification. Normal tear film flow through the ocular puncta is facilitated by blinking and by the apposition between the eyelid and the ocular surface. Each punctum is drained by a canaliculus that connects to a common lacrimal sac. This lacrimal sac is drained by the nasolacrimal duct, which runs through the skull and empties through the nasal puncta, located on the ventral floor of the nasal passage (Fig. 2).

The nasolacrimal system exists in a balance between tear production and tear drainage by the nasolacrimal drainage system. Normal horses have an ocular surface tear volume of approximately 230 μL and a tear flow rate of 33.62 μL/min, with

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Fig. 1. Diagram of the nasolacrimal drainage system in a normal horse. Two proximal ocular puncta (dorsal and ventral) are each drained by a canaliculus to a common lacrimal sac. The nasolacrimal duct drains the lacrimal sac to the distal nasal punctum.

the entire volume of the tear film recycled every 7 minutes.[1] Any abnormality in the system can result in the tear film overflowing the system and draining onto the face. Diseases of the nasolacrimal drainage system can be roughly divided into two categories: diseases of excessive tear production and diseases of decreased tear drainage.

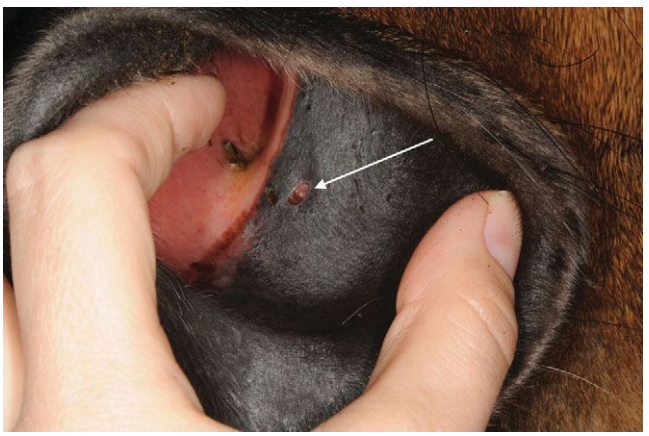


Fig. 2. The normal nasal puncta can be observed on the ventral floor of the nares as a pink mucosal oval in otherwise haired skin (arrow).

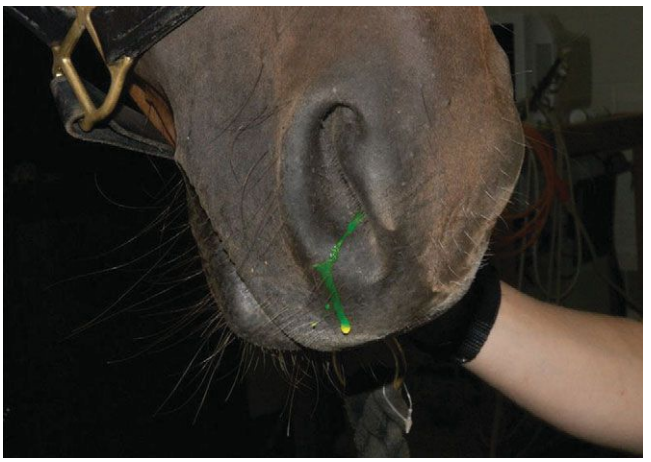


Fig. 3. A positive Jones test is demonstrated in this horse with a normal nasolacrimal drainage system. Fluorescein is observed draining freely from the nares.

3. Assessment of the Nasolacrimal System

Tear production can be assessed in the horse by the Schirmer tear test (STT). To perform an STT, a folder Schirmer tear strip is placed in the lower conjunctival fornix of an unsedated horse for 60 seconds. The STT is useful for diagnosing deficiencies of tear production, but is not particularly helpful for diagnosing excessive tear production. The function of the nasolacrimal drainage system can be assessed several ways, most importantly by performing a complete ophthalmic examination on the patient to identify any cause for excessive tearing.

The function and patency of the nasolacrimal system is assessed by the Jones test, whereas patency alone can be assessed by lavage of the nasolacrimal duct. It is important to distinguish the clinical implications of the results of these two tests: lavage of the nasolacrimal system only confirms patency, not function. The Jones test is performed by instilling a small amount (0.3–0.5 mL) of fluorescein solution onto the ocular surface and observing fluorescein at the nasal puncta within 5 to 20 minutes (Fig. 3). Sedation may facilitate rapid passage of fluorescein by lowering the horse's head. A negative Jones test is one in which no fluorescein is observed at the nares.

Nasolacrimal duct lavage can be performed as retrograde (through the nasal puncta) or normograde (through the ocular puncta). Retrograde lavage is easier to perform because it does not require topical anesthesia or magnification to identify the puncta. When retrograde lavage is unsuccessful, normograde lavage should be performed. A suitable cannula should be used, which may include a 4F to 6F polyethylene urinary catheter, an open-ended tomcat catheter (best for normograde lavage), or a 16-gauge intravenous (IV) catheter with the stylet removed (best for retrograde lavage). To perform retrograde lavage, the patient should be

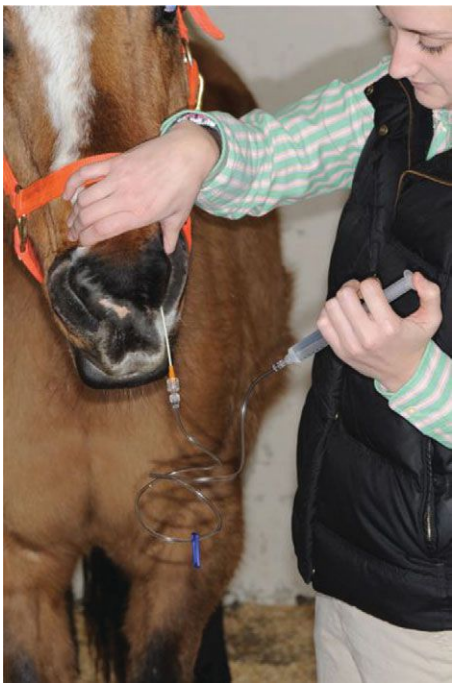


Fig. 4. Retrograde lavage of the nasolacrimal duct. The practitioner stands to the side of the horse and uses the far-side hand to place a cannula (in this case, a 16-gauge IV catheter with the stylet removed) into the nasal puncta. The near hand then occludes the nasal puncta and secures the cannula while the far hand injects saline solution through IV extension tubing attached to the cannula. This positioning allows the practitioner to keep the cannula in a secure position while easily injecting with less chance of the patient dislodging the cannula.

sedated and the cannula gently fed several centimeters into the nasal puncta (Fig. 4). The cannula should be retracted if resistance is encountered because some horses may have a blind-ended pouch in their distal nasolacrimal duct that can be inadvertently cannulated, making lavage more difficult. Intravenous extension tubing can be attached to the cannula to allow the practitioner more freedom to attach a syringe with less risk of dislodging the cannula. Sterile saline (10–12 mL) may then be slowly injected through the cannula and observed to drain from the eye. No more resistance should be encountered during injection than is expected with an IV injection. Gentle pulsing pressure may be useful for dislodging obstructions. Normograde lavage is performed similarly, with the exception that a topical anesthetic should be applied to the ocular surface before cannulation of the ocular puncta.

Advanced imaging techniques have been described for the nasolacrimal duct. Dacryocystorhinography refers to the technique of imaging the nasolacrimal drainage system through the use of radiopaque contrast agents and can be performed by means of radiography or computed tomography.[2,3] Endoscopy of the nasolacrimal duct has also been reported. These techniques are not widely used in clinical practice but are useful for localization in cases of nasolacrimal duct obstruction.[4,5]

Table 1. Possible Causes of Ocular Pain in Horses

Blunt trauma	Corneal perforation/ penetrating trauma
Eyelid abrasion/laceration	Corneal ulcer
Entropion	Corneal abrasion
Eyelid margin defect	Corneal foreign body
Eyelid neoplasia	Stromal abscess
	Ocular surface neoplasia (ie, squamous cell carcinoma)
Ectopic cilia	
Conjunctivitis	Conjunctival foreign body
Uveitis	Glaucoma

4. Diseases of the Nasolacrimal System: Excessive Production

The most common cause of excessive tearing in horses is excessive production of tears secondary to an irritating stimulus. A complete ophthalmic examination, including fluorescein staining, tonometry, and funduscopy, should be performed on every horse presented with a complaint of tearing. Excessive tear production is frequently accompanied by blepharospasm, another indicator of ocular pain. Sign of ocular pain is an important observation for differentiating ocular discharge associated with excessive production of tears from that associated with decreased drainage of tears. Decreased drainage of tears is not associated with ocular pain and should not be associated with blepharospasm. When blepharospasm and excessive tearing are observed concurrently, a painful ocular condition is the most likely cause of the excessive tearing.

Possible causes of ocular pain are extensive and diverse (Table 1). The astute practitioner will thoroughly examine the patient for subtle clinical signs of ocular disease. Careful attention should be paid to the intraocular exam. Miosis accompanying blepharospasm is a sign of intraocular disease and should prompt further examination. The application of topical anesthetic to the ocular surface may be a useful diagnostic test. If blepharospasm resolves with the application of topical anesthetic, an ocular surface disease is the most likely cause of the excessive tearing and blepharospasm. If blepharospasm does not resolve, intraocular disease is the most likely cause.

One important cause of ocular discomfort and excessive tearing is environmental irritants. These may include allergens, particulate matter, wind, ultraviolet light, or extreme temperatures. This is a diagnosis of exclusion after eliminating other causes of excessive tearing. Treatment beyond environmental modification may not be required for such cases. A flymask may be useful in decreasing environmental irritants. Topical antihistamines[a] are rarely indicated but may be useful in some cases.

5. Diseases of the Nasolacrimal System: Decreased Drainage

Congenital Malformations

Congenital abnormalities of the nasolacrimal drainage system are most frequently diagnosed in horses <1 year of age.[6–10] Ocular discharge may be serous or mucopurulent in nature. The nasal puncta is the most common site of atresia, and diagnosis can be confirmed by visual assessment of the nasal floor. The Jones test and nasolacrimal duct lavage will be negative in cases of nasal punctal atresia. The nasolacrimal duct itself may also have atresia, and dacryocystorhinography may be warranted.

Nasal punctal atresia is treated by surgical creation of a nasal puncta. The procedure can be performed under general anesthesia or standing sedation with topical and local anesthetic. The upper or lower ocular puncta are cannulated with the use of 5F plastic tubing (such as a urinary catheter) and advanced distally until the catheter tip can be observed at the ventral nasal floor. A No. 15 scalpel blade is used to make a single full-thickness incision through which the catheter is fed. Hemorrhage associated with this incision may be significant, so a single decisive incision is recommended. The catheter is then sutured at the medial canthal skin and nares with the use of three to five sutures per site for maximum security. After surgery, the patient is treated with a broad-spectrum topical antibiotic solution and systemic nonsteroidal anti-inflammatory drugs. The catheter is left in place for 4 to 6 weeks as a stent while the newly created puncta heals. Patency can be confirmed by the Jones test before removal of the catheter. Premature dislodging of the catheter can be addressed by replacing the catheter as previously described. Dacryocystitis and other complications associated with catheter placement is uncommon.

Other congenital malformations, including anomalous puncta, have been reported in the horse.[5] Surgical repair of such abnormalities require dacryocystorhinography to ensure that the anatomic location is identified and that appropriate surgical repair is selected.

Functional Obstruction: Dacryocystitis, Foreign Body, and Dacryolith

The nasolacrimal drainage system may become inflamed or infected (termed dacryocystitis), resulting in functional obstruction to outflow. Intraluminal foreign bodies are suspected as an occasional cause of nasolacrimal drainage system obstruction. Dacryoliths (mineral concretions within the nasolacrimal duct) are rarely reported.[11] External compression from sinonasal disease may also decrease nasolacrimal duct flow.[12]

Clinical signs include serous or mucopurulent discharge. The Jones test is negative, and nasolacrimal duct lavage should be attempted in all cases. If lavage is successful, post-lavage treatment with broad-spectrum systemic antibiotics and anti-inflammatories for 2 to 3 weeks is warranted. A topical antibiotic-corticosteroid solution may also be used, provided that the cornea is fluorescein-negative and free of ulceration. When the obstruction is difficult to dislodge or recurrence occurs, cannulation of the nasolacrimal duct as is described for nasal punctal atresia may be warranted. Any catheter should be left in place for 4 to 6 weeks, and concurrent antibiotic and anti-inflammatory treatment should be implemented. Advanced surgical intervention, including creation of alternate tear flow pathways into the nearby nasal cavity or sinus (canaliculorhinostomy or conjunctivorhinostomy), has been infrequently reported in the horse.[9,13]

Malpositioned Globe

Enophthalmos (caudal displacement of the globe within the orbit as the result of decreased orbital contents) results in poor apposition of the eyelid margin against the globe. This malpositioning of the globe relative to the eyelids results in decreased tear flow into the ocular puncta and canaliculi. Enophthalmos is more common in older horses and horses in poor body condition caused by atrophy of orbital fat. Treatment includes improving body condition where possible and diligent facial hygiene, with special attention to keeping the periocular skin clean and dry.

Ocular Punctal Occlusion

Atresia of the ocular puncta is rare, but chronic conjunctival inflammation may lead to fibrosis and occlusion of the ocular puncta. Treatment is similar to nasal punctal occlusion, although cannulation of the nasolacrimal duct must be performed in a retrograde fashion.

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[a]Opcon A, Bausch & Lomb, Rochester, NY 14604-2701.

How to Medically Manage a Case of Equine Eosinophilic Keratoconjunctivitis

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1. Introduction

Equine eosinophilic keratitis or keratoconjunctivitis (EK) is an inflammatory disease of the conjunctiva and cornea. This disease has been described in other veterinary species, including cats,[1–6] dogs,[7] and rabbits[8]; however, there is a dearth of information in the veterinary literature regarding this disease in horses.[9–18] The disease appears to have a seasonal component, with the majority of cases occurring in summer and early fall. EK has been reported in the Quarter Horse, Thoroughbred, Arabian, Warmblood, Mustang, and Icelandic horse breeds and in one donkey. No definitive sex or breed predisposition has been identified. The exact etiology and pathogenesis of EK are still unknown, but EK is postulated to be the result of a hypersensitivity reaction to an unknown stimulus, a theory supported by the response to treatment with immunomodulating drugs in horses and other species.[1–17] In horses, parasitic causes, such as *Onchocerca*, *Thelazia*, and *Habronema* infections, have been investigated and have not been found to play a role in the development of EK.[16] Although a wide range of treatment approaches has been used in horses, including systemic and topical medications and surgical interventions, no treatment approach has been rigorously tested and shown to be superior to any other. Irrespective of treatment, the course of the disease is often extended, and it is not unusual for treatment to extend beyond 1 month. The exception is surgical intervention with a superficial keratectomy, which results in a relatively rapid resolution once performed.

In one case series, Yamagata et al[10] reported on seven horses diagnosed with EK in the Midwestern United States from 1976 to 1994. These horses were treated either medically or surgically. Time to resolution of clinical signs ranged widely, from 14 days for one horse that was treated with a superficial keratectomy, 90 days for one horse treated with topical antibiotics only, and between 45 and 106 days for five horses treated with a combination of topical medications, including antibiotics and corticosteroids. Secondary corneal bacterial or fungal infections were not identified in these cases. Although this is a small case series, it supports the hypothesis that topical corticosteroid treatment may increase the time to resolution, whereas surgical intervention with a superficial keratectomy may hasten resolution of EK.

After this case series was reported, several case reports and case series have described EK in equids.

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A single case of EK in a 29-year-old donkey was reported by Jennings.[11] The donkey presented with a 2-day history of unilateral ocular discharge and a limbal mass. After treatment with topical dexamethasone and topical antibiotics, the lesions had resolved at the time of a follow-up appointment 30 days after initiation of treatment. In another single case report of EK, bilateral conjunctival and periocular skin biopsies were performed in a 17-year-old Icelandic mare with a 4-week history of bilateral conjunctivitis.[12] In this mare, BPV-1/-2 DNA and oncogene E5 transcript were identified, suggesting a role for a viral component in the development of EK in horses.

The following year in a conference abstract, Sandberg et al[13] reported EK in a group of three horses recurring in the summer months over a 2-year period. All three horses responded poorly to medical therapy with topical antibiotics and topical as well as systemic corticosteroids but demonstrated rapid resolution after a superficial keratectomy. Histopathology and transmission electron microscopy of the keratectomy specimens identified eosinophils in the anterior corneal stroma. Allergic dermatitis was diagnosed in two of these horses, raising the possibility of a multi-system eosinophilic disease process or general immunoglobulin E–mediated hypersensitivity disease. Kafarnik[15] reported a single case of EK in a 7-year-old Cob gelding with a 3-month history of bilateral corneal ulcers. Superficial keratectomy led to resolution in 14 days, with no recurrence reported in 6 months of follow-up. Diamond burr debridement has been used to treat horses previously diagnosed with EK and with subsequent nonhealing corneal ulcers.[18] Taken together, these cases support the hypothesis that superficial keratectomy may be associated with more rapid resolution of clinical signs than medical therapy alone.

Although the superficial keratectomy has shown the most promise for achieving rapid resolution of EK, surgical intervention is not the best "first-line" treatment for mild or recurrent cases. In addition, even in cases with severe and prolonged disease, surgical intervention is often declined by owners because of concerns about risks of general anesthesia and/or financial constraints. Therefore, identifying the best non-surgical approach to treatment of EK is important and useful in managing a disease that has a seasonally reoccurring component. The most recent insight into medical management of EK is in a retrospective study of 46 eyes in 26 horses.[17] In this study, the signalment, history of EK, presenting signs, medical therapy, time to resolution, and recurrence during a follow-up period (mean, 1.9; standard deviation [SD], 1.6; range, 0.3–4.7 years) were evaluated in horses diagnosed with EK from 2008 to 2012. The results of this retrospective study support the use of systemic corticosteroids and a systemic histamine 1(H1)-receptor antagonist in cases of equine EK and provides additional evidence that EK is a recurrent, seasonal disease in horses.

On the basis of this literature, medical management of equine EK should include standard ulcerative keratitis regimen (described in Treatment Approach section) in addition to a short course of systemic corticosteroids to combat the plausible allergic hypersensitivity component. An H1-receptor antagonist, cetirizine, should also be prescribed as both an immediate treatment and future preventative measure.[17] Although use of topical corticosteroids in some cases of EK may result in short-term improvement, the use of topical corticosteroids in the face of a corneal ulcer is dangerous because it may increase the incidence and severity of secondary bacterial and fungal corneal infections.[17]

2. Clinical Signs and History

Clinical signs of EK include moderate to severe ocular pain, caseous ocular discharge, severe conjunctival hyperemia and chemosis, and pink proliferative corneal plaques, most often originating at the corneolimbal junction.[9–17] White, cellular plaques may be present overlying the pink proliferative lesions. Individual cases of EK can present with clinical signs at any point along the clinical spectrum, from severe conjunctivitis and blepharitis with or without corneal ulceration, to geographic corneal ulceration with secondary bacterial and/or fungal involvement. The majority of horses are exquisitely painful on presentation with moderate to severe epiphora and blepharospasm. The disease can present unilaterally or bilaterally, and severity can differ between left eye and right eye when bilateral. Reflex uveitis is often present. There was a clear seasonal component, with 92% of cases in one study having developed clinical signs from June through October.[17]

Many horses present with a history of similar ophthalmic clinical signs observed by the owner during the summer and early fall months of previous years. In one study, 44% of horses diagnosed with EK had a 1- to 5-year history of confirmed EK.[17] An owner may also report that within a single year, their horse exhibits repeated bouts of conjunctivitis, blepharitis, and corneal ulceration, which respond poorly to routine cornel ulcer management, including topical antibiotics and antifungals, systemic non-steroidal anti-inflammatory drug (NSAID) medication, and topical mydriatic/cycloplegic treatment. Although in many cases the definitive diagnosis of EK cannot be made for previous episodes, awareness of a possible recurrent disease process may raise a red flag to the clinician that this is not a typical case of ulcerative keratitis.

3. Examination and Diagnosis

A complete ophthalmic examination should be performed to rule out other causes of conjunctivitis, blepharitis, corneal ulceration, and anterior uveitis, if present. Differential diagnoses for EK include

other infiltrative, potentially ulcerative corneal diseases, including fungal, bacterial, parasitic, or viral keratitis, neoplasia, and calcific band keratopathy.[16] A definitive diagnosis of EK is made on the basis of predominance of eosinophils on corneal or conjunctival cytology in conjunction with clinical signs.[16] Eosinophils may be accompanied by smaller numbers of mast cells, lymphocytes, plasma cells, and neutrophils, in addition to bacteria or fungal hyphae, depending on whether a secondary corneal infection is present. A brightly eosinophilic, acellular, granular material may surround the corneal plaques. On the basis of the results of a general ophthalmic examination and cytological evaluation, additional microbial diagnostics should be performed, including bacterial culture and antimicrobial susceptibility and fungal culture, because the risk of secondary corneal infection is high in any case of ulcerative corneal disease in horses.

Corneal cytological evaluation is critical to making the diagnosis of EK. The easiest way to initially misdiagnose a case of EK as a case of "simple" ulcerative keratitis or conjunctivitis of another etiology is to fail to perform cytology.

4. Medical Treatment Approach

On the basis of data from a retrospective study of 46 eyes in 27 horses,[17] the most effective medical treatment regimen includes a short, tapering regimen of systemic dexamethasone; systemic H1-receptor antagonist (cetirizine); topical antibiotic and antifungal treatment to prevent and/or treat secondary corneal infection; topical mydriatic/cycloplegic to address reflex uveitis; and systemic NSAIDs after cessation of systemic dexamethasone.[17]

Eosinophilic keratoconjunctivitis cases are at risk for secondary corneal infection, with 51% of eyes having a positive bacterial or fungal culture in one study.[17] Therefore, topical antimicrobial therapy is critical. The medical management of horses diagnosed with EK is routine with respect to treating corneal ulceration and reflex uveitis. Treatment should include a topical antibiotic and antifungal, topical mydriatic/cycloplegic, and systemic NSAIDs. With the exception of mild cases of EK, a tapering regimen of systemic dexamethasone is used as follows: 0.04 mg/kg for 1 day, 0.03 mg/kg for 2 days, and 0.02 mg/kg for 3 to 5 days.[17] The initial dose is typically administered intravenously, with all subsequent doses given orally. After treatment with systemic dexamethasone, a significant improvement in conjunctivitis and blepharitis is typically observed within 24 to 48 hours.

Horses diagnosed with EK are also treated with systemic cetirizine given orally at a dose of 0.4 mg/kg twice daily.[17] Cetirizine is an H1-receptor antagonist used for the treatment of seasonal allergies in humans. It acts by blocking the H1-receptor and preventing the histamine-dependent increase in adherence and migration of eosinophils.[21] Blocking additional eosinophils from being recruited and migrating to the conjunctiva and cornea could help alleviate the clinical signs of EK in horses. This is supported by in vitro research in an epithelial cell culture model in which epithelial cells showed signs of toxicity and a decrease in the rate of wound repair after treatment with the major eosinophil granule product, major basic protein.[19,20] On the basis of these studies, it can be hypothesized that the presence of degranulating eosinophils on the equine cornea could lead to corneal ulceration and failure of normal corneal healing.[20] Therefore, preventing additional eosinophil recruitment and migration to the corneal surface could benefit horses diagnosed with EK. Pharmacokinetic studies have identified a safe oral dose in horses that achieves similar plasma concentrations to those achieved in humans.[21] In a histamine-induced cutaneous wheal formation study in horses, it was shown that pretreatment with cetirizine inhibited wheal formation.[22] These studies support the use of cetirizine in cases of EK as a way to reduce the severity of clinical signs while the horse is having an active bout of EK or during at-risk times of year, in an effort to reduce the recurrence rate.[17]

Extended treatment is the reality for the majority of cases of equine EK, with more than 80% of cases requiring medical treatment for at least 3 months.[17] In one retrospective study, time to resolution was significantly shorter for horses treated with systemic corticosteroids (mean, 2.23; SD, 1.13 months), relative to patients that were not so treated (mean, 4.20; SD, 1.47 months).[17] Recurrence is common in this disease, with a 33% recurrence rate; however, horses treated with cetirizine are significantly less likely to have recurrence during the following year.[17] In one study, recurrence of EK was reported for only one of 13 horses (8%) treated with cetirizine, relative to recurrence in eight of 14 horses (57%) that had not been treated with cetirizine.[17]

5. Monitoring and Follow-Up

It is imperative that frequent re-evaluations are performed on horses diagnosed with EK, just as with any case of equine ulcerative keratitis. However, following up with owners dealing with equine EK is perhaps even more important compared with uncomplicated ulcerative keratitis because of the prolonged treatment time often required to achieve resolution of clinical signs. As many veterinarians have probably seen in other cases of ophthalmic disease requiring frequent daily treatments, an owner's inability to treat ulcerative keratitis in their horse can lead to significant worsening of the disease in a short period of time if not reported to or appreciated by the veterinarian. A subpalpebral lavage delivery system is often necessary to permit extended treatment of horses with EK. Additionally, serum total protein should be monitored weekly and serum creatinine every 2 weeks because of the well-established gastrointestinal and renal

adverse effects associated with chronic NSAID administration.

6. Environmental Management

The role of environmental management in the development and duration of clinical signs of EK is not completely understood. Controlling EK by environmental modification is predicated on the hypothesis that EK is a hypersensitivity reaction to an environmental allergen. The role of environmental modification in controlling EK was reported by Utter[6] on a single farm in July to August 2009. Utter reported the incidence of EK in 19 Standardbred broodmares, representing an incidence of 10% on the farm in a single year. With the addition of specific management changes, including the use of fly masks, fly repellant, night turnout, keeping pasture cut close to the ground, and longer periods of time housed in stalls, fewer horses had EK the following year, and overall clinical signs were less severe than during the previous year. This approach to controlling recurrence of EK is especially significant in horses not treated with cetirizine because of financial constraints.

7. Conclusions

Eosinophilic keratoconjunctivitis in horses is a disease that can be frustrating for veterinarians, owners, and horses alike because of the severity of clinical signs and prolonged treatment often required to achieve resolution. Additionally, it is not uncommon for horses to have additional bouts of disease during the same year or later years because of the high recurrence and seasonality of the disease. Correctly diagnosing the disease as EK is the first step to beginning an appropriate therapeutic plan with the best chance of success. Treating the disease with systemic dexamethasone and cetirizine, in addition to topical antibacterial, antifungal, and mydriatic/cycloplegic medications, has proved to be the best medical treatment option currently available. Additionally, continuing treatment with cetirizine during future summer and early fall months and recommending environmental modification can help to decrease recurrence and severity of clinical signs in horses that have repeated bouts of confirmed EK.

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How to Insert and Manage a Subpalpebral Lavage System

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1. Introduction

A subpalpebral lavage system (SPL) is an ophthalmic catheter that delivers topical liquid medication onto the surface of the equine cornea. The system consists of a long silicone tube that passes through the upper or lower eyelid and is secured to the facial skin. The tubing, which is woven through the mane and draped along the crest of the neck, ends in a closed port that is secured to the mane. Medication is injected into the port and pushed through the tube by an air bolus or by additional medications to reach the tear film.

An SPL is an essential tool for treating serious equine ocular conditions such as corneal lacerations, deep ulcers that are infected or melting, or severe uveitis.[1] Management of these ocular problems requires multiple treatments per day and may extend for several weeks. SPL medication is delivered without the handler touching the face or periocular region; therefore, treatment of fractious or painful eyes is simplified. An SPL makes therapy as easy as possible for the caretakers and ensures that all medication reaches the tear film to medicate the cornea. The use of an SPL is mandatory in any globe that is at risk for rupture caused by tectonic instability of the cornea or sclera.

The use of an SPL is not limited to the hospital setting. Practitioners can place SPLs and train farm personnel to implement treatment in the field. SPLs can be used on any size of horse and have been used to treat neonates and foals as well as adults. The use of an SPL increases the safety and efficacy of topical therapy, improves compliance, and reduces caretaker fatigue in complex, multidrug treatment plans.

2. Materials and Methods

Commercial SPL kits are available through two veterinary manufacturers.[a] This author prefers kits that provide a solid 12-gauge insertion trochar that is swaged onto the silicone tubing.[b] The kits offer a choice of a straight or angled insertion trochar; practitioners who use a gloved finger as a guide to slip the trochar beneath the lid may favor the angled version. The kits are sold in 36- and 60-inch lengths with a single 5F lumen diameter. The 60-inch kit is most versatile because the longer tubes are suitable for large horses and can be shortened to accommodate small patients. The 36-inch tubes may be too short for use in some horses.

The following checklist summarizes all items the author commonly uses to facilitate restraint, seda-

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tion, and site preparation and to secure the tube and construct a durable injection port:

- SPL Kit, 60 inches
- Sterile gloves (for trochar insertion)
- Topical bottle of 2% povidone iodine solution and gauze 4 × 4 gauze pads (to clean the periocular region)
- Proparacaine or tetracaine topical ophthalmic solution (to numb the corneal surface)
- Mepivicaine or lidocaine injectable (for blocking the eyelid)
- Several 3-mL syringes
- Detomidine (for sedation)
- Small rubber bands (to braid the mane)
- A roll of duct tape (to make butterfly tabs for securing the tubing)
- A roll of white adhesive tape (to make mane braids snug and create the base for the injection port)
- 20-gauge, 1.25-inch catheter (to create injection port)
- Male catheter cap (to create the injection port)
- Wooden tongue depressor
- Nonabsorbable suture (1–0 to 1 diameter, to secure the tube to the skin)
- Olsen-Hegar needle holders, 6 inches long (for tube suturing)

SPL treatment plans require a large quantity of tuberculin (1-mL) syringes and 25-gauge, five-eighths–needles. These may be dispensed in box quantities. A selection of colored tape rolls will aid in labeling different medication syringes.

Installation

The clinician should ensure that the horse is restrained in a clean, wind-free, well-lit, quiet area. Placement of an SPL is not difficult, but it carries a risk to the patient because insertion involves brief passage of a sharp trochar over the corneal surface. The clinician must insist that barn activity be kept to a minimum when the trochar insertion takes place. All necessary items should be placed on a clean, elevated surface. Folding tables sold for dog grooming make handy equipment stages in the field.[c]

The owner or veterinary assistant should braid the mane and forelock into a series of "pigtails" that will serve as guides to secure the tubing to the crest of the neck. Because the tubing may be maintained for several weeks, it is helpful to secure the top and bottom of each pigtail with a wrap of adhesive tape so the braids do not loosen.

Insertion is safest when the horse's mandible is supported on a solid object (stacked bales, oil drum, recycling bin, etc). Stocks are helpful if they are available. The horse should be sedated heavily with the use of detomidine at a dose of 0.02 to 0.04 mg/kg. Deep sedation will cause the horse to rest the mandible on the support material, and the handler will only have to lightly brace the head while standing on the opposite side to the affected eye (Fig. 1).

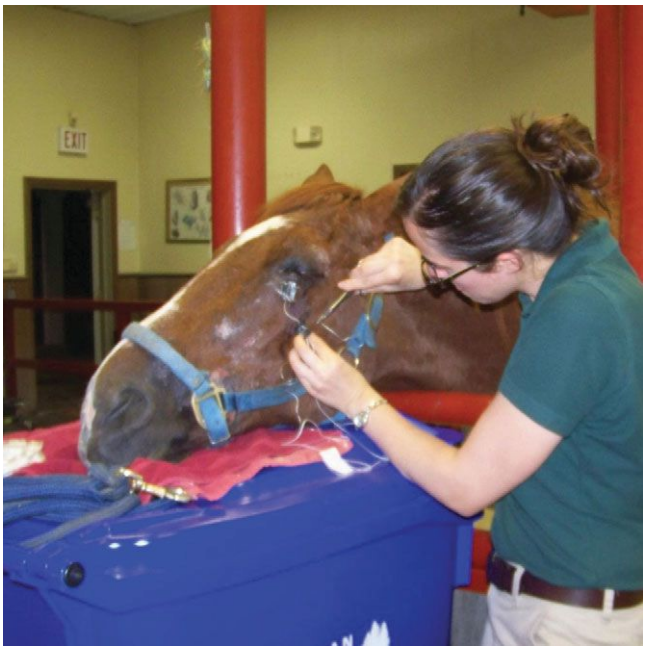


Fig. 1. A wheeled recycling bin or other stationary prop is used to support the horse's mandible for safe SPL insertion.

The periorbital region is cleaned with a 1:50 povidone iodine/saline solution. The target area for trochar insertion is blocked with local infiltration of mepivicaine or lidocaine. If the tube is intended for the upper eyelid, an auriculopalpebral and supraorbital block should be performed. One milliliter of topical anesthetic is drawn up into a tuberculin or 3-mL syringe and sprayed onto the corneal surface with the use of the hub of a broken-off 25-gauge needle as a spraying device.

The SPL tube can be inserted into either the upper eyelid or the lower eyelid (Fig. 2A,B). Some clinicians think that the use of the dorsotemporal fornix of the upper lid provides beneficial "waterfall effect" in which the medication cascades over the ocular surface. Others argue that insertion through the ventronasal cul-de-sac of the lower lid provides protection against iatrogenic ulceration because the nictitans is a barrier if the tube footplate migrates in from the conjunctiva. There is good evidence to support that a lower-lid SPL is an effective and safe choice.[2] Ultimately, the choice will depend on clinician preference, patient temperament, and location of the lesion. Securing the tube to the face can be done in a linear pattern from upper eyelid insertion sites. If the lower lid is used, the tube must be secured to the face in a J-shaped pattern.

The following steps (Fig. 3A–D) describe the insertion process once the mane is braided, the horse

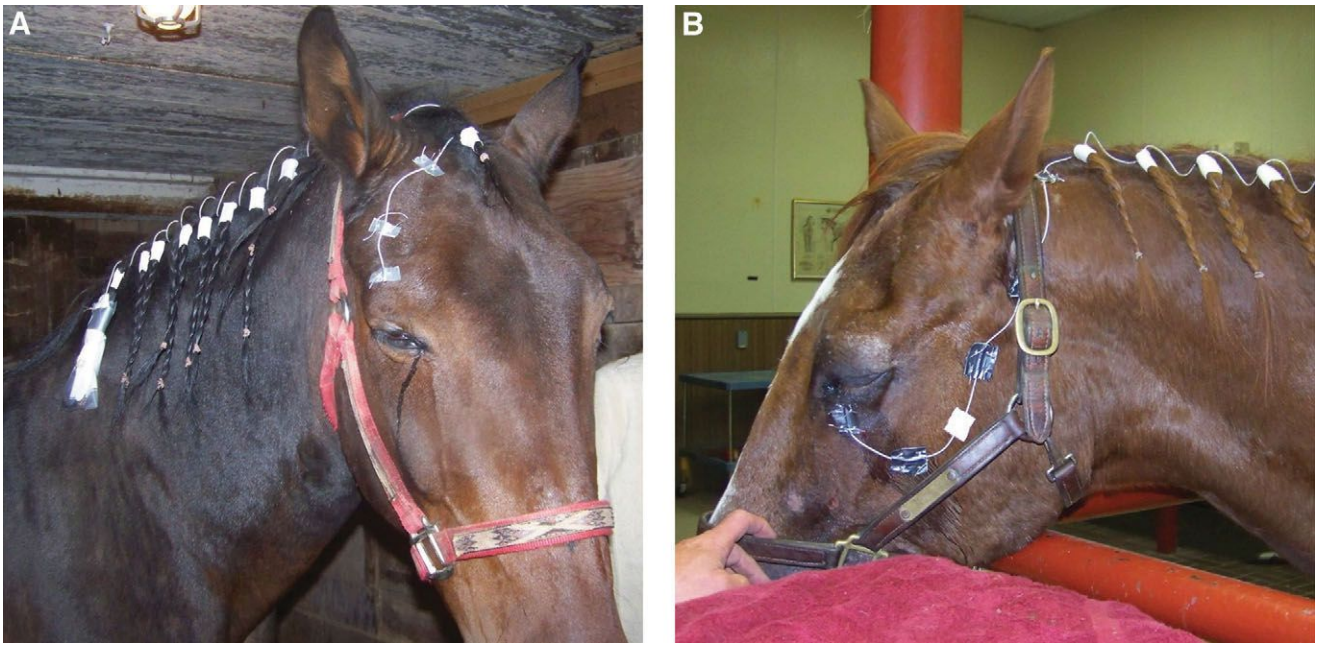


Fig. 2. A, SPLs can be inserted into the upper or lower lid. This system was inserted into the upper eyelid. B, This SPL was inserted into the lower eyelid.

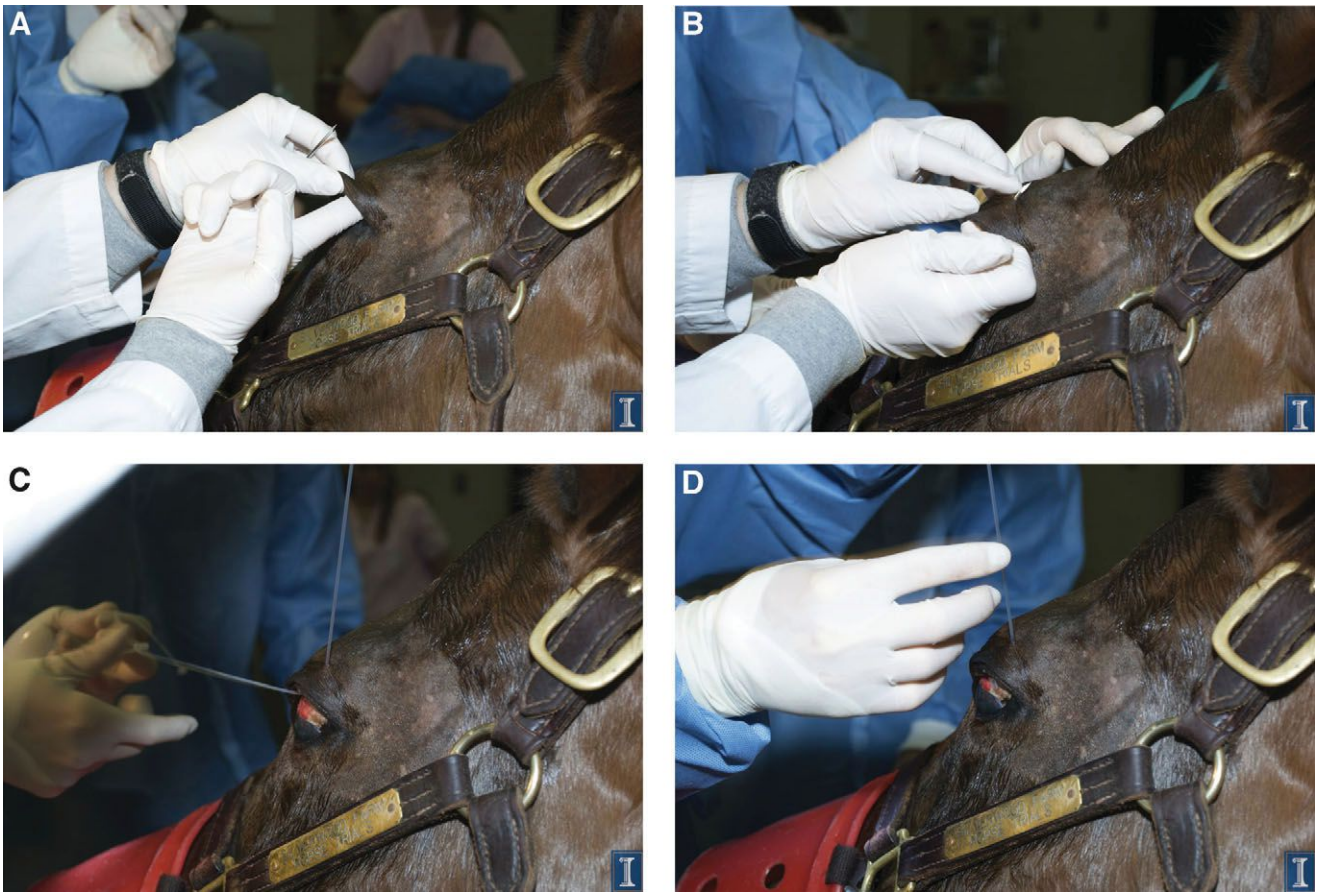


Fig. 3. A, SPL insertion: A gloved finger is used to probe the depths of the fornix and test the horse's reaction. B, The trochar is carefully pushed through the palpebral conjunctiva and eyelid. C, The SPL tubing is pulled through the eyelid. D, The tubing footplate is pulled under the lid. Care must be taken to align the angle of the footplate with the angle of the palpebral conjunctiva. Photo courtesy of University of Illinois Comparative Ophthalmology Service, Amber Labelle, DVM, DACVO.



Fig. 4. A, Adhesive or duct tape wings are folded over the tubing and sutured to the facial skin. B, The SPL trochar is used to weave the tubing through the mane.

is sedated and blocked, and topical anesthetic is applied to the cornea:

(1) A gloved finger or a blunt sterile probe (cotton-tipped wooden swab or the hollow insertion tube provided with the SPL kit) into the deepest palpebral reflection is used to test the eyelid block and tolerance of instruments at the site. Additional sedation and/or anesthetic is indicated if the horse reacts (Fig. 3A).
(2) With the use of either a gloved finger or the hollow insertion tube as a guide, the trochar point is then carefully advanced to the target puncture site adjacent to the orbital rim. The trochar tip is pushed through the lid skin (Fig. 3B). The trochar and tube are then pulled through the eyelid until the disc-shaped footplate of the SPL tube rests against the palpebral conjunctiva. Care is taken to ensure that the oblique angle of the footplate matches the slant of the eyelid conjunctiva and that the footplate is snug against the mucosa (Fig. 3C,D).
(3) The section of the tubing that exits the eyelid and traverses to the forelock is sutured to the facial skin (Fig. 4A). The author makes two to four half-inch wide "butterfly" wings out of duct tape or adhesive tape and fastens the first wing to the tubing at the eyelid skin exit hole. Other wings are taped to the tubing at appropriate intervals toward the ear. The wings are then sutured to the skin with the use of four simple interrupted sutures per wing. Alternatively, the tubing can be laced through the plastic U-shaped guides provided with the SPL kit, and the guides can be sutured to the skin in a similar pattern.
(4) The trocar is used to pull the tubing through the base of the forelock braid and then weave the tubing through the mane braids, with the use of a consistent "under-over" or "over-under" pattern of inserting the trochar into the center of the braid base (Fig. 4B). A stout braid near the withers is selected as the anchor for the injection port. When this braid is reached, the trochar is capped for safety before assembly of the port.
(5) The tubing is cut near the withers. The unused portion may be saved for future repairs.
(6) A 1.25-inch, 20-gauge catheter is carefully threaded into the lumen of the open tubing with the use of the catheter stylet (with the sharp tip pulled back slightly into the catheter lumen) as a probe to aid in the sleeving process (Fig. 5A). The stylet is removed, and a standard male catheter cap is twisted onto the catheter hub.
(7) The injection port is secured to the mane braid as described below.

The author prefers to cut a wooden tongue depressor in half and use two folded 4×4 gauze squares as a "bed" for the capped injection port, leaving a half inch of tongue depressor extending beyond the place where the cap rests (Fig. 5B). Adhesive tape is wound around the tubing, catheter, and gauze to seat the catheter firmly, leaving only the injection

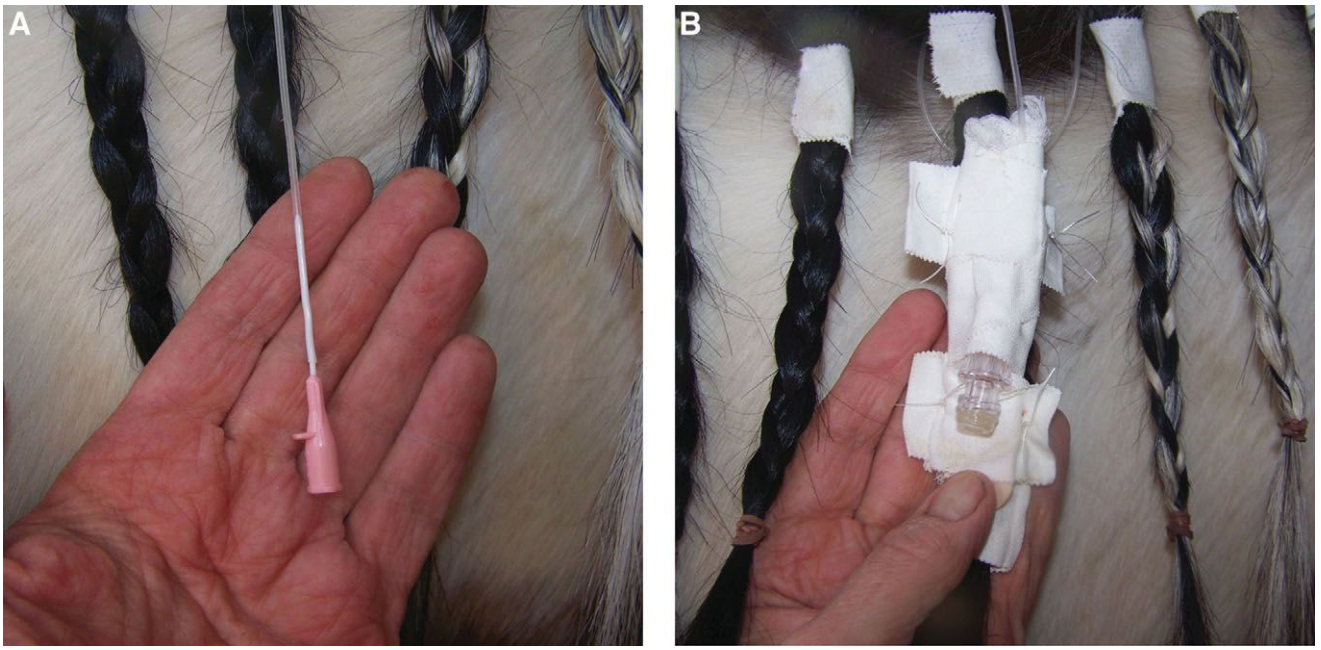


Fig. 5. A, The 20-gauge, 1.25-inch catheter is inserted into the SPL tubing near the withers to create the injection port. B, The injection port is closed with a male catheter cap. The tubing end and port are braced with a few pieces of gauze and a tongue depressor and taped and sutured to the anchoring mane braid.

port exposed. Tape wings are then used to secure the "braced" injection port to the anchoring braid. In some cases, tape wings are then sutured to the braid twists to further ensure that the port is well attached to the mane braid. Care taken with securing the port should ensure that the braid will not unravel with normal stable activity, even if the device remains in place for several weeks. A heavy plastic bag can be taped over the injection assembly to keep it clean.

Neonates and very young foals must undergo short-term general anesthesia or heavy sedation to induce lateral recumbency for safe insertion of an SPL, but all other steps in the process remain the same. Very young foals will not have enough mane to make braids, but adhesive tape can be wrapped around short mane hairs to create a series of tufts that will serve the same purpose. The injection port assembly may need to be sewn to the skin (Fig. 6). Some clinicians slip a section of wide stockinette over the head and neck of the foal to protect the tubing from damage. Holes can be cut in the stockinette to provide openings for the eyes.

A series of tape butterfly guides must be sutured close to the crest of the neck to secure an SPL in an adult horse that has had the mane shaved off (roached).

Use of an SPL to Deliver Ocular Medication

Treatment involves injection of medication into the tubing cap. The tongue depressor support taped to the mane braid makes a secure handle to access the injection cap. Medication that has been drawn up into a tuberculin syringe is injected through a 25-gauge, five-eighths–inch needle (Fig. 7). Most medication is dosed at a volume of 0.2 mL. Some very costly medications are dosed at a volume of 0.1 mL.[d] The medication is then "pushed" toward the eye by a



Fig. 6. Tufts of mane can be used as a channel for SPL tubing in young foals. Two injection ports were secured to the skin of this foal's neck.



Fig. 7. Medication is injected into the SPL through a 25-gauge, five-eighths–inch needle attached to a tuberculin syringe. The injection port has been covered with a plastic bag.

second injection of air. The air injection should be done slowly to minimize discomfort and reduce washout. A volume of 1.5 to 2.0 mL of air is sufficient to push the medication beyond the footplate, where it then mixes with the tear film. Most horses will react with a slight head shake when the medication exits the tube.

Almost all SPL medication schedules require that multiple drugs, which may include mydriatics, antibiotics, anti-fungals, anti-inflammatories and anti-collagenases, are given several times a day. Most clinicians advise that each drug be given separately, with an interval of a few minutes before the next product is injected. Definitive research has not been performed to determine if "stacking" medication in a tube (injecting several products one after another and then using the air bolus to propel all of them at once to the eye) is as effective as individual injections, but a large volume (>0.4 mL) of liquid that reaches the eye all at once may spill out onto the face. Alternately, the SPL line can be loaded with medication, and each individual dose then may be administered at 5-minute intervals so that only 0.2 mL of medication is administered to the ocular surface with each dose.

Battery-powered, single-use infusion pumps designed to deliver continuous ocular medications into an SPL are commercially available.[e] These pumps have a 10-mL fluid reservoir that can be filled with liquid topical medication. They can be ordered in flow rates that vary from 0.06 mL per hour (10-mL reservoir is slowly discharged over 7 days) to 1.0 mL per hour (the entire reservoir is discharged in 10 hours). "T" and "W" adaptors that allow more than one pump to be hooked to the same SPL are available from the manufacturer. It is important to follow the directions when filling the reservoir and securing the discs to the SPL port because the pumping assembly relies on the intake of a small volume of air into the device to deflate the reservoir. Care must be taken to ensure that the air intake valve is not obstructed. The disc bladder should be checked periodically to ensure that the medication reservoir bag is deflating on schedule. When the disc is empty, it can be disconnected from the SPL and replaced with a new disc.

The author has used these pumps a few times in fractious horses and in situations in which manual treatment was not practical. Some clinicians have reported good success with these devices. It is important to consider the effect of ambient temperature on drug stability when using such devices because some drugs may not remain stable with prolonged exposure to room temperature.

Maintenance

The SPL catheter cap should be cleaned daily with application of an alcohol wipe. Catheter caps are changed at 3- to 7-day intervals in hospitalized patients. Cases handled in the field may undergo cap changes at veterinary recheck appointments. Some clinicians advise running a small amount (1 mL) of 1:50 povidone iodine/saline solution through the tube several times a week as an antiseptic, then flushing the tube lumen with air before the next scheduled medication application. Experimental evidence suggests that colonization of the SPL system by bacteria is uncommon; thus, the need



Fig. 8. The SPL footplate is removed by use of the adjacent section of tubing as a "pole" to push the disc away from the conjunctiva.

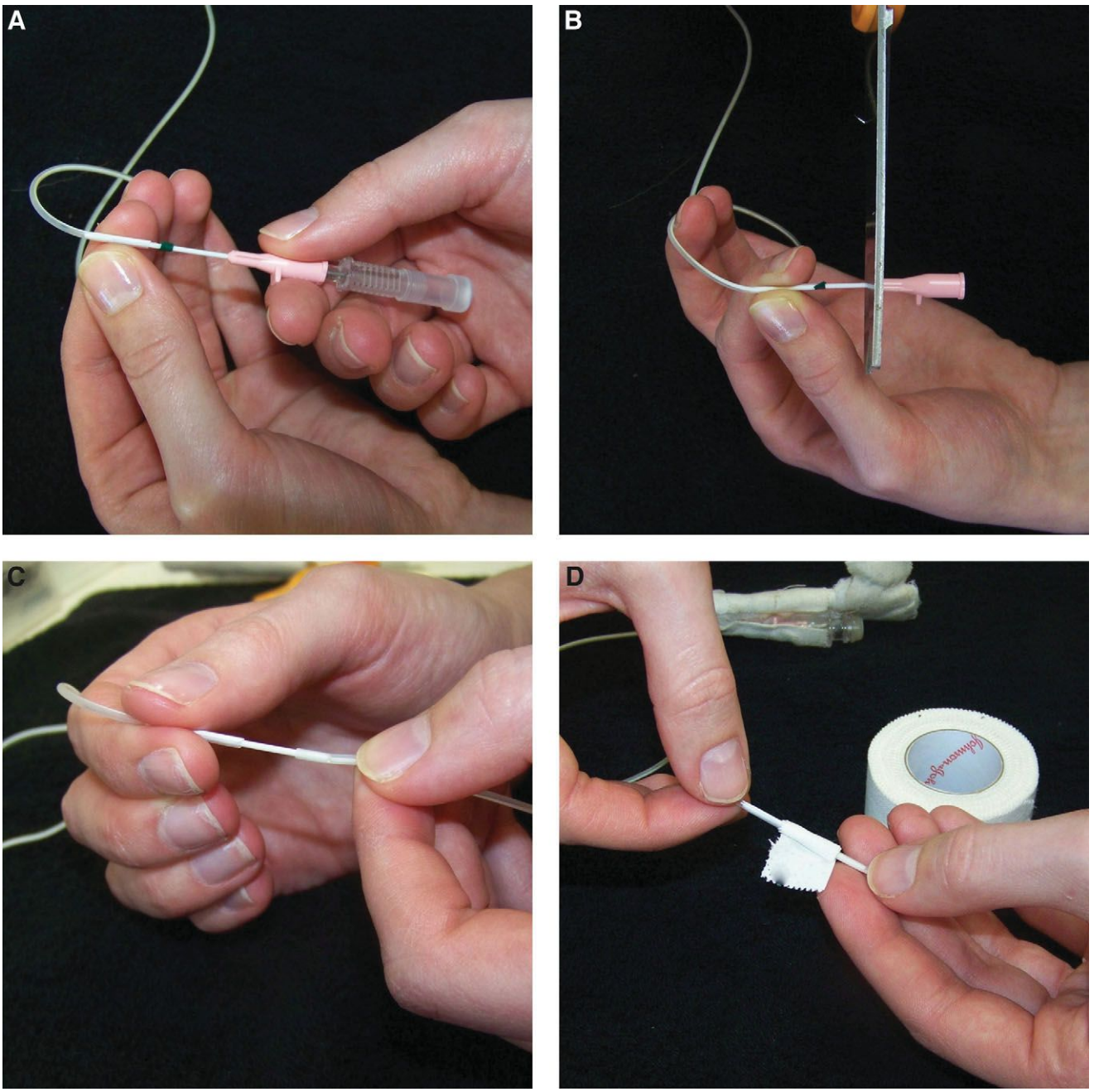


Fig. 9. A, SPL repair: The 20-gauge, 1.25-inch catheter is threaded halfway into one end of the broken tubing. The catheter stylet is then removed. B, The hub of the catheter is cut off. C, The other end of the broken SPL tubing is carefully "shimmied" onto the remaining length of catheter until the two ends meet. D, The spliced tubing section is wrapped with tape, then sutured with a separate tape wing to the horse's skin.

for lavage with an antiseptic solution is unconfirmed.[3] The tube suspension arrangement (braids, sutured tape wings) may require rebraiding, resuturing, or new tape application.

Removal

SPL tubes are easily removed in the standing, sedated horse by cutting the tubing a few centimeters away from the fornix or cul-de-sac exit site and removing all wings or guides that attach the cut section to the face. The tubing that remains attached to the footplate can be used as a "pole" to push the footplate away from the conjunctiva and into the fornix space (Fig. 8). A gloved finger is used to fish the footplate and short length of tubing out of the fornix and remove it.

Occasionally, the footplate will remain buried in the conjunctival tissues. In these instances,

sometimes the attached tubing can be used to pull the footplate out through the skin exit hole. Alternatively, the exit site in the eyelid skin can be infiltrated with a small amount of local anesthetic, and a simple cutdown surgery can be performed around the tubing stump to retrieve the footplate and associated remnant.

3. Results

Both veterinary ophthalmologists and general equine practitioners rely on these devices to treat their most difficult cases, and properly installed systems usually work very well in both hospital and field situations. SPL tubes have been left in patients for as long as 10 to 12 weeks. Treatment regimens range from multiple medications injected on an hourly basis for initial treatment of serious problems to treatment that is delivered just a few times a day for issues nearing resolution. The use of SPLs to deliver targeted treatment for serious equine ocular problems has preserved vision and saved thousands of eyes.

Complications

Although SPLs are managed in most horses without problems, complications do occur in some patients. The two major issues are iatrogenic corneal ulceration from footplate irritation and lack of patency caused by damage to the tube lumen (breakage or development of small punctures).

Corneal ulceration occasionally occurs after an SPL is placed in the upper lid if the device is not inserted deep in the fornix and the footplate rubs against the cornea. It can also occur if the footplate is not snug against the conjunctiva. Removal and replacement of the SPL is necessary to stop further mechanical irritation. A new SPL should be placed and firmly secured to avoid SPL-associated corneal trauma. The second SPL is often placed in the ventronasal aspect of the lower lid because at this site, the nictitans provides a protective barrier between the footplate and the cornea. Ulcers associated with lavage tube irritation are often very slow to heal; choice of treatment medication is dependent on analysis of cytology and culture of the ulcer bed.

Although the silicone tubing of an SPL is quite strong, it can break or develop leaks if the horse snags it on an object. Breaks will be obvious, but leaks may not be noticed immediately. The person treating the horse should be suspicious that the system is not working if a horse stops reacting when enough of an air bolus is injected to push medication into the tear film—the lack of a reaction may indicate that the medication is leaking out of the tube before it reaches the globe surface.

A simple method to check for patency is to tear off a small piece of a fluorescein dye strip and mix it with saline solution. One to 2 mL of the dyed saline is then flushed through the tube. The tear film will turn green as the dye/saline mixture exits the eyelid if the tube is patent. If the tube is leaking, the tear film will remain clear, but the dyed saline mixture will leak from the damaged part of the tube.

SPL tubing that breaks or tears near the withers can be repaired by simply shortening the tube. The open end of the tube is flushed with a quantity of 1:50 betadine/saline antiseptic, and a new catheter and cap are inserted. An injection port assembly is created as described above and taped and sutured to a more proximal braid.

SPL tubing that breaks near the ear or above the eyelid can be repaired with the use of a 20-gauge catheter as a splice to connect the two separated ends (Fig. 9A–D). The catheter is inserted into one end of the broken tubing, with the stylet retracted slightly back into the catheter lumen. The tubing is "shimmied" over half of the catheter length. The stylet is then removed, and the hub of the catheter is cut off. Half the Teflon catheter is exposed beyond the section that is sleeved with one end of the broken SPL tubing. The other end of the SPL tubing is then carefully pushed over the exposed catheter. This is somewhat difficult without the use of a stylet but can be done with a light touch and a little patience. When both of the broken ends of silicone tubing meet in the center of the catheter tubing, a small piece of white tape is wound around the junction to secure the splice. Patency of the spliced segment is tested with a saline flush, which should pass through the catheter lumen. The section of spliced tubing is then secured to the face or neck crest with a sutured tape wing.

4. Discussion

Horses diagnosed with deep stromal or melting ulcers and stromal abscesses usually require an SPL to deliver the intense treatment that is required to save the eye and preserve vision. Horses with conditions such as severe uveitis or indolent ulcers may also benefit from an SPL when the problem is very painful or requires prolonged therapy. Many of these cases can be handled effectively in the field. However, some cases are best referred—even when a practitioner has the skills to insert an SPL, optimum outcome may only be realized with specialist evaluation and hospitalization. Practitioners must consider several factors beyond the simple mechanics of installing an SPL when making clinical decisions on a problem eye. Serious infections and melting ulcers can quickly progress and threaten vision and ocular integrity; therefore, it is important that the treatment plan is based on a comprehensive examination and appropriate diagnostic testing. Practitioners who install SPLs must have skills and experience in assessing such cases and a broad knowledge of rational therapy choices.

SPL insertion is not inexpensive; the kit materials and sedation are costly, and the process of placing and securing the tube is somewhat time-consuming. Owners who bring their horses to a referral facility are prepared for significant expense and readily give permission for SPL placement. However, the first

veterinarian who sees the case usually does the assessment at the home stable. In these settings, owners are often unwilling to authorize tube placement because of the expense and the idea of "a tube in the eyelid." A common response when an SPL is advised is, "Let me just try to treat this myself for a few days."

That approach may be acceptable for a simple shallow corneal ulcer in a cooperative horse, but whenever a serious vision-threatening condition is diagnosed, the veterinarian must act as a strong advocate for the horse and recommend immediate SPL insertion. Corneal infections and deep stromal defects respond best if intense treatment is instituted right after initial diagnostics are completed, and an SPL is the key element for safe administration of effective therapy. Owners who decline tube placement often change their minds several days later when the treatments are not reaching the eye consistently, the horse has become unmanageable, and they are exhausted from trying to comply with the rigorous treatment schedule. At this point, the total management cost may actually increase because of progression of disease in the face of ineffective treatment, or it may be too late to initiate effective therapy.

Another element to consider in managing serious ocular problems in an ambulatory setting is that there must be sufficient manpower to carry out the prescribed treatment schedule; treatment may need to be delivered through the SPL six to 12 times per day. If the desired schedule cannot be delivered at the home barn, referral to a full-service hospital should be encouraged. If referral is not an option, the clinician should stress that having the tube in place is only as useful as the number of times it is used to actually treat the horse.



Fig. 10. Horses can be successfully treated on the farm with the use of aids such as fly masks and/or neck covers to protect the SPL.



Fig. 11. Colored tape is an effective way to label medication syringes and help caretakers keep track of treatments.

The author has installed and managed SPLs for a variety of problems in horses of all breeds and ages. Although some of these patients were hospitalized, the majority were treated on the farm (Fig. 10). Stall rest was usually prescribed during the early phase of treatment, but horses that had an SPL in place for several weeks were usually allowed turnout, and some were ridden lightly.

Several simple strategies can be recommended to keep an SPL intact, clean, and protected at home:

- Securing each tape wing on the face with four sutures helps ensure that the wing will stay in place for several weeks when veterinary oversight is infrequent.
- White adhesive tape wraps around the base of each mane braid to help the braids stay tight and keep the SPL tubing close to the crest.
- A plastic bag taped over the injection port assembly keeps the port clean and protected, especially if the catheter cap comes off the catheter. Treatment injections can be done directly through the plastic bag wall, and the bag can be replaced as needed.
- Fly masks provide helpful covers to protect the periocular region and cover the tubing that is exposed on the face. The author rarely uses hoods with hard plastic cups but advises fly masks for the majority of patients.
- Stretchy nylon hoods that cover the head and neck (known as "sleazy sleepwear," made by a variety of manufacturers) may also protect the SPL from stable or pasture trauma.
- Stalls and paddocks should be inspected for any hooks, nails, or other elements that might snare the tubing: all such items should be

removed or taped over. J-shaped hooks at the base of bucket handles should be taped up.
- Solitary turnout is best because pasture mates may pull at SPL elements and damage them.

Even though the technical process of injecting medication through an SPL is simple, many horses are still difficult to treat because of their temperament or ocular pain. A multidose medication schedule can also be confusing. Several "practice tips" are listed below to help caretakers or technicians manage the treatment plan:

- Medication syringes can be labeled with a thin strip of colored tape for easy distinction of different products (Fig. 11).
- A blank "treatment spreadsheet" that is laid out in grid form with spaces for up to six medications should be issued to the caretaker. This can be stored on the ambulatory computer and emailed or pre-printed. A checkbox system will clarify multidrug treatment regimens and keep track of compliance.
- Horses that react strongly when medication contacts the tear film may become more cooperative if a small treat (sliced carrot, peppermint, gingersnap) is given before and after the process.
- Caretaker "burnout" is a threat to successful therapy. Time spent explaining possible consequences of treatment lapses pays off.
- Digital photography is an invaluable owner education and compliance aid.[4] Sharing images with caretakers will inspire the caretakers to continue with a challenging medication schedule, particularly if sequential images demonstrate positive progress.

Finally, clinicians must make good decisions on when to remove the SPL. Generally, all surface ocular problems must be treated until the corneal surface has epithelialized and the stromal bed has begun to fibrose. Recheck visits with careful examination of the cornea, coupled with administration of surface ocular stains, will be required to assess the status of healing. However, a clinician may want to stop therapy but still may be uncertain about whether the condition is truly healed. In these cases, the tube may simply be left in place for an additional 5 to 7 days. If the condition remains stable, the tube then can be removed, but if there are any negative developments, the SPL is in place and can be used again for further treatment.

5. Conclusions

Lavage systems are essential tools for the management of serious ocular disease because they provide an easy, safe means to deliver ocular medications in patients with painful ocular problems. Rational medication selection for individual cases depends on the results of a thorough ocular examination coupled with appropriate diagnostics and any indicated stall-side ocular therapy or procedures. Clinicians who use SPLs to treat major ocular problems in ambulatory practice must have strong skills in ophthalmic examination and have a thorough understanding of corneal disease and ophthalmic pharmacology. Caretakers who use SPLs to deliver complex treatment schedules must dedicate sufficient time to deliver the treatment schedule as prescribed.

Horses tolerate SPLs very well, and the systems may be maintained for months if necessary. The most common complication noted is external trauma to the tube that causes leaks or breaks. Lavage tubing that breaks or develops a hole can be repaired by shortening or splicing the tubing. Lavage system damage from pasture, stall, or patient trauma can be minimized by the use of suture and tape to secure the system to the patient and by external head and neck covers such as fly masks and hoods.

Insertion of an SPL is a simple technique that all equine practitioners can master. This report has outlined numerous tips for installation and management of SPLs that can be implemented in both hospital and ambulatory settings.

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[e]Infu-Disk, Mila International, Erlanger, KY 41018.

How to Diagnose and Manage Horses With Glaucoma

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1. Introduction

Glaucoma is a painful ocular disease that often results in vision loss and can be frustrating to treat.[1–4] Equine glaucoma is most often secondary to chronic, recurrent episodes of intraocular inflammation as occur with equine recurrent uveitis (ERU).[1–8] Diagnosis of glaucoma requires tonometry, or measurement of the intraocular pressure (IOP). Many types of portable, affordable tonometers are available to the veterinary practitioner, and tonometry is becoming more widely used in equine practice. Once a diagnosis of glaucoma has been established, therapy options range from topical and oral medications to advanced surgical procedures. Referral to a veterinary ophthalmologist is appropriate for all cases of equine glaucoma. Determining the cause of the glaucoma and deciding on an appropriate treatment plan is crucial to obtain the ultimate goal of maintaining a comfortable and visual eye. When vision has been lost and IOP is uncontrolled, surgical procedures to ensure long-term comfort, including enucleation, should be considered.

2. Anatomy and Pathophysiology of Glaucoma

Knowledge of normal ocular anatomy allows the practitioner to recognize abnormalities in the equine eye and pathologic changes associated with glaucoma. Immediately posterior to the iris is the ciliary body, which is hidden from view by the iris face even when the pupil is fully dilated. The ciliary body processes produce aqueous humor, the clear intraocular fluid that provides metabolic support to the posterior cornea and the internal structures of the eye. Aqueous humor flows from the posterior chamber, through the pupil, into the anterior chamber, and drains from the eye through the uveoscleral or iridocorneal outflow pathways (Fig. 1).[9] The iridocorneal angle is visible as an intraocular meshwork at the temporal and nasal limbus, with pectinate ligaments extending from the iris root to the corneoscleral junction (Fig. 2).[10,11] Horses are unique in that up to 50% of their aqueous humor drainage is through the uveoscleral (also called "unconventional") outflow pathway.[12,13]

Intraocular pressure is a balance of aqueous humor production and aqueous humor outflow. Glaucoma is a series of intraocular pathologic events culminating in IOP elevation, optic nerve damage, and vision loss. Elevated IOP results in decreased blood flow and optic nerve axoplasmic flow, ultimately resulting in retinal cell death, compression of

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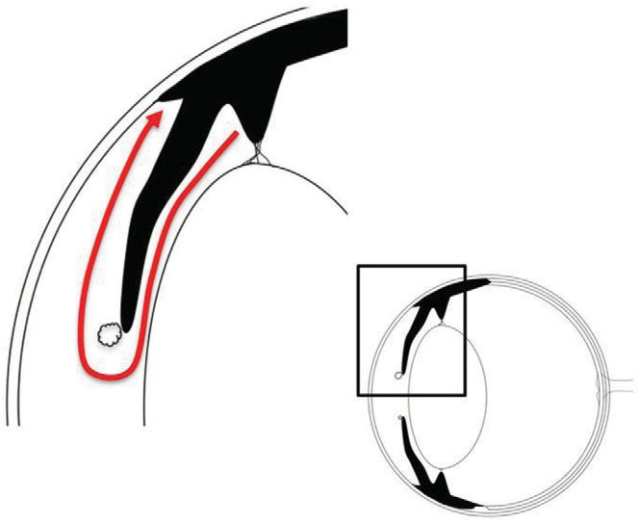


Fig. 1. The equine aqueous humor pathway. Aqueous humor is produced in the ciliary body (shaded), flows into the posterior chamber, through the pupil into the anterior chamber, and then drains through the iridocorneal angle or uveoscleral outflow pathway.

the optic nerve, and blindness.[14] In humans, forms of glaucoma are recognized that result in optic nerve degeneration, retinal degeneration, and vision loss without elevation in IOP. Similar forms of glaucoma have not been documented in the horse; elevation in IOP is thought to be the most important risk factor for glaucomatous optic nerve damage in equine patients.[14,15] Intraocular pressure elevation typically results from obstruction of the aqueous humor outflow pathway. Aqueous humor overproduction has not been reported in any domestic animal species and is not considered to be a cause of glaucoma in the horse.

Glaucoma in the horse is often divided into three categories: primary, secondary, and congenital.

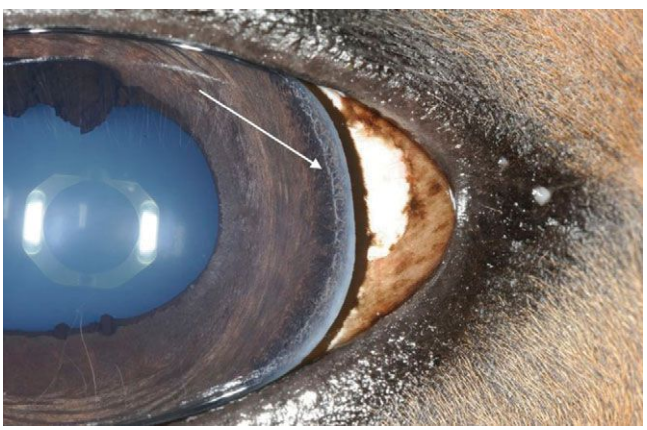


Fig. 2. Photograph of a normal equine eye. The iridocorneal angle is visible just axial to the limbus (white arrow). Normal pectinate ligaments are visible as a meshwork.

Congenital glaucoma is present at birth and is unlikely to respond to therapy.[7,8,16] Primary glaucoma in small-animal species is a heritable, bilateral condition that results from abnormal functioning of the aqueous humor outflow system. Primary glaucoma is infrequently reported in the horse. Glaucoma in horses is most often secondary to a concurrent ocular disease such as ERU, intraocular neoplasia, corneal perforation, or lens luxation.[2–4,17] Although ERU is widely regarded as the most common cause of glaucoma in horses, the mechanism by which ERU leads to secondary glaucoma is poorly understood.[4]

3. Examination Techniques

The minimum equipment necessary for any equine ophthalmic examination includes a Finhoff transilluminator, magnification head loupe, fluorescein stain, a tonometer, tropicamide,[a] and a retinoscope (such as a direct ophthalmoscope or hand lens). If possible, the horse should be observed navigating in its environment before sedation. An obstacle course can be created with the use of large objects in the barn (such as trash cans and poles on the ground) to allow the practitioner to more critically assess vision. Menace responses and dazzle reflexes should also be observed before sedation. Ophthalmic examination should be performed in a dimly lit location to maximize detection of subtle lesions. Sedation with detomidine hydrochloride[b] or xylazine hydrochloride[c] and an auriculopalpebral block are important for completion of a complete examination. Butorphanol tartrate[d] is not recommended because it tends to result in jerky, spontaneous head movements.

Tonometry is recommended for all horses with clinical signs of ophthalmic disease. Whereas sedation with α-2 agonists can decrease IOP readings, an auriculopalpebral block has no effect on IOP.[18,19] Head position does have a critical effect on IOP; lowering the head below the heart causes significant elevations in IOP.[20] It is important to maintain a head position level with or above the heart while performing tonometry. Consistency is recommended when performing serial IOP measurements, with the use of the same sedation, nerve block, tonometer, and examiner each time to achieve maximal comparability between measurements. Careful attention to technique is also important. Any excessive pressure on the globe may artificially increase IOP.

Two types of tonometers are commercially available for use in the horse: rebound and applanation.[21–23] The rebound tonometer[e] (Fig. 3) uses a magnetic probe that is projected at the cornea. The rebound tonometer is handheld, battery-operated, and does not require daily calibration. The noise emitted from the machine while taking the required six readings is very quiet and not likely to disturb the horse, even if not sedated. The readings are averaged within the machine and displayed



Fig. 3. Photograph of rebound tonometry being performed in a patient with glaucoma. Note that the tonometer is held perpendicular to the ground, and the practitioner is using caution to avoid excessive digital pressure on the globe.

in an easily viewed window opposite the probe. Topical anesthesia is not required. The mean intraocular pressure with the use of a TonoVet is 22.1 ± 5.9 mm Hg, with a range from 10 to 34 mm Hg.[23]

Multiple applanation tonometers are commercially available[f,g] (Fig. 4). Applanation tonometers differ from the rebound tonometer in that they measure the force required to flatten the corneal surface. All of the devices are handheld and portable. Applanation tonometers require a latex cover to protect the tip of the device. Both of the listed applanation tonometers require use of topical anesthesia to obtain accurate readings. One advantage of an applanation tonometer is that it can be used with the patient in any position, unlike the rebound tonometer, which must remain perpendicular to the patient with the magnetic probe parallel to the ground. The mean intraocular pressure with the use of a Tono-Pen is 21.0 ± 5.9 mm Hg, with a range from 9 to 33 mm Hg.[23]

Table 1. Clinical Signs of Equine Glaucoma

Acute signs
Vision loss
Epiphora
Blepharospasm
Corneal edema
Conjunctival hyperemia
Mydriasis
Chronic signs
Vision loss
Epiphora
Blepharospasm
Buphthalmos
Corneal edema
Descemet's striae
Corneal ulcers
Lens luxation
Mydriasis
Changes in the appearance of drainage angle (collapse, fibrosis)
Retinal degeneration
Optic nerve cupping

4. Clinical Signs of Glaucoma

The clinical signs of glaucoma vary.[1–3,7,8,17,24] Many horses with glaucoma do not exhibit the classic signs of ocular pain, including blepharospasm and epiphora. Acute signs often differ from chronic signs (Table 1).

Glaucomatous horses exhibit variable pupillary light reflex deficits. Glaucoma generally results in a mydriatic pupil caused by effects on the optic nerve and iris sphincter muscle, but horses with concurrent intraocular inflammation may have miotic pupils instead. Dyscoria, an abnormally shaped pupil, may result from posterior synechiae (adhesions between the iris and the lens capsule) (Fig. 5). Posterior synechiae may result in pupil size being discordant with concurrent ocular disease (ie, a miotic pupil in an end-stage glaucoma eye).



Fig. 4. Photograph of applanation tonometry being performed in a patient with glaucoma. Note that the tonometer can be held in any plane, and the practitioner is using caution to avoid excessive digital pressure on the globe.



Fig. 5. Photograph of the right eye of a horse with end-stage ERU. Note the small globe size, corneal fibrosis, dyscoria caused by posterior synechiae, and cataract. This eye is not visual and has no potential to regain vision.



Fig. 6. Photograph of the left eye of a horse with glaucoma. Note the diffuse corneal edema and bullae (fluid-filled vesicles or "blisters" that can be present with severe corneal edema).

Conjunctival hyperemia and episcleral injection is common in glaucomatous horses. Mild to severe diffuse corneal edema is observed frequently. Corneal edema may be focal or begin as a vertical stripe across the cornea, then progress to diffuse edema (Fig. 6). Edema results from dysfunction of the endothelium and physical distortion of corneal stromal fibers. Descemet's striae (also termed "corneal striae" or "Haab's striae") are areas of discontinuity in Descemet's membrane and may later appear at these sites of linear edema traversing the cornea (Fig. 7). Severe corneal edema may be associated with fluid-filled subepithelial blisters or bullae. Bullae may rupture and result in corneal ulceration.

Corneal vascularization may be superficial, perilimbal, and extend several millimeters from the limbus toward the axial cornea. Keratic precipitates (pinpoint conglomerates of fibrin and inflammatory cells) may be present on the corneal endothelium.



Fig. 7. Photograph of the left eye of a horse with chronic glaucoma. Descemet's striae are visible, traversing the cornea as white curvilinear lines.

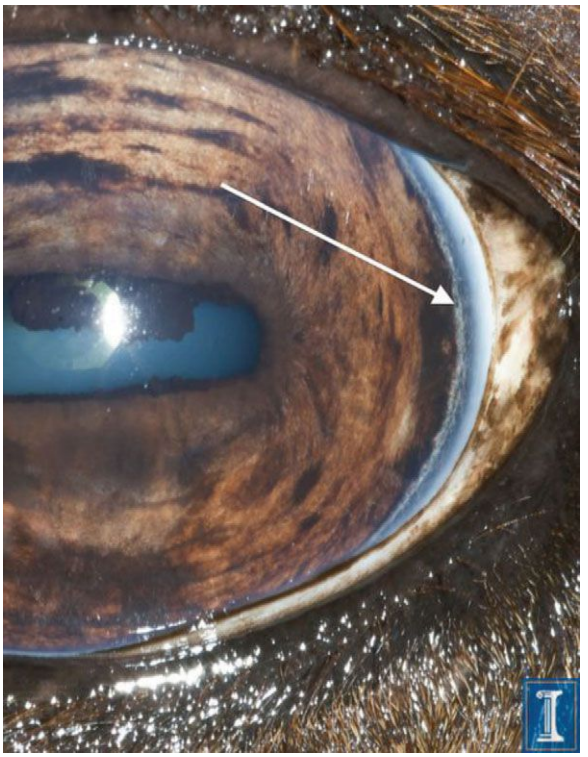


Fig. 8. Photograph of the left eye of a horse with chronic glaucoma. The iridocorneal angle is collapsed with marked fibrosis (white arrow).

Keratic precipitates are often difficult to visualize without magnification.

The diffuse corneal edema, frequently present in horses with glaucoma, makes intraocular examination challenging. Performing the exam in a dark area and directing a light source across the anterior chamber from limbus to limbus facilitates visualization of intraocular structures. Common intraocular exam findings may include clinical signs seen with ERU, including iridal hyperpigmentation, atrophy of the corpora nigra, posterior synechiae, dyscoria, aqueous flare, and cataract. The iridocorneal angle is most visible laterally, in which the opening of the trabecular meshwork is visible just axial to the limbus. Pectinate ligaments are visible as a meshwork traversing the opening of the iridocorneal angle. Abnormal iridocorneal angles may be sclerotic, with a band of white fibrous tissue replacing the normal pectinate ligaments, or narrowed (Fig. 8). Appearance of the iridocorneal angle has not been correlated with the onset of glaucoma or prognosis in horses. Lens subluxation or luxation is usually a result of chronic expansion of the glaucomatous globe (termed "buphthalmos") and subsequent zonular stretching and degeneration (Fig. 9).

In the early stages of glaucoma, the appearance of the retina may be normal. With time, the optic nerve may become atrophic, pale, and "cupped" in appearance, as if the center of the optic nerve was



Fig. 9. Photograph of the left eye of a horse with chronic glaucoma. Note the complete anterior lens luxation of a cataractous lens with a mydriatic pupil.

being pushed away from the viewer. Retinal blood vessels may appear absent or attenuated. Chronic elevations in IOP may lead to expansion in the size of the globe, termed buphthalmos.

5. Treatment of Glaucoma

The goal of glaucoma treatment is maintaining IOP in a range that prevents continuing optic nerve and retinal damage. The "ideal" IOP is unknown, but most clinicians agree that <25 mm Hg would be acceptable. Because IOP is a balance of aqueous humor production and drainage, all glaucoma treatments are aimed at one of two targets: reducing aqueous humor production or increasing IOP drainage. Glaucoma treatment can be divided into medical and surgical therapies; medical therapy is typically the first-line treatment. The underlying causes of glaucoma, such as ERU, may also require concurrent treatment. In addition to topical antiglaucoma medications, such cases may also need topical and systemic anti-inflammatories. The use of atropine in cases of concurrent ERU and glaucoma is controversial because atropine may be associated with elevations in IOP.[3,25,26] Atropine should only be used in cases of glaucoma when IOP can be closely monitored.

Medical therapy is generally the first line of treatment for glaucoma.[27] Drug classes that reduce aqueous humor production include β-adrenergic antagonists and carbonic anhydrase inhibitors (CAIs). β-Adrenergic antagonists, also called β-blockers, inhibit cAMP activity in the ciliary body. Timolol maleate 0.5%[h] is the most commonly used drug in this class. It has been shown to reduce IOP in clinically normal horses.[28] Other commercially available β-adrenergic agonists include betaxolol, levobunolol, and metipranolol. These drugs are widely used and have not been evaluated in horses. β-Adrenergic agents should be applied every 8 to 12 hours.[28]

Carbonic anhydrase inhibitors inhibit carbonic anhydrase, an enzyme in the ciliary body epithelium that is required for aqueous humor production. Commercially available CAIs include brinzolamide 1%[i] and dorzolamide 2%.[j] Both have been demonstrated to reduce IOP in clinically normal horses, although brinzolamide results in slightly greater reduction.[29,30] Generic dorzolamide is affordable and widely available, which has made it more popular for the treatment of equine glaucoma in the United States. Dorzolamide 2% and timolol 0.5% solution is also available as a combination drug. The efficacy of a dorzolamide/timolol combination versus dorzolamide alone versus brinzolamide alone has not been evaluated in horses with glaucoma. Topical CAIs should be administered every 8 to 12 hours.[29,30] Acetazolamide, an oral CAI, is frequently used as a potassium-wasting diuretic in horses with hyperkalemic periodic paralysis at doses of 2 to 3 mg/kg PO every 12 hours.[31] The efficacy of this drug in decreasing IOP is unknown; however, it is available as an oral formulation. This makes it advantageous for use in patients in which topical administration of a solution is not possible.

Few drugs are available for increasing IOP outflow. Prostaglandin analogues include lantanoprost 0.005%[k] and travaprost 0.004%.[l] This drug class results in miosis and increased IOP outflow in other species but is not consistently effective in the horse.[32] Complications are reported with use of prostaglandin analogues, including increased ocular pain and intraocular inflammation.[32] A high rate of complications, including increased ocular pain, is reported. This drug class should be used with great caution in horses with glaucoma associated with ERU.

Surgical therapy for glaucoma can either increase aqueous outflow or decrease production. The most common surgical procedure is cyclophotocoagulation (laser ablation of the ciliary body epithelium), which results in decreased aqueous humor production.[1,3,33–36] The procedure can be performed transclerally or endoscopically with the use of a diode laser that preferentially targets pigmented tissue such as the ciliary body epithelium. Complications of cyclophotocoagulation include corneal ulceration, uveitis, hyphema, cataract formation, and retinal detachment. Continued topical medications are often necessary despite surgical intervention.[1]

Surgical therapies for glaucoma were previously limited to those decreasing aqueous humor outflow. Recent evidence supports the use of drainage implants that direct aqueous humor subconjunctivally.[37] Ahmed valved shunts have been placed experimentally with good success. Clinical trials are underway evaluating the use of this type of drainage implant in clinical glaucoma patients.[37]

When glaucoma therapy fails, the patient is left with a painful, blind globe, and eliminating ocular pain is essential for the welfare of the horse. Enucleation is one therapy to eliminate glaucoma pain in a blind eye. Surgical alternatives to enucleation include placement of a cosmetic intraocular or in-



Fig. 10. Photograph of a chemical ciliary body ablation performed on the left eye of a horse with vision loss and elevated IOP secondary to chronic glaucoma.

traorbital prosthesis.[38,39] Chemical ciliary body ablation can be used to decrease aqueous humor production and induce shrinkage of the globe (phthisis bulbi).[40] Intravitreal injection of 50 to 75 mg of gentamicin sulfate results in necrosis of the ciliary body with long-term reduction of IOP. Intravitreal injection can be performed in the sedated horse with the use of topical anesthesia; a retrobulbar block may be necessary. After the application of topical anesthesia, a 22- to 25-g needle should be positioned 10 to 12 mm posterior to the dorsolateral limbus and aimed toward the optic nerve (Fig. 10). Ensuring the appropriate angle of the needle is essential to avoid lacerating the posterior lens capsule during intravitreal injection, because intractable uveitis probably will result. If IOP is elevated at the time of injection, aqueous paracentesis should be performed first to lower IOP into a normal range and prevent further IOP elevation. Tonometry should be performed after injection, and, if the IOP is still elevated, the aqueous paracentesis should be repeated.

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[a]Mydriacyl, Alcon Laboratories, Fort Worth, TX 76134.
[b]Dormosedan, Orion Corporation, Espoo, Finland.
[c]Rompun, Bayer Animal Health Division, Shawnee, KS 66216.
[d]Torbugesic, Fort Dodge Animal Health, Fort Dodge, IA 50501.
[e]TonoVet, Icare Finland Oy, Helsinki, Finland.
[f]Tono-Pen XL, Reichert, Depew, NY 14043.
[g]Tono-Pen AVIA, Reichert, Depew, NY 14043.
[h]Timolol maleate, Bausch & Lomb Pharmaceuticals Inc, Tampa, FL 33637.
[i]Azopt, Alcon Laboratories, Fort Worth, TX 76134.
[j]Trusopt, Merck, West Point, PA 19454.
[k]Travatan, Alcon, Irvine, CA 92618.
[l]Xalatan, Pfizer, New York, NY 10017.

How to Diagnose the Cloudy Eye

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1. Introduction

Diagnosing the cloudy equine eye is often met with trepidation in the field setting because of the variety of differentials for this condition, many of which are clinically difficult to distinguish. Most equine veterinarians feel comfortable with diagnosis and treatment of uncomplicated corneal ulceration because of its high prevalence in equine practice. However, the cloudy eye with intact epithelium is a less common complaint and can prove to be a diagnostic challenge. Furthermore, accurate diagnosis is vital. Treatment for one condition is often contraindicated for another, particularly with regard to the use of corticosteroids. The importance of accurate diagnosis and treatment is accentuated by the fact that maintenance of a clear visual axis is often a necessity for the career of the equine patient. Equine eyes have the propensity to degenerate rapidly, leaving very little room for error. The objective of this report is to present a stepwise approach to diagnosis of the cloudy equine eye. Correct diagnosis will allow accurate treatment.

The disease processes that will be discussed include the following:

- Stromal abscess
- Calcific band keratopathy
- Eosinophilic keratitis
- Nonulcerative keratouveitis
- Onchocerciasis
- Squamous cell carcinoma
- Subepithelial keratomycosis
- Immune-mediated keratitis
- Equine recurrent uveitis
- Glaucoma

2. Materials and Methods

For the initial ophthalmic examination, the same basic tools are needed as for any ophthalmic ambulatory procedure.

- Fluorescein stain
- Mepivicaine or lidocaine for local nerve blocks
- Chemical restraint: α_2-agonist sedation (xylazine, detomidine)
- Focal light source with cobalt-blue filter
- Dimly lit area

Step 1: Is the Eye Painful?

Examination should start with a patient who has not yet been sedated and whose eyelids have not

NOTES

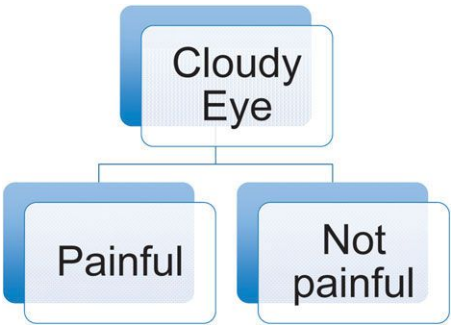


Fig. 1. Determining the degree of ocular pain.

been blocked, to allow accurate assessment of ocular pain. Hallmark clinical signs of ocular pain include epiphora and blepharospasm. Determining the degree of ocular pain is important for formulation of differential diagnoses of corneal opacity (Fig. 1). After gross assessment for pain through a visual examination, the function of cranial nerves II, III, V, and VII is assessed by the menace response, dazzle reflex, pupillary light reflexes (direct and indirect), and palpebral reflex. Pupil size, shape, and symmetry between right and left pupils should be evaluated. Miosis, mydriasis, and anisocoria may occur coincident with corneal disease, the most common examples being when the corneal opacity is associated with anterior uveitis, synechia, or glaucoma.

Step 2: Is There an Ulcer?

After assessment of vision and reflexes, the horse can be sedated and perineural anesthesia can be performed if needed. If corneal culture is warranted, that should occur before instilling any topical solutions into the eye. The eye must be stained with fluorescein, even if it does not appear that a corneal ulcer is present. The only way to determine fluorescein stain uptake accurately is with use of a cobalt-blue filter light. Use of a focal light source is key to careful examination of corneal opacities. A systematic approach should be used every time, so that no area is missed, even if the lesion is immediately apparent. Start at the limbus and examine circumferentially, moving axially toward a more central part of the cornea as you proceed. Often dimming the focal light source and taking breaks will improve the tractability of the patient. Be sure to examine both eyes.

Step 3: What Color Is the Opacity?

The cornea readily displays pathologies as the result of its biologically clear appearance. Furthermore, it has a limited number of characteristic reactions to disease. The ability to visually recognize these reactions allows for accurate differentiation of keratopathies. The following is a list of the equine cornea reaction to disease accompanied by its clinical appearance:



Fig. 2. Corneal edema.

- Edema: hazy gray-blue, can be focal or diffuse (Fig. 2)
- Vessels: originate from the limbus
- Perfused or ghost
- Depth indicates location of inciting lesion
- Superficial–branching
- Deep–hedge
- Fibrosis: gray, may note associated ghost vessels
- Inflammatory cell infiltrate: yellow to green discoloration
- Lipid or mineral infiltrate: shiny white, crystalline (cholesterol or calcium)

It is vital to be able to recognize these processes when examining an eye with corneal disease.

Step 4: Making the Diagnosis

(1) Diseases Generally Characterized by a Nonpainful Eye

If initial assessment shows the patient to have normal ocular comfort, the differential diagnosis list is significantly shortened. Any disease that has a component uveitis is excluded from this category, because the condition is typically painful (Fig. 3).

(a) Immune-mediated keratitis (IMK) should be considered as a differential for a nonpainful eye with corneal vascularization.[1] In IMK, vascularization depth generally corresponds to the depth of the lesion. There are often striking corneal changes while the eye is quiet and comfortable (Fig. 4). The specific pathogenesis of this disease is not known, and the clinical presentation can be highly variable. The unifying characteristics of the disease are the ocular comfort of the patient and the lesion response to immunosuppressive therapy.

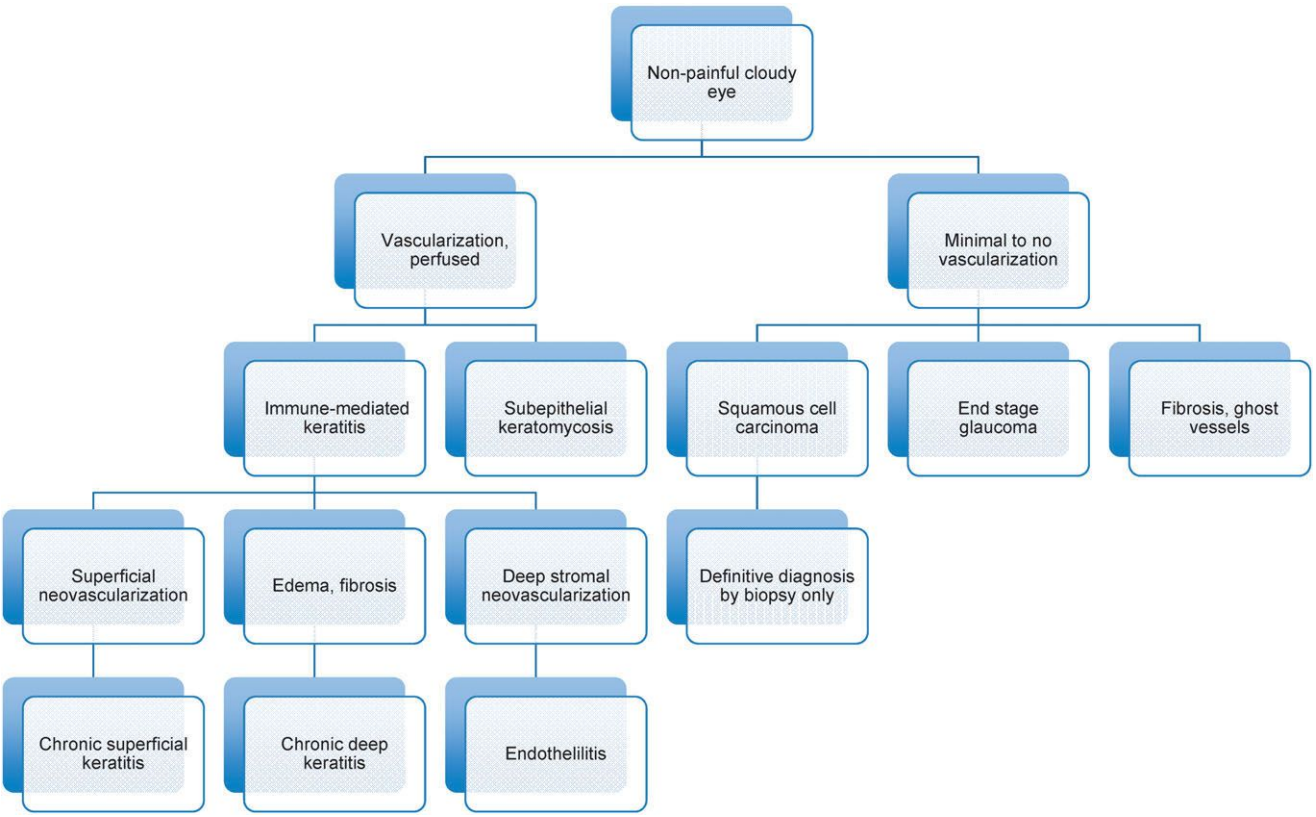


Fig. 3. Differentials for the non-painful, cloudy eye.

Ruling out an infectious cause through culture or empirical antimicrobial therapy is important when making this diagnosis because the cornerstone of therapy for IMK is immunosuppression with corticosteroids.

(b) Similar in appearance to IMK but of infectious origin is subepithelial keratomycosis.[2] Horses with this disease only exhibit mild to no blepharospasm. Hallmark corneal changes are multifocal punctate to geographic cellular infiltrate opacities that resolve with anti-fungal therapy. Culture results of these lesions are variable. Therefore, it is important to rule out an infectious disease such as this, for example, with initial treatment of empirical antimicrobial therapy before treating for immune-mediated disease.



Fig. 4. IMK of manifests with striking corneal changes.

(c) Another differential for a nonpainful corneal opacity is the most common tumor of the equine cornea—corneal squamous cell carcinoma.[3] This can originate from the cornea, conjunctiva, or limbus. Although lesions commonly appear nodular and elevated, some may appear simply as stromal infiltrate (Fig. 5). Notably, all of the forms should cause little to no discomfort. When squamous cell carcinoma is high on the differential diagnosis list, biopsy is needed for diagnosis. This may be in the form of keratectomy under general anesthesia if the lesion is smooth and cannot be grasped for easy removal.

(d) End-stage glaucoma can manifest as a comfortable, cloudy eye. The corneal opacity in this case is usually edema.[4] This corneal change is often accompanied by other gross changes such as buphthalmos and striae, and the eye is usually blind.

(e) Despite its chronicity, fibrosis may be newly noted by a client or seen on initial examination such as during a pre-purchase exam. Corneal scars vary



Fig. 5. Some neoplastic lesions appear as stromal infiltrate.

significantly in size and density. The deeper the injury, the denser the scar; they should appear white, consolidated, and smooth, with no ocular discomfort (see Fig. 6).

(2) Diseases Generally Characterized by a Painful Eye

If the eye is painful, a wide variety of conditions are possible (Fig. 7).

(a) Both eosinophilic keratitis and calcific band keratopathy appear as raised corneal lesions. However, they can be differentiated on the basis of cytology and location. Eosinophilic keratitis is characterized by numerous eosinophils on cytology and is typically limbal[5] (Fig. 8). Conversely, calcific band keratopathy should be both negative for eosinophils and infectious causes and is typically distributed in the interpalpebral fissure.

(b) For a smooth, intact epithelium, differentiating between a focal lesion and segmental disease can sometimes be difficult. Anterior uveitis is often secondary to discrete opacities created by nonulcerative keratouveitis (NUKU)[6] and stromal abscessation (Fig. 9).[7] Severe, diffuse corneal edema may mask the whitish to yellow cellular infiltrate that is characteristic of these diseases. This infiltrate may be located in any area of the cornea and at any depth. Nonspecific signs of ocular pain and severe inflammation are concurrent. The gold standard for diagnosis and differentiation of both diseases is histopathology; however, this requires general anesthesia. Therefore, often the diagnosis may be made by response to therapy because the intact epithelium usually prevents adequate cytology and culture.



Fig. 6. Scars should appear white, consolidated, and smooth, with no ocular discomfort.

(c) A differential for the cloudy, painful eye with diffuse or focal corneal edema is uveitis. Anterior uveitis alone without primary corneal disease may manifest as the result of equine recurrent uveitis[8] or ocular trauma. In these cases, the predominant corneal change is diffuse corneal edema. Extensive neovascularization or cellular infiltrate should not be present. Clinical signs of note are miosis, often severe, and circumlimbal vascularization. Careful inspection of the quality, opacity, and coloration of the cornea should allow for differentiation.

(d) Early glaucoma is another differential for painful, diffuse corneal edema, especially that which does not respond to anti-inflammatory medication. In the early, acute stage, the eye may be painful. Measurement of intraocular pressure is the gold standard for diagnosis of this disease and will be discussed later. However, a sign to differentiate this edema from that of uveitis without the ability to measure pressure is through pupil size. A uveitic pupil is most often miotic, whereas an early glaucoma pupil without synechia should be at least slightly mydriatic. Often, vertical corneal edema with linear band opacities is noted.

(e) Finally, onchocerciasis may also appear as a painful opacity usually located limbally with accompanying signs of corneal inflammation (edema, vascularization). The most common concurrent clinical sign, conjunctivitis, distinguishes it from other corneal infiltrates.[9] Definitive diagnosis is made through a conjunctival snip biopsy.

Step 5: Further Diagnostics

Once the initial examination has been performed, further diagnostics are often warranted for definitive diagnosis.

(a) Applanation or rebound tonometry. This is needed to accurately diagnose glaucoma and uveitis by providing an objective measurement of intraocular pressure. Tonometry facilitates early diagnosis of glaucoma, which is often subtle. Comparison between globes within a horse as well as comparison of published reference ranges is helpful. This diagnostic is vital to monitoring response to treatment for glaucoma.

(b) Culture and cytology. Culture and cytology are a frustrating aspect to nonulcerative corneal disease. Because of the intact epithelium, a diagnostic sample is difficult to obtain. Cytology is a requirement for diagnosis of certain conditions (eosinophilic keratitis). It also can guide therapeu-

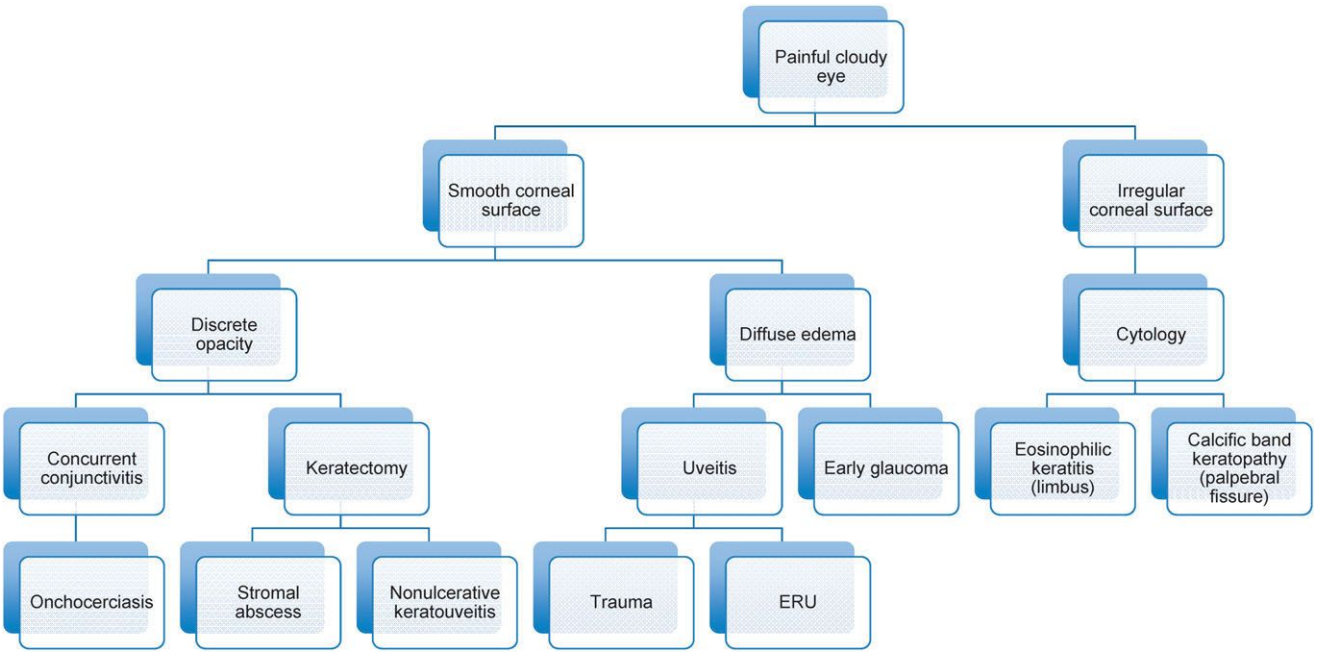


Fig. 7. If the eye is painful, a wide variety of conditions are possible.

tic choices even if definitive diagnosis cannot be obtained. Therefore, an attempt at culture and cytology can be made with the knowledge that an inconclusive sample or sterile culture is not definitive. Culture can be obtained with the use of a sterile cotton-tipped applicator culturette on the corneal surface. After this, cytology can be obtained after the application of topical anesthetic agent (proparicaine, tetracaine) with the use of the blunt end of a sterile scalpel blade. Gently scrape the margins of the opacity, and smooth the sample onto glass slides. The slides should then be stained before analysis. If scraping of the lesion for cytology has created ulceration caused by poorly adherent epithelium, a second culture should be obtained of the exposed stroma.



Fig. 8. Eosinophilic keratitis is characterized by numerous eosinophils on cytology and is typically limbal.

(c) Biopsy. Biopsy of corneal tissue should only be performed with extreme caution. Rupture of the globe as the result of structural instability is more common in equids than other veterinary species. Globe rupture is a surgical emergency, and eyes can only be saved if prompt surgical intervention is performed. A proliferative, nonpainful lesion is the best candidate for corneal field biopsy.

(d) Referral for keratectomy. This procedure requires heavy sedation or general anesthesia. It is indicated both for diagnosis of a deep stromal opacity through histopathology but also is a treatment to remove neoplastic or infected corneal tissue.

The importance of an accurate diagnosis is highlighted by the decision to treat the eye with topical corticosteroids. Corticosteroids have been shown to decrease vascularization and limit potentially blinding anterior uveitis.[10] Conversely, they also inhibit epithelial regeneration and can predispose the ocular surface to infectious colonization.[11–13] It is veterinary dogma never to use a topical steroid when a corneal ulceration is present. However, other contraindications also exist. An infection may still be present because of the difficulty to obtain a positive culture through the intact epithelium. Furthermore, horse eyes appear to be more susceptible to the adverse effects of topical corticosteroids and infectious colonization. If a culture cannot be obtained and diagnosis is open, initially, presumptive treatment with topical antimicrobials



Fig. 9. Severe inflammation and pain accompany stromal abscessation.

is indicated. If the eye fails to respond, corticosteroids then may be justified with caution. Finally, frequent reassessments are vital because eyes are highly dynamic, and clients may not perceive subtle changes in disease processes.

3. Results

When a systematic and consistent approach is used to examine and diagnose ocular lesions, you are less likely to miss subtle clinical signs and prescribe inappropriate therapy. Once the correct diagnosis is made, searching for current therapies is simplified. Inappropriate use of corticosteroids can be disastrous in the equine eye, and therefore careful examination and thought should be given before their administration.

4. Discussion

Characterizing a lesion on the basis of the hallmark corneal changes as well as the use of a stepwise, logical approach allows for the more accurate diagnosis of corneal disease. Ideally, all corneal lesions would be examined through the use of slit-lamp biomicroscopy. This allows for a more precise assessment of depth and composition by means of microscopic inspection. However, the cost and learning curve associated with these devices typically precludes their use for the general practitioner. Instead, the general practitioner is often put in a position of doing an in-depth corneal examination with limited tools and must therefore be careful not to miss any subtle clinical signs. Referral should always be offered, especially in cases in which the diagnosis is not straightforward or the eye fails to respond to therapy.

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How to Perform a Standing Enucleation

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1. Introduction

Enucleation is one of the most frequently performed orbital surgeries.[1] Indications for enucleation include a blind, painful eye; ocular neoplasia; severe corneal or intra-ocular infection; and ocular trauma not amenable to surgical repair. Several publications recommend that equine enucleation only be performed under general anesthesia.[2,3] However, per several recent reports, enucleation can be performed safely on standing horses with appropriate sedation and local anesthesia.[4,5]

The option to perform a standing enucleation allows the surgeon to avoid the risks of general anesthesia including fractures, myositis, and neuropathies. Ocular surgery has been associated with an increased incidence of movement and hypotension during anesthesia followed by difficult or prolonged recovery.[6] These complications probably occur as the result of the deeper plane of anesthesia required to diminish ocular sensation and mobility. Geriatric patients, horses with orthopedic disease, or heavy draft horses therefore might be better candidates for a standing enucleation than an enucleation performed under general anesthesia.

2. Materials and Methods

Case Selection

Not every horse is an appropriate candidate for standing surgery. The horse's temperament or an extremely painful eye might not allow a standing procedure. During the initial assessment of the horse, if the horse will not tolerate an ocular examination after sedation, local nerve blocks, and application of a topical anesthetic, it probably will be difficult to perform enucleation as a standing procedure. Also, an enucleation for neoplasia that requires extensive resection and advanced reconstructive procedures is better performed under general anesthesia.

Positioning

The horse should be placed in stocks. Care must be taken to pad the cheek pieces of the halter with towels or foam to prevent pressure and damage to the facial nerve during surgery. An effective head support can be constructed by inverting the lid of a 50-gallon trash can and setting a foam pad on its top (Fig. 1). The padding prevents trauma to nerves within the muzzle. In the field, a headrest can also

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Fig. 1. The "table" was created with the use of foam pads and a trash can, on which the horse's head is placed during surgery.

be created with hay bales covered with towels. Cross-ties can be used to position the head. However, the cross-ties often hinder the surgeon's movement, and the halter cheek piece could be pulled into the surgical site. Portable dental rests can be helpful, but some may tip or fall over if the horse lifts its head.

Preoperative Medications and Sedation

An intravenous (IV) catheter is placed in the jugular vein. Flunixin meglumine (1.1 mg/kg IV), gentamicin (6.6 mg/kg IV), and potassium penicillin (22,000 units/kg IV) are administered before surgery. A bolus of detomidine (0.01–0.02 mg/kg IV) and butorphanol (0.01–0.02 mg/kg IV) are administered. Although systemic opioids have been associated with sudden head movements,[3] their addition can reduce responses to touch and provide better antinociception.[7] Muzzle tremors are often not noted unless high doses of opioids are used.[7] Detomidine is then diluted (10 mg detomidine/250 mL lactated Ringer's solution or 0.9% sodium chloride) for use as a constant-rate infusion. Depending on the horse's level of sedation after the initial bolus, the detomidine infusion is initiated at a rate of 0.2–0.4 μg/kg per minute for the first 15 minutes and then decreased to 0.1–0.2 μg/kg per minute for the next 15 minutes. The rate is then typically maintained at 0.1 μg/kg per minute for the duration of the procedure. With the use of a 0.04-mg/mL dilution of detomidine, an infusion rate of 100 mL/h (25 drops per minute for a 15–drops/mL IV set) would be equal to an approximately 0.1–μg/kg per minute dose for a 500-kg horse. If additional sedation is required, the rate can be increased, but typically not more than 0.6 μg/kg per minute is required.[8] The detomidine infusion allows for a more consistent level of sedation without increased levels of ataxia or other side effects.[7] Clipping or injecting local anesthetics should not be performed until the appropriate level of sedation has been reached.[7] If tolerated, cotton balls can be placed in the ears to decrease stimuli from sounds in the environment.

Local Anesthesia

Before local anesthetics are administered, the periocular area should be clipped and prepared for surgery with a 1:50 betadine solution. Before preparing the ocular surface, local nerve blocks should be performed.[9] A retrobulbar nerve block is performed with the use of a 22-gauge, 3.5-inch spinal needle. The orbital fossa is palpated just caudal to the posterior aspect of dorsal orbital rim. The needle is introduced perpendicular to the skin surface and advanced to the extraocular muscle cone (Fig. 2). As the needle is advanced through the extraocular muscles, the eye will deviate slightly dorsally, and a "popping" sensation can be palpated. The plunger is then pulled back to ensure that the needle is not positioned within a vessel; 10 mL of a 50:50 lidocaine and bupivacaine mixture is then injected. This combination is used for its rapid onset and good tissue penetration (lidocaine) and the prolonged duration of action, up to 8 hours, of bupivacaine.[10] The block should take effect within 5–10 minutes. Frontal and palpebral nerve blocks are performed with the same 50:50 lidocaine and bupivacaine mixture. A ring block along the orbital rim is performed to ensure adequate analgesia of the peri-orbital skin. A topical anesthetic (0.5% tetracaine viscous ophthalmic solution) is then applied to the corneal surface to further improve corneal and conjunctival anesthesia. Tetracaine ophthalmic solution is preferred over proparacaine ophthalmic solution because tetracaine better anesthetizes the equine cornea.[11] A 2.5% phenylephrine ophthalmic solution is applied to the ocular surface to constrict conjunctival vessels and thereby decrease bleeding and increase the duration of action of the topical anesthetic. Routine preparation of the globe for surgery is then completed.

Surgery

A transpalpebral or transconjunctival enucleation can be performed. The presence of infection or neoplasia warrants the transpalpebral approach. Oth-

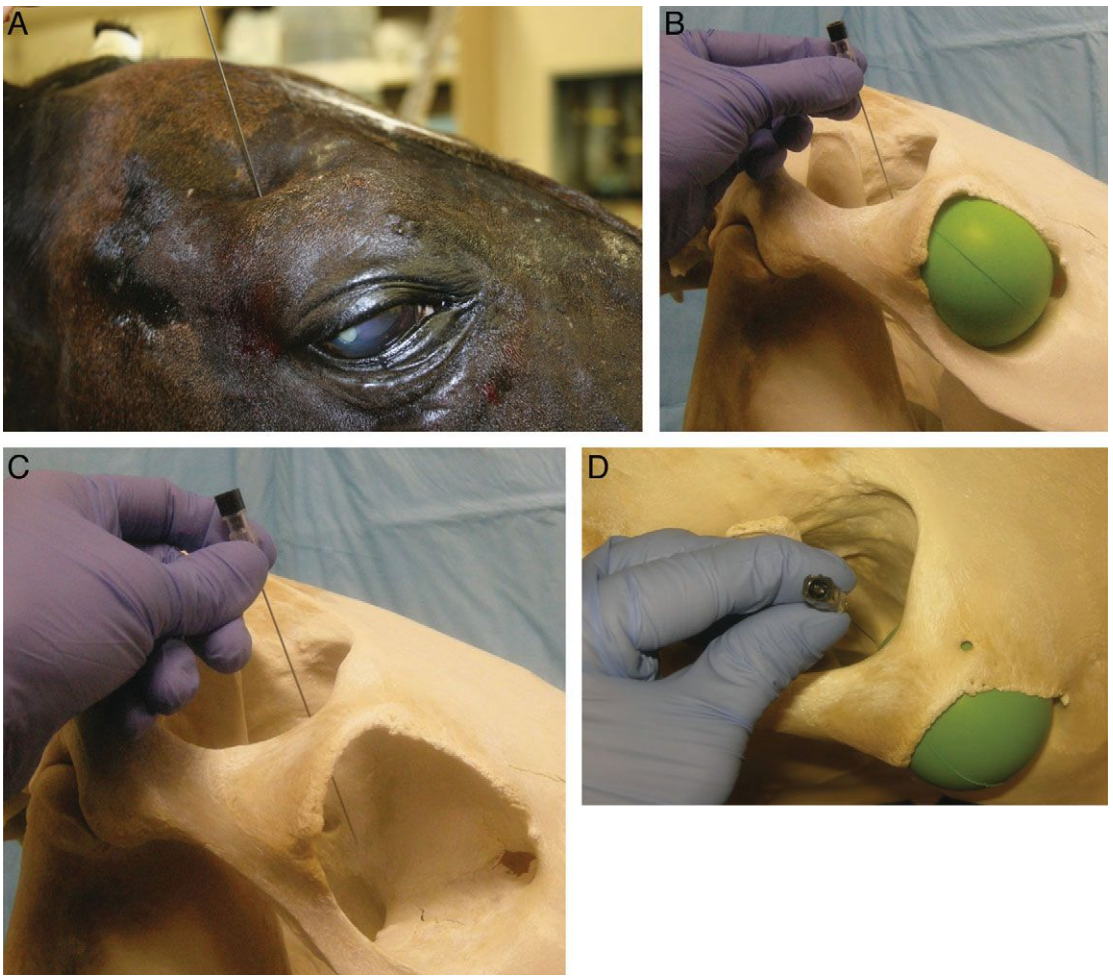


Fig. 2. A–D, Proper needle placement for the retrobulbar nerve block.

erwise, the transconjunctival approach is simpler and allows better visualization with less bleeding throughout the procedure. A drape should be placed over the surgical site. However, some horses, despite sedation, do not tolerate having the ears or contralateral eye covered with the drape. The towel clamps to secure the drape should be placed either within areas that were blocked or hooked to the halter because other facial skin has not been anesthetized. For the transconjunctival approach, an eyelid speculum is used to improve visualization. To better secure the speculum, it can either be supported by an assistant or the hinge can be hooked to the halter with a towel clamp. If the horse becomes reactive during the procedure, the rate of the detomidine infusion can be increased, a bolus of butorphanol or detomidine can be administered, or additional local anesthetic can be infiltrated into the sensitive area. The enucleation is performed the same as under general anesthesia.

Postoperative Care

The detomidine infusion is discontinued. Gauze pads (4 × 4) are placed over the incision site and covered with a stockinette (6-inch) with holes cut out for the ears and remaining eye. The stockinette is secured with an elastic adhesive bandage in a figure-of-8 pattern around the ears and contralateral eye. Usually within 10–20 minutes of discontinuing the detomidine infusion, the horse is able to return to the stall. Flunixin meglumine (1.1 mg/kg IV or PO q 12–24 hours) is continued for 3–5 days after surgery. Antibiotics are used as needed on a case-by-case basis. The bandage is typically removed 1 day after surgery. The horse is usually discharged the day after surgery. Sutures are removed in 10–14 days.

3. Results

Enucleation can be safely and effectively performed in the standing horse with adequate sedation, sufficient local anesthesia, and appropriate restraint. Whereas complications such as orbital abscess formation, retrobulbar hemorrhage, and stimulation of the oculocardiac reflex have previously been reported with the retrobulbar nerve block,[12] complications are rare, and the author has not seen any complications. In the author's experience, place-

ment of surgical drapes and maintenance of a sterile surgical field are slightly more challenging than in the recumbent patient. However, the author has performed more than 30 standing enucleations, with no increase in complication rate compared with enucleations performed under general anesthesia. Most horses stand quietly for the procedure, but if the horse becomes restless or reactive, an increase in the rate of the detomidine infusion, a bolus of butorphanol, or the infusion of additional local anesthetic has been sufficient to allow completion of the surgery.

The author compared the surgical times for 4 enucleations performed standing and 4 performed under general anesthesia. The average surgical times were identical. For the surgeries performed with standing sedation, the total average time from walking the horse into the stocks to the horse returning to its stall was 99 minutes. For the surgeries performed under general anesthesia, the total average time from induction to standing was 158 minutes. The anesthesia and surgery charges for an enucleation performed standing were 66% of the cost of the procedure performed under general anesthesia. However, when the anesthesia and surgery charges were divided by the total time that the staff were with the horse (returned to the stall or standing in the recovery stall), the charges per minute were 105% more for the procedures performed standing.

4. Discussion

In most horses, an enucleation can be performed with standing sedation. However, appropriate case selection is important. Horses requiring extensive surgical resection or with exceptionally painful globes are better candidates for enucleation under general anesthesia. The use of a detomidine constant-rate infusion allows for excellent control of the level of sedation and avoids the marked ataxia that can occur when administration of repeated boluses of sedation are required. Performing an enucleation in the standing horse is straightforward and allows the surgeon to avoid the potential complications associated with general anesthesia. As such, the author prefers to perform enucleation as a standing procedure in the horse unless there are mitigating factors.

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How to Perform the Ocular Portion of the Pre-Purchase Examination

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1. Introduction

An important yet commonly overlooked portion of the pre-purchase is the ocular examination. It is important for the veterinarian to be able to recognize lesions that may affect vision or function of the horse. Failure to notice characteristic ophthalmic abnormalities can result in frustration for the buyer.

Common abnormalities of which the veterinarian should be aware include the following:[1]

- Lid tumors
- Globe size abnormalities
- Exophthalmos/enophthalmos
- Corneal edema
- Corneal opacities
- Aqueous flare, hyphema, hypopyon
- Iris synechiae
- Cataracts
- Vitreous opacities
- Chorioretinal scars
- Retinal detachment

2. Materials and Methods

The veterinarian should come to the pre-purchase examination with the following items:

- Focal light source (example: Finoff transilluminator)
- Direct ophthalmoscope
- Indirect funduscopic lens (example: 20-D or 2.2 Panretinal lens)

Diagnostic Approach

Step 1: Facial Symmetry

The initial part of the examination should include evaluation of globe and facial symmetry. This is best evaluated with the veterinarian standing directly in front of the horse.[2] From this position, it is easy to assess globe position and size as well as ocular comfort. Standing directly in front of the horse is also the ideal position to assess ocular comfort by comparing the symmetry of the upper eyelid cilia. Another important way to assess ocular comfort is by examining the horse's face closely for signs of either present or past tearing.

Step 2: Palpation

Palpation can be used to investigate any asymmetry noted and to characterize the texture and sensitivity

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of the globe. Retropulsion is performed by pushing the globe back into the orbit through closed lids. It is important to note that this should not be performed if there is any threat of globe rupture. Resistance to retropulsion suggests the presence of a retrobulbar mass. Retropulsing the contralateral eye can provide a baseline for the individual patient. This technique can be used to help distinguish between changes in globe size and globe position (eg, exophthalmos, which would be associated with abnormal retropulsion and buphthalmos, which would be associated with normal retropulsion). Last, when retropulsion of the globe is performed, the third eyelid is displaced within the orbit and therefore passively elevates to cover the globe. Retropulsion is an essential part of any examination to allow close examination of the third eyelid for abnormalities. This is especially true in horses with lack of pigmentation around the eye. Lack of a third eyelid is important to note because it raises the suspicion of prior neoplasia. There are multiple published reports of squamous cell carcinoma metastasis months to years after third eyelid removal; therefore, a potential buyer should be made aware of this.

Step 3: Vision and Normal Reflexes

To determine if the horse is visual, it is important to note how it reacts to its surroundings. However, many horses adapt well to loss of vision and can continue to perform well despite a degree of vision loss, especially if the vision loss is chronic. There are multiple ways to test a horse's vision, including menace response, dazzle reflex, and maze testing. Maze testing is less commonly performed in equine patients and is usually reserved for cases that are difficult to interpret.

The menace response is commonly used in vision evaluation; however, it is important to keep in mind that vision and the menace response are not directly related. Completion of the menace response requires an intact visual and motor cortex, including cranial nerves II (optic nerve) and VII (facial nerve).[2] Keep in mind that this is a protective response that is learned and may not be present in animals younger than 2 weeks. To perform this test, the veterinarian should touch the periocular area once or twice to first stimulate the palpebral reflex. This should be followed by movements that create visible motion without creating stimulation of aural or tactile senses. The menace response should be evaluated throughout the horse's monocular visual field. For the horse, this extends approximately 150° (starting directly behind the horse), leaving a small section perpendicular to the forehead that requires binocular vision.[3]

The dazzle reflex is a very simple test that, similar to the menace response, evaluates the function of the retina, CN II, and CN VII. When a focal light source is directed toward the eye, the horse should respond by blinking.

Step 4: Pupillary Light Reflexes

After vision is fully assessed, the focal light source is used to evaluate pupil size and pupillary light reflexes. Pupil symmetry can be evaluated by standing approximately 6 feet in front of the horse with a focal light directed at the center of the horse's head to visualize both tapetal reflexes simultaneously.[2] The normal horse pupil is round when dilated and oval-shaped when constricted, with the horizontal axis being longer than the vertical axis. After pupil symmetry is evaluated, pupillary light reflexes should be evaluated individually. This is done by directing the focal light into each eye from approximately 2 to 3 centimeters. When the light is directed into one eye, both pupils should constrict (direct/indirect response). It is normal for the equine pupil to respond slowly, with vertical movement being more noticeable than horizontal movement.

The palpebral reflex is stimulated when the medial or lateral canthus is touched. A normal response is complete closure of the eyelids. Failure of this reflex occurs if there is damage to either the trigeminal (CN V) or facial nerve (CN VII) or if the eyelids are unable to close properly (example: trauma). Incomplete lid closure for any reason can predispose the horse to exposure keratopathy and other related consequences.[2]

Step 5: Adnexa

The eyelids and conjunctiva are best examined with a direct focal light. The most commonly seen eyelid abnormalities are tumors (sarcoids, squamous cell carcinoma) and tarsal margin irregularities.

Step 6: Cornea and Anterior Chamber

The remainder of the examination is best performed in a dimly lit area. The Purkinje-Sanson reflexes can be elicited with the use of a direct focal light source. Reflections can be seen at the cornea, anterior lens capsule, and posterior lens capsule. The clarity and location of these reflections can be altered with disease.[3] The presence of corneal opacities should be further evaluated with the use of slit lamp biomicroscopy. It is important to determine if the opacity is part of a larger disease process such as keratitis or uveitis. Even if the corneal opacity is not active or of a recurrent nature, a lesion that is large enough may interfere with vision.

To examine the anterior chamber, the focal light should be directed at a 45° angle (transillumination) to the corneal surface. The presence of cells within the anterior chamber results in internal reflection of the light known as aqueous flare.[2]

Step 7: Lens

Pharmacologic dilation of the pupil is not typically part of the pre-purchase examination, but without it, the lens cannot be viewed in its entirety. It should be clearly stated in the examination report that the pupil was not dilated, if dilation is not

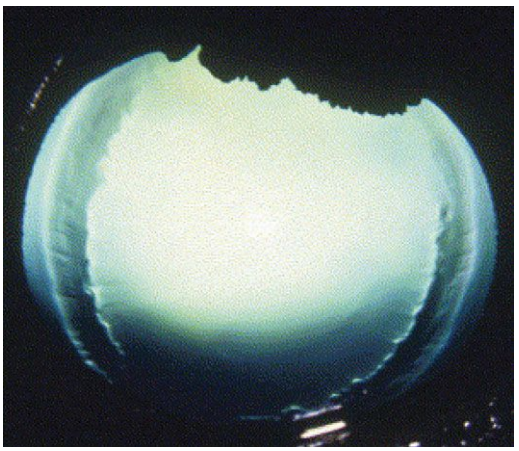


Fig. 1. Example of an equatorial cataract.

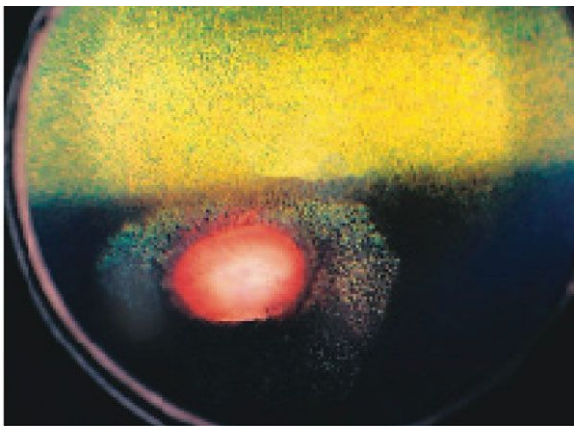


Fig. 2. Typical peripapillary or butterfly lesion.

elected. If the pupil is pharmacologically dilated, the owner or agent should be informed that depth perception may be altered, and thus handling and athletic activity should only be undertaken after accepting the risks of doing so with a horse whose vision may be impaired. Dilation of the pupil with 1% tropicamide can reveal subtle posterior synechiae and is the only way to evaluate the entirety of the lens. Retroillumination is commonly used to evaluate the lens. A focal light source is directed at the lens, starting approximately an arm's length from the horse's head. Slowly, the veterinarian moves closer to the horse while searching for opacities. Retroillumination causes opacities to appear dark against a light background.

All cataracts have the possibility of progression; cataract characteristics can help determine the likelihood of progression. Cataracts can be classified according to etiology (primary, secondary), age of onset (developmental, senile), stage of maturity (incipient, immature, mature, hypermature), and location.[4] Age of onset and etiology may be difficult to determine without a complete history. Developmental cataracts are the result of abnormal growth during embryogenesis and are commonly nonprogressive or very slowly progressive cataracts. Common developmental cataracts are seen at the anterior/posterior suture line and within the nucleus. The nucleus is the first part of lens to form in utero and therefore these cataracts are generally congenital. Senile cataracts are a form of nuclear cataracts generally seen in horses more than 20 years old; it is important to differentiate these from nuclear sclerosis. Nuclear sclerosis does not cause vision loss or interference with the tapetal reflection. The equator is the most metabolically active portion of the lens; therefore, cataracts in this location (Fig. 1) tend to be progressive, especially if vacuolation is also seen.[5]

Step 8: Fundus

The horse's fundus can be imaged through the use of either direct or indirect ophthalmoscopy and ideally should be examined with both techniques. Indirect ophthalmoscopy requires a focal light source and handheld lens. This technique provides a wide field of view; however, the image visualized is upside-down and backward. Conversely, direct ophthalmoscopy (diopter wheel set at 0 to −3) provides an upright with a very magnified view (approximately 15 times that of indirect ophthalmoscopy). Either technique can recognize important abnormalities such as retinal detachment, optic nerve atrophy, or chorioretinitis. The nontapetal fundus is often overlooked but is more often affected than the tapetal fundus, with lesions such as chorioretinitis and chorioretinal scarring.[6] Two common patterns of chorioretinal scarring are peripapillary (butterfly lesions) and multifocal (bullet-hole lesions) (Figs. 2 and 3). Sedation is important and often necessary



Fig. 3. Multifocal chorioretinopathy lesions or bullet-hole lesions.

to lower the horse's head so that the nontapetal fundus can be visualized in its entirety.

3. Conclusions

The two most basic parts of the ophthalmic examination are also the most important. The horse's vision and ocular comfort should be assessed before administration of any sedative agent. The ophthalmic portion of the pre-purchase examination is frequently overlooked, but its importance should not be underrated. Chronic ocular disease can result in temporary or permanent loss of use and frustration for the buyer. On the other hand, it is important that normal variants not be interpreted as lesions of clinical significance. When an unusual abnormality presents itself, it is important to offer referral to a board-certified ophthalmologist to complete the pre-purchase examination.[7]

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Significant Osteolysis in the Equine Distal Phalanx Associated With the Development and Progression of Laminitis

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Progressive distal phalangeal osteolysis is associated with equine laminitis and may affect sensitive lamellar pathology, disease progression, or response to therapeutic management. Authors' addresses: Department of Pathobiology (Engiles); Laminitis Institute, Department of Clinical Studies (Galantino-Homer, Boston, McDonald, Hankenson); School of Veterinary Medicine, University of Pennsylvania, 382 West Street Road, Kennett Square, PA 19348; Department of Bioengineering, School of Engineering and Applied Sciences (Dishowitz); Department of Orthopaedic Surgery (Hankenson), Perelman School of Medicine, University of Pennsylvania, 3900 Spruce Street, Philadelphia, PA 19104; e-mail: engiles@vet.upenn.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Laminitis-associated pathology of the equine distal phalanx (DP) has not been well characterized. The objective of this study was to identify, characterize, and quantify progressive DP osteopathology associated with equine laminitis through the use of micro-computed tomography (microCT).

2. Materials and Methods

Thirty-six feet from 15 horses with and without clinical history or signs of laminitis were evaluated for lamellar pathology using a novel, multi-modal pathological grading schematic and for quantitative bone loss through the use of microCT. Semi-quantitative pathology grades incorporating radiologic, gross, histopathologic, and temporal factors were correlated with DP microCT volumetrical and morphometric data.

3. Results

MicroCT measurements show significant decreases in bone volume within DP trabecular, compact bone, and total bone compartments as well as bone density and trabecular structural configurations that correlate with severity and progression of lamellar pathology. These bony changes are observed in performance and non-performance animals as well as in acute to subacute or mild/subclinical stages of disease.

4. Discussion

A detailed semi-quantitative pathological grading scheme and a novel method to evaluate and quantify

Research Abstract

NOTES

osteopathology of the equine DP through the use of microCT were developed. Progressive DP osteolysis correlated with severity and temporal progression of laminitis, which has the potential through shared blood supply and close anatomic approximation to influence lamellar pathology and disease progression.

How to Perform and Interpret Navicular Bursography

Tracy A. Turner, DVM, MS, Diplomate ACVS, ACVSMR

The injection of contrast material into the navicular bursa, accompanied by a subsequent radiographic examination, provides new and relevant information about pathologic damage to the navicular flexor fibrocartilage, the deep digital flexor tendon, and adjacent structures. A small percentage of injuries that can be identified by bursography have not been identified on magnetic resonance imaging. Author's address: Anoka Equine Veterinary Services, 16445 70th Street NE, Elk River, MN 55330; e-mail: turner@anokaequine.com. © 2013 AAEP.

1. Introduction

Palmar foot pain is a common cause of lameness. Many aspects of this lameness syndrome, including the pathogenesis, diagnosis, and treatment, are controversial, in part because there is little agreement as to what characterizes this common disease.[1,3] Traditionally, the diagnosis of palmar foot pain is based on history, clinical signs, response to palmar digital analgesia, and the detection of radiographic abnormalities. However, many recent reports suggest difficulties in reliably interpreting radiographic changes within the navicular bone.[2] Hence, magnetic resonance imaging (MRI) has become the standard for definitive diagnosis of underlying pathology in this lameness syndrome.[3] Lesions that have been diagnosed by MRI include core deep digital flexor injuries, superficial tears of the deep flexor within the navicular bursa, erosions of the navicular flexor fibrocartilage, and desmitis of the distal digital annular ligament.[3,4] Unfortunately, not all clients with horses with palmar foot pain choose MRI because of the expense or the inconvenience. In these cases, other methods must be used when radiography fails to define a diagnosis. The objective of this report is to discuss how to perform navicular bursography and discuss interpretation of results.

2. Materials and Methods

Injection into the bursa was made from the palmar surface with the limb flexed at the carpus. Aseptic injection techniques were used to inject a 3-mL mixture of 1:1 contrast material and local anesthetic or medication.[5] The landmark for needle insertion was a point just proximal to the central sulcus of the frog, with the needle directed toward the apex of the frog and in a direction parallel to the ground surface of the hoof. Needle insertion is made easier if regional analgesic injection of the medial and lateral palmar nerves is performed first, or, in the instance of "blocking" the bursa for diagnostic purposes, a small bleb of local anesthetic over the site of insertion is very helpful. A 20-gauge, 3.5-inch (9-cm) needle was used. The needle was inserted until resistance was encountered; this was usually at two-thirds the length of the needle (Fig. 1A). If the needle was inserted further before encountering resistance, it usually indicated incorrect placement.

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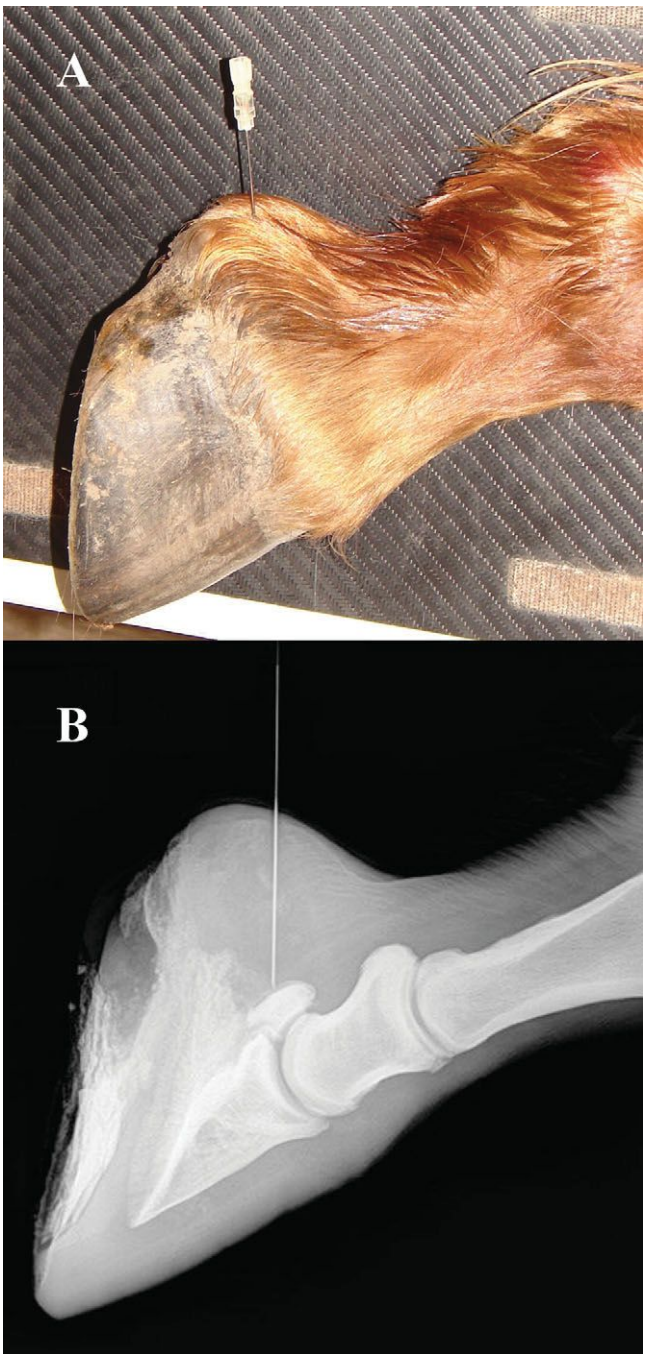


Fig. 1. A, Placement of the needle, just above the central sulcus in a plane parallel to the round surface. B, Lateral radiograph of needle placement. Note that the needle tip is in the central portion of the navicular flexor surface.

A lateral radiograph of the hoof was taken to confirm the position of the needle before injection. Ideally, the needle tip was positioned midway between the proximal and distal borders (Fig. 1B). Once the needle position was confirmed, the bursa was injected with the contrast mixture and a second lateral hoof radiograph was taken to confirm the filling of the bursa. If the bursa had been successfully injected, then a palmaroproximal-palmarodistal (PP-PD) oblique projection of the navicular bone was obtained. The contrast material seen from a lateral view normally had the shape of an apostrophe. The contrast, seen from the PP-PD projection, was a distinct line of contrast material juxtaposed to the deep digital flexor tendon that was normally separated from the navicular cortical bone by a layer of radiolucent fibrocartilage (Fig. 2).

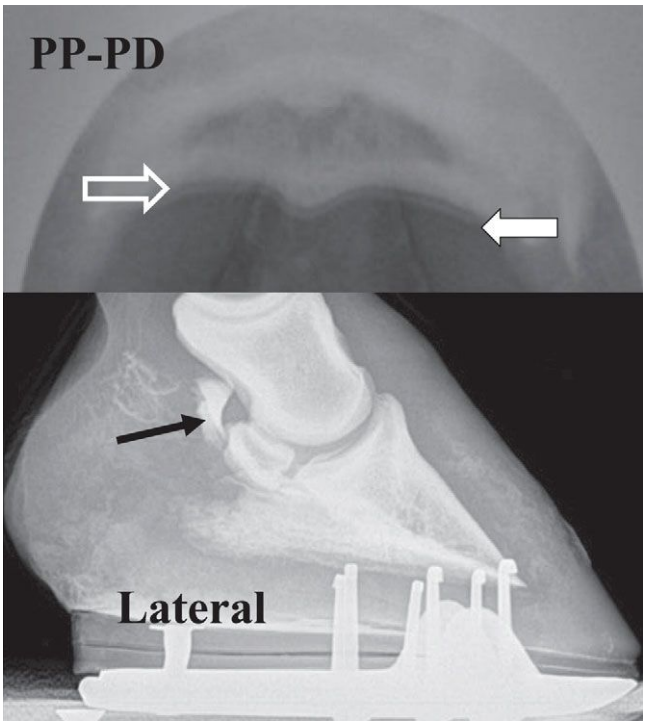


Fig. 2. Normal navicular busograms (PP-PD). Note the even black line palmar to the navicular flexor surface (open arrow); this is the flexor fibrocartilage. White line (solid arrow) is the bursa, which also outlines the dorsal margin of the deep flexor tendon. Lateral: apostrophe appearance (black arrow).

The bursograms were evaluated for several different changes: (1) normal flexor fibrocartilage seen as a uniform radiolucent area 1 to 2 mm in thickness covering the flexor surface of the navicular bone; (2) thinning or erosions of the flexor fibrocartilage, seen as a loss of the thickness of the previously mentioned radiolucent line; (3) fibrillation or splits of the deep flexor tendon within the navicular bursa, which was noted as filling defects along the bursal surface of the deep flexor tendon; (4) presence of flexor subchondral bone cystic defects, which were noted as focal filling of the flexor cortical area with contrast (5) communication of the navicular bursa with the distal interphalangeal joint, seen as leakage of the contrast from the bursa into distal interphalangeal joint; (6) complete focal loss of the dye column, which was thought to be a result of flexor tendon adhesion to the bone; (7) narrowing or enlargement of the proximal to distal borders of the

bursa (bursa change) thought to represent inflammatory changes of the bursa; (8) leakage of contrast from the bursa, suggesting a tear of the border of bursa; (9) marked widening of the contrast thickness thought to indicate loss of palmar support of the tendon by the distal annular ligament; (10) contrast within the body of the tendon thought to be a focal deep flexor tear; (11) contrast within the impar ligament assumed to be indicative of tearing or damage to the impar ligament; and (12) contrast within or surrounding the proximal suspensory of the navicular bone indicative of ligament injury.

3. Results

The author has performed 344 bursograms. Normal fibrocartilage was seen in 29% of examinations. Thinning or erosion of the flexor fibrocartilage was seen in 62% of the bursograms. Fibrillation of the deep flexor tendon was recognized in 24% of the bursograms. Subchondral defects were seen in only two cases. Communication between the navicular bursa and the distal interphalangeal joint was noted in 11% of the contrast studies. Bursal tears were seen in 18% of the bursograms. Bursa changes were noted in 24%. Proximal suspensory tears were noted on 5%. Four of the changes were only seen in horses with unilateral lameness: (1) adhesions (loss of the dye column) were noted in 8% of the horses; (2) loss of distal annular ligament support was found in 1% of the horses examined; (3) contrast filled areas of the deep flexor in 3% of the horse; and (4) the impar ligament was involved in 1% of horses.

Evaluation of bilateral bursograms revealed two populations: 43% of the horses with bilateral examinations had similar changes on both feet, but 57% of the horses had completely different changes. Secondly, the bursa changes were more numerous on the more lame leg in all cases. Four horses had repeat bursograms. In two of the horses, the changes noted were worse on subsequent bursograms, whereas in the other two, the abnormalities seen in the repeat bursograms were unchanged.

Therapeutic injection of the navicular bursa was an important aspect of managing these cases, and treatment was considered successful in 87% of the horses. Therapy was unsuccessful on horses with adhesions, horses in which the most striking lesion was deep flexor fibrillation, and horses with complete loss of cartilage from the flexor surface of the navicular bone.

Since the original description of navicular bursography in 1998, the author has noted six other changes that have not been previously described on bursograms: (1) tears in the core of the deep flexor tendon extending from the bursa up the pastern (Fig. 3); (2) tears in the bursa seen as leakage from the bursa (Fig. 4); (3) displacement of the deep flexor tendon from its normal position behind the navicular bone (Fig. 5); (4) contrast within the impar ligament (Fig. 6); (5) contrast within and around the proximal navicular ligament (Fig. 7); and (6) changes in the normal shape of the navicular bursa (Fig. 8A,B).

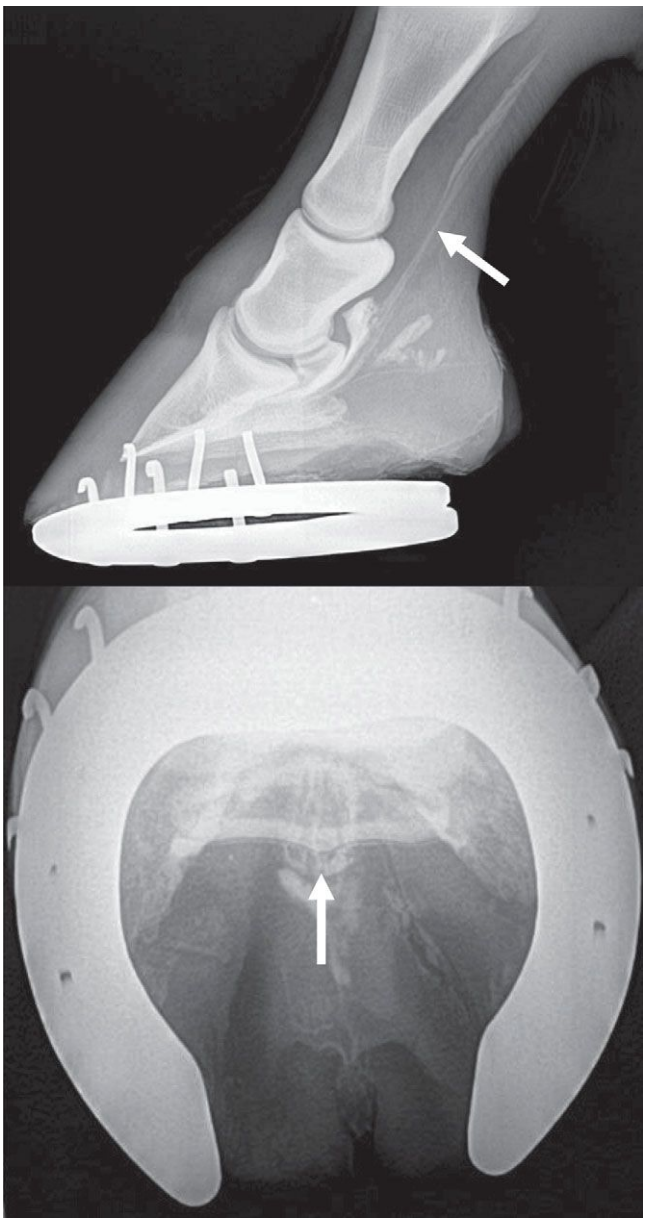


Fig. 3. Filling of the tendon with dye from the lateral and PP-PD views. Arrows show where the dye fills the tendon.

4. Discussion

Navicular bursography was originally devised to confirm an injection of local anesthetic into the bursa.[5] Navicular bursography is used to further evaluate the navicular bursa region when standard radiographs failed to discern the pathology. The most common finding noted is thinning or erosions of the flexor fibrocartilage. This is consistent with pathologic studies that have shown thinning and erosions of the flexor fibrocartilage to be the most

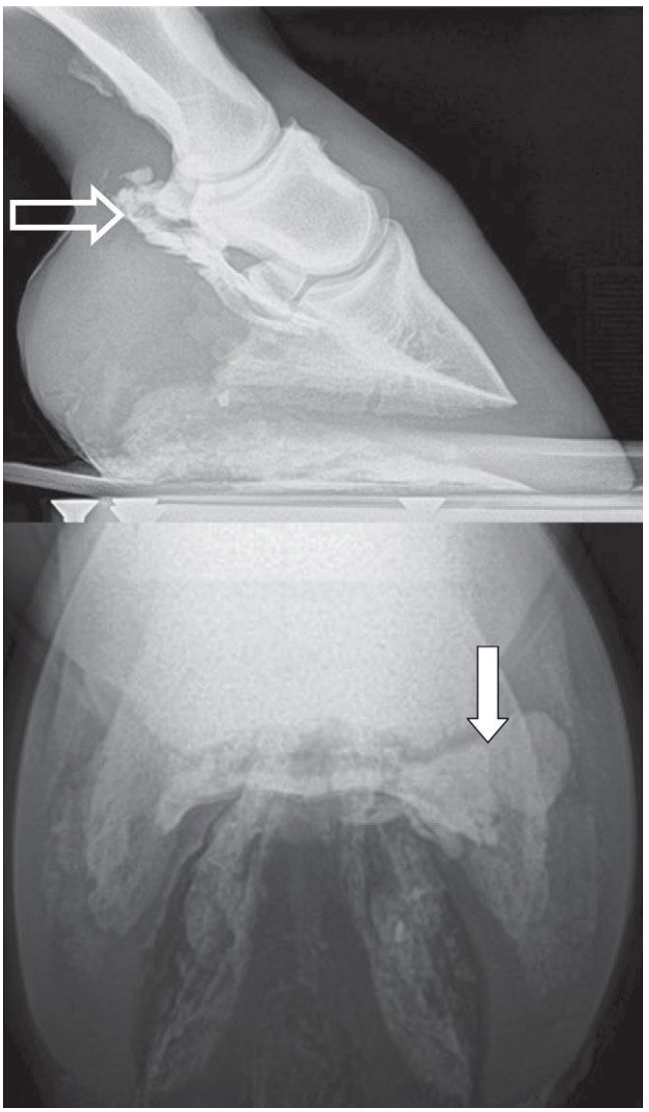


Fig. 4. Leakage of dye from the bursa. Leak is most easily seen on the lateral projection (open arrow). PP-PD projection shows leakage from the lateral aspect of the bursa (arrow).

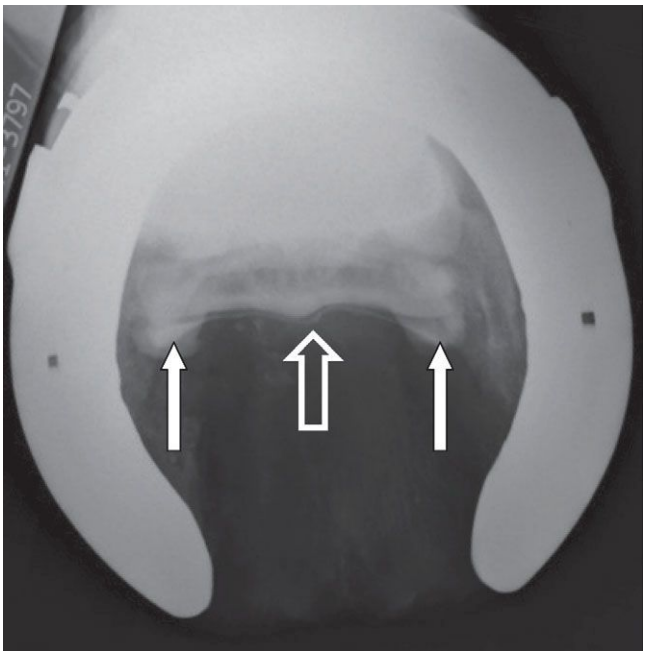


Fig. 5. Displacement of the deep digital flexor tendon from its normal position. Note widening of the bursa space (solid arrows) and the mismatch of the deep digital flexor tendon on the sagittal ridge (open arrow).

common pathology seen in navicular disease.[6] It follows that the flexor fibrocartilage is the most fragile structure in this area and therefore the most readily damaged. Although this is the most common change seen, it is not more important than any other change. The third most common finding is splits or filling defects along the bursal surface of the deep digital flexor tendon. This is a common MRI finding as well.[3] In these cases, these splits were invariably associated with thinning and erosions of the flexor surface of the navicular bone.

Communication between the bursa and the distal interphalangeal joint is a sign of coffin joint capsule injury and is often associated with collateral ligament desmitis.[7] The bursa and joint are in close proximity, and injury to the palmar capsule could result in this communication. Adhesions of the deep flexor to the navicular bone were seen as gaps in the dye column. A previous study proved these to be adhesions on necropsy.[5]

MRI has detected fibrous scar tissue between the proximal suspensory ligament of the navicular bone and the deep digital flexor tendon.[3] Contrast studies show this finding as a shortening of the length of the bursa. The bursa loses its apostrophe shape and is seen as a small fluid sac only as long as the navicular bone. This illustrates some of the restriction that is thought to occur as a result of this

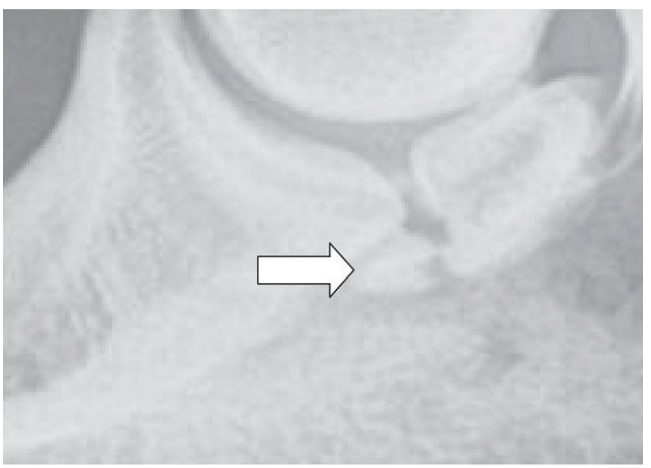


Fig. 6. Damage to the impar ligament seen as dye within the impar ligament (arrow).

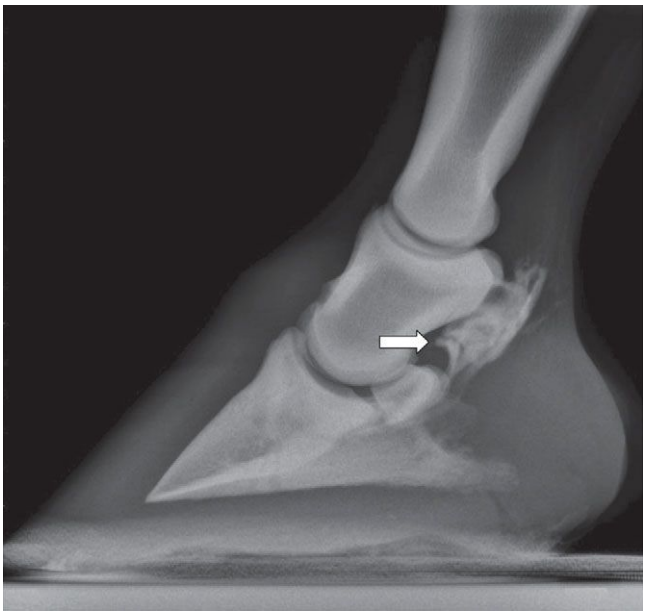


Fig. 7. Dye surrounding the collateral sesamoidean ligament (arrow).

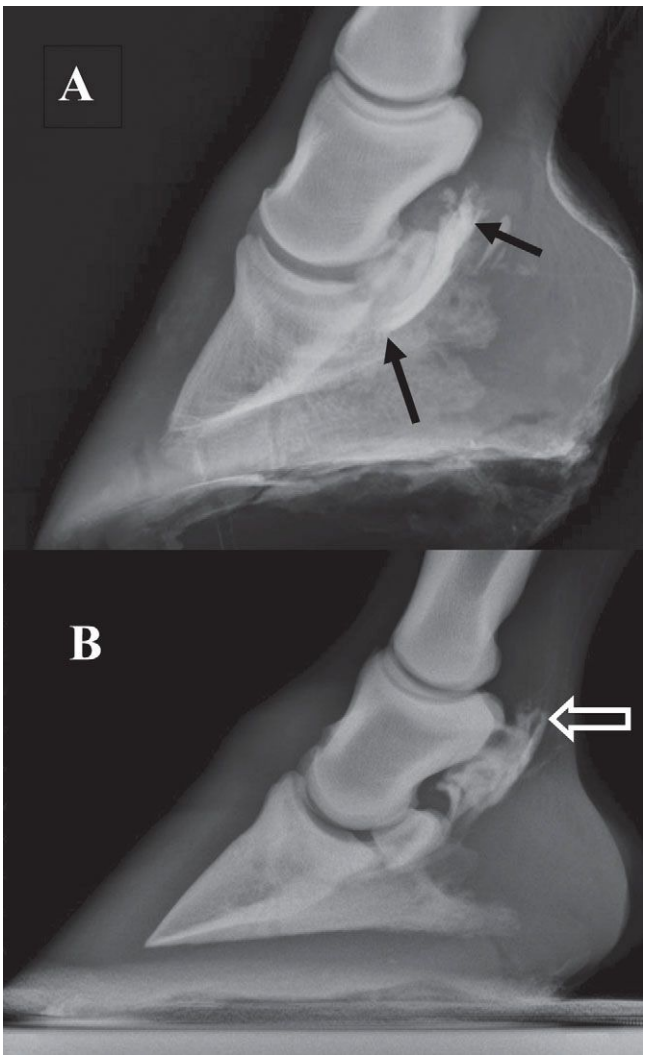


Fig. 8. Restricted bursa. A, Note that the dye barely extends beyond the navicular borders (arrows). B, Enlarged bursa with the proximal border extending nearly to the pastern joint (open arrow).

pathology.[3] This was described as bursal change in this study, and it included both the constriction and enlargement of the bursa. Tearing of the abaxial border of the bursa is seen as leakage of the dye from the bursa. This is a lesion that has not been reported by MRI or any other pathologic studies. The reason for this is that the leaks are small and only detectable by a dynamic study such as was performed here. From a pathologic standpoint, it would be expected to cause bursitis.

Dye may fill into the deep flexor tendon. This is one of the most common findings seen with MRI. It would be expected that bursography would underestimate this lesion because to fill the injury, it must communicate with the bursa. However, if identified, this offers another avenue to treat the tendon by injecting the bursa. For example, stem cells or platelet-rich plasma could be injected into the bursa and fill the same defect as the dye. Injury was also noted in this study to the impar ligament and collateral sesamoidean ligament (proximal suspensory of the navicular bone). Both have been seen with MRI. Another injury seen with MRI is distal annular ligament desmitis.[4] In these cases, the lesion caused constriction of the deep flexor tendon. In this study, this injury was diagnosed when the contrast filling the bursa delineated a wider area. The only way this can occur is if there is more space between the deep flexor tendon and navicular bone. The PP-PD radiographic projection is performed with the leg positioned so that the tension on the deep flexor tendon and between the deep flexor tendon and navicular bone is at its greatest. Therefore, to allow this greater filling of the bursa, an injury has had to occur to allow the tendon to displace in a more palmar direction. Desmitis of the distal annular ligament would fulfill these criteria. It is hypothesized in the cases reported here that this may be a more acute phase of the same injury. In the cases in which bursography identified this lesion, the horses were kept active to prevent constriction of the annular ligament as it healed.

5. Conclusions

Navicular bursography is a simple technique that can be used to confirm injection into the navicular bursa and can also give valuable new information regarding pathology in the region of the navicular bone. Changes seen by means of contrast navicular bursography represent stages of pathologic damage and allow a more timely therapeutic intervention, more targeted management, and more accurate

prognostication. Bursography offers another imaging modality to study pathology of navicular region. This is a technique that can be performed when MRI is not an option.

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How to Perform Ultrasound Evaluation of the Jugular Vein

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1. Introduction

Disorders of the jugular vein are relatively common and potentially serious problems for the equine practitioner, with thrombophlebitis being the most common. Early recognition, proper assessment, and appropriate treatment are required to avoid serious consequences such as complete thrombotic occlusion of the vein or sequelae to sepsis within the vein, which could include hematogenous extension to other locations of the body.

Jugular thrombophlebitis is a common complication in sick horses with hypercoagulable states. It has also been observed to occur in horses receiving repeated injections (such as might occur with certain athletic disciplines), with errant intravenous injections in horses that are difficult to handle or with injections given by an unskilled individual, and with intravenous injection of potentially irritant substances.[1,2] Traditional evaluation of changes in the jugular vein have included clinical evaluation by digital palpation and determining whether it can be distended by distal occlusion, indicating the presence of normal blood flow. Ultrasound is a valuable tool for assessing pain and swelling along the jugular vein—in early detection and extent of jugular thrombosis—as well as for determining the likelihood of septic thrombophlebitis.[3] Ultrasound evaluation of the jugular vein is an easily applied technique that is often underutilized in clinical practice. It can provide immediate visual imaging and definition of the anatomic location of swellings or alterations along the jugular vein and can guide clinical decision-making. This presentation should provide the practitioner with the knowledge of the technique for ultrasound imaging of the jugular vein as well as a brief review of ultrasound changes associated with jugular vein thrombosis and thrombophlebitis.

2. Materials and Methods

Technique for Ultrasound Imaging of the Jugular Vein

The approach to ultrasound imaging of the jugular vein involves selection of an appropriate ultrasound probe, patient preparation, and occlusion of the vein distally to allow for maximal distension of the lumen, if any blood flow is present. Higher-frequency ultrasound probes (7.5 mHz or higher, preferably 12 mHz) are necessary to allow for fine detail imaging of the jugular vein and other adjacent superficial anatomic structures within the neck. Smaller contact footprint probes or microconvex probes may fit against the jugular groove most eas-

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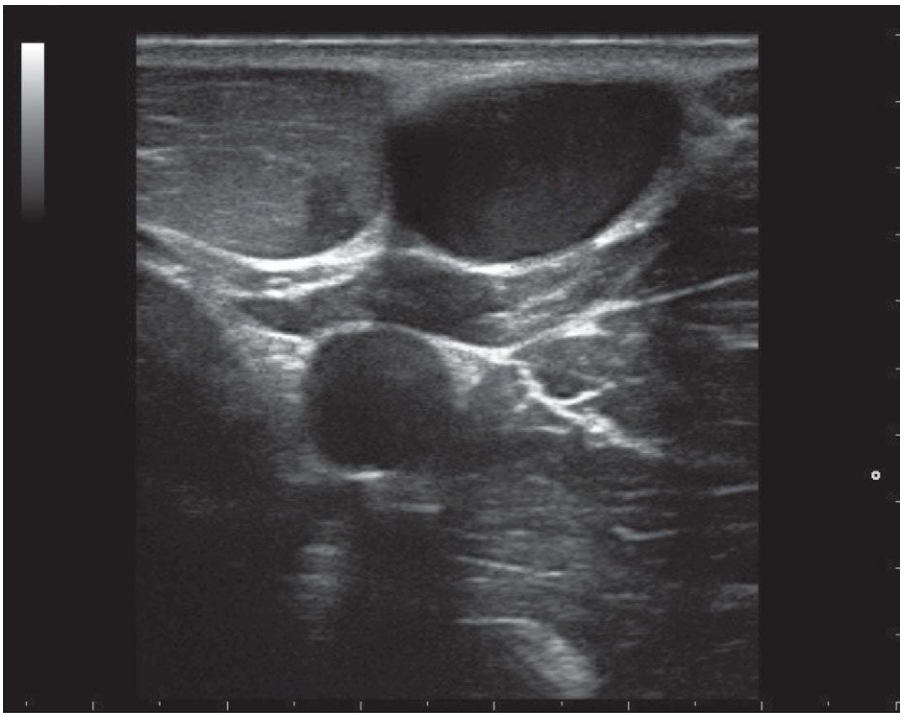


Fig. 1. Ultrasound image of a normal, distended jugular vein shows a thin wall and relatively echolucent blood within in the lumen. Adjacent anatomic structures include the carotid artery, esophagus, and trachea.

ily, but commonly available linear rectal probes can also be used. Scanning of the jugular vein may involve simply wetting the skin and hair on the neck with isopropyl (or similar) alcohol in horses with a thin hair coat. In those patients with thicker hair coats, or those in which best clarity and detail of image is desired, clipping the hair along the jugular groove from the mandible to the thoracic inlet might be necessary. Application of ultrasound contact gel may facilitate best contact of the probe with the skin along the jugular groove. The examination is performed by occluding the jugular vein with pressure

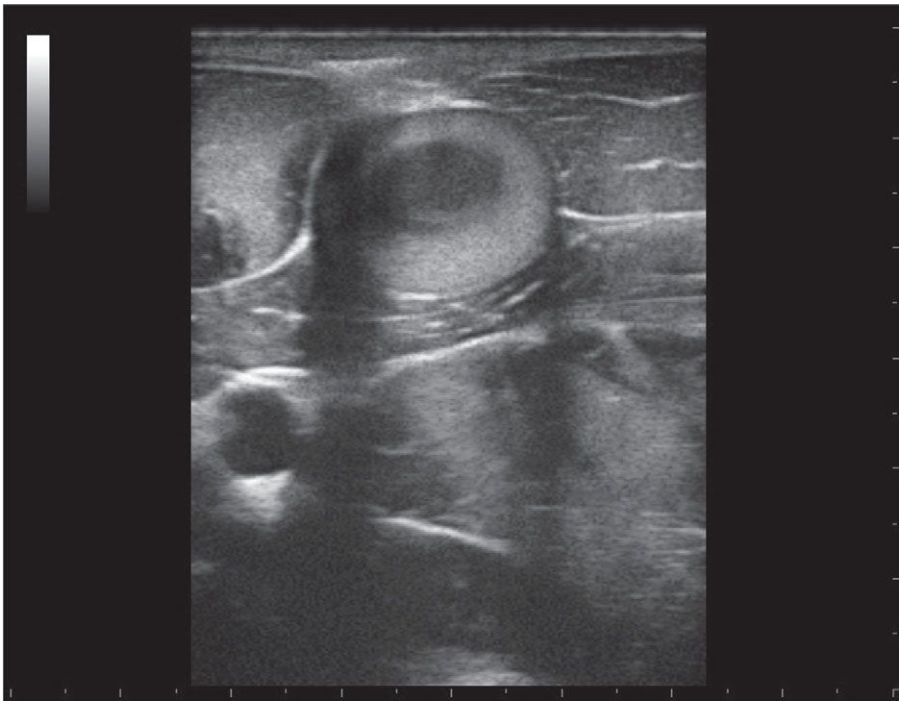


Fig. 2. Ultrasound image of a normal, occluded jugular vein with swirling echogenicity of the static blood within the lumen.

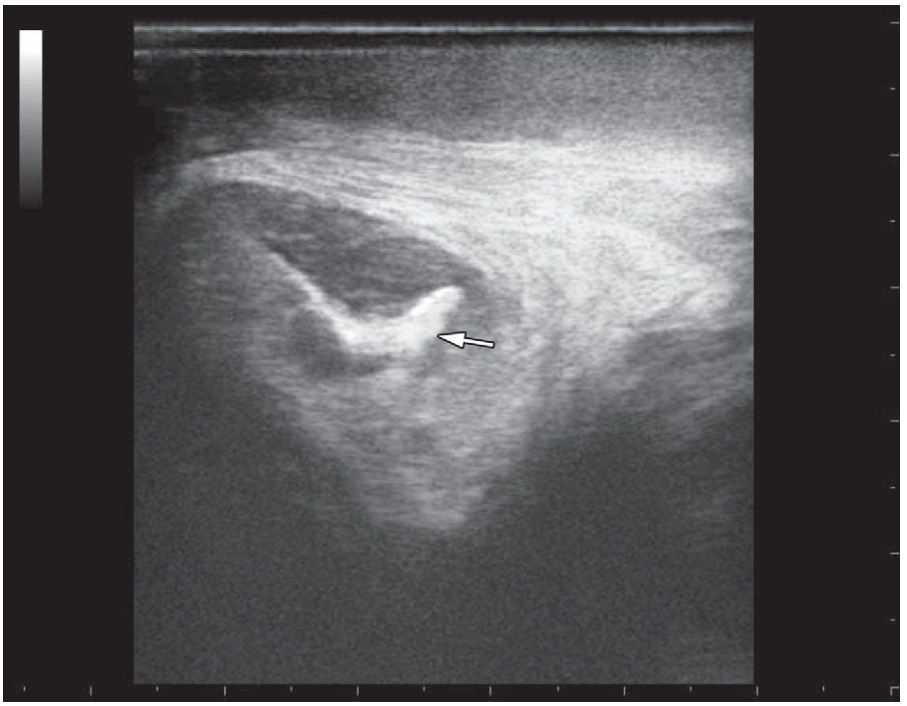


Fig. 3. Irregular echogenic appearance of the central aspect of the sternocephalicus muscle (arrow) and perivenous connective tissues from an aberrant attempted jugular vein injection in a fractious horse. Clinically, this appeared as swelling and pain along the jugular groove and required ultrasound examination to differentiate the location from a true jugular vein injury.

by the thumb near the thoracic inlet to achieve maximal distension of the vein (if there is venous flow) to allow for better definition of the contents of the lumen of the vein. This can be done with the opposite hand of the sonographer, or an assistant might perform this task. The jugular vein should be scanned along the entire the extent of the vein in both directions—along the visible branches of the jugular vein proximally just behind the mandible, distally to the thoracic inlet. The examiner may

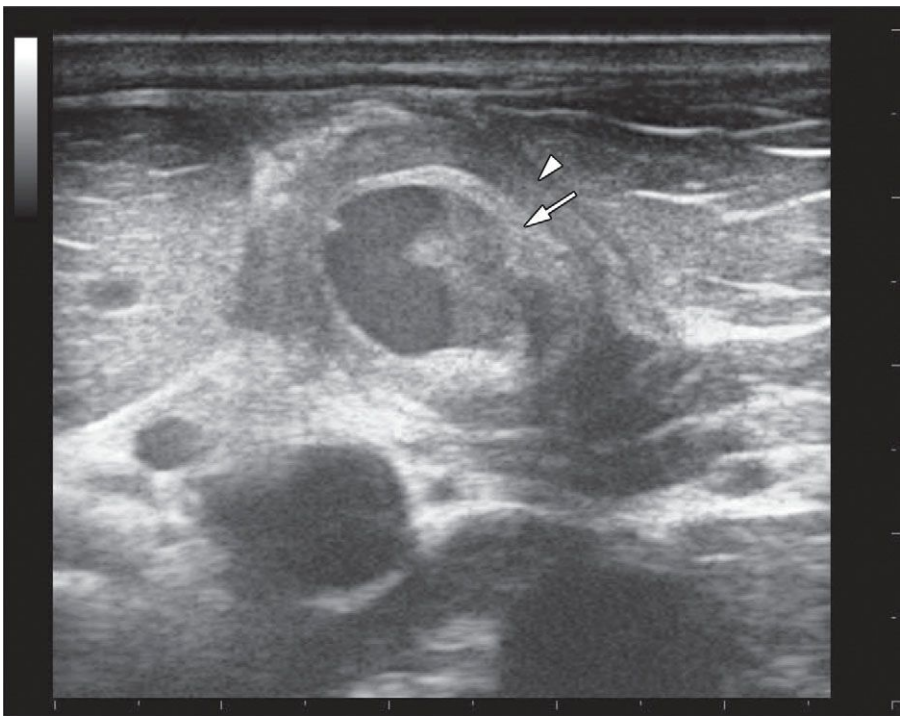


Fig. 4. Partial luminal thrombus in the jugular vein after intravenous catheterization. The tunica muscularis (arrowhead) and intimal layers (arrow) are thickened by reactive inflammatory response.

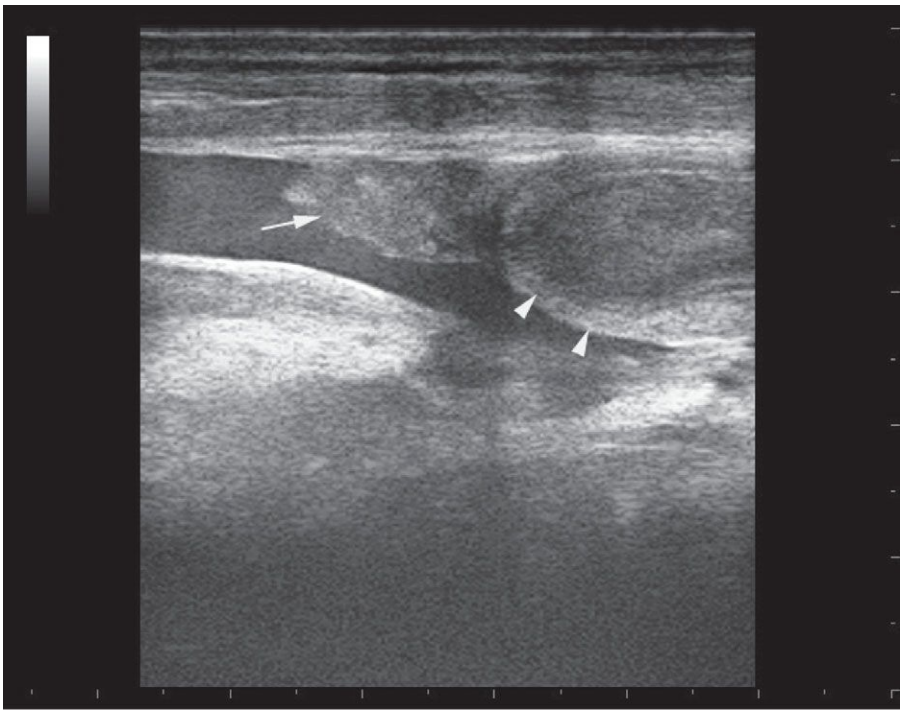


Fig. 5. Longitudinal ultrasound image of an acute thrombosis of the jugular vein associated with intravenous catheterization. Arrowheads denote the more mature component of the thrombus, with the more recent fibrin deposition denoted by the arrow. For anatomic orientation: proximal is to the right, distal to the left in this image.

eventually focus on the region of interest—a site of swelling adjacent to an intravenous catheter site or a region of swelling from prior intravenous injection. The author finds it most useful to initially evaluate the vein in a transverse (cross-sectional) plane and then follow with a longitudinal plane, which is useful to demonstrate linear anatomic relationships, such as linear extent of a thrombus along the course

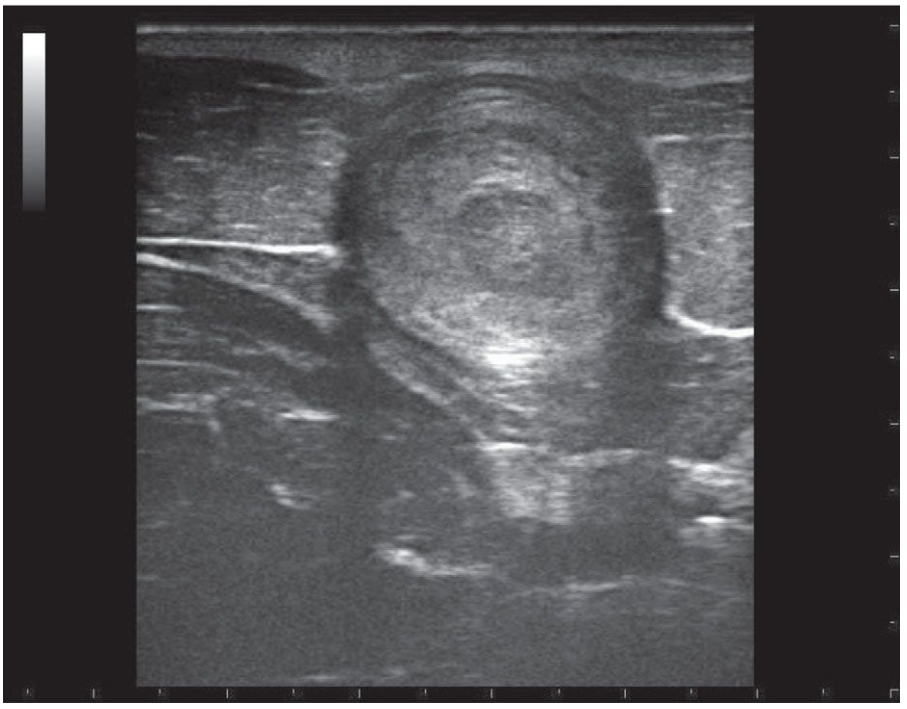


Fig. 6. Chronic thrombosed jugular vein. The venous lumen is obliterated by organized fibrous connective tissue with a somewhat laminar appearance.

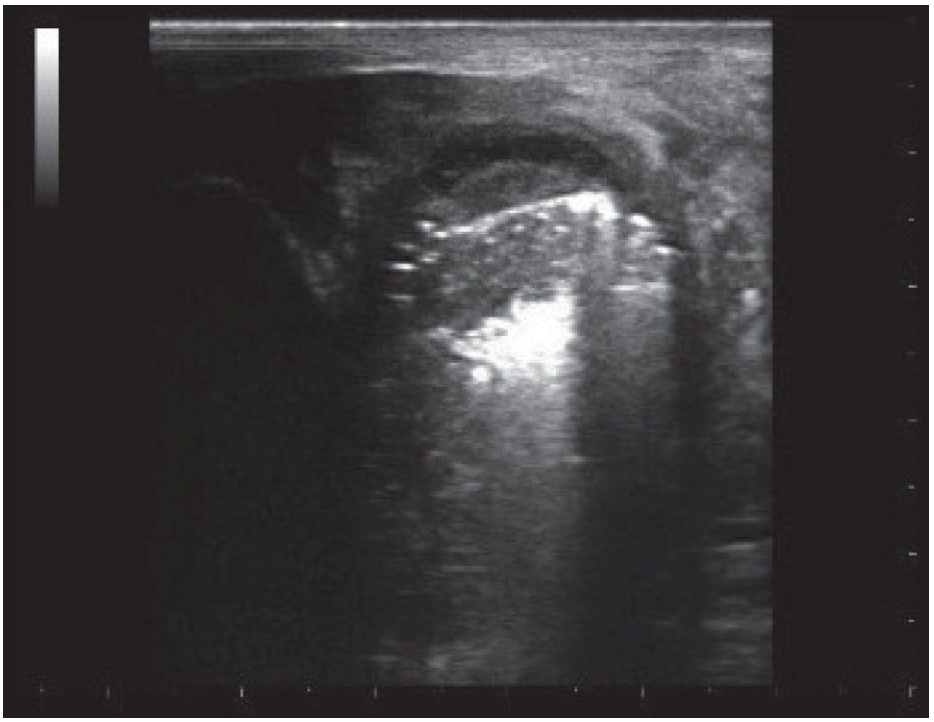


Fig. 7. Multifocal echogenic regions and gas echoes within the luminal thrombus, suggestive of suppurative exudate, from an anaerobic infection septic thrombophlebitis.

of the jugular vein. It may also be useful to scan the contralateral jugular vein for comparison with more normal architecture within that individual horse.

Anatomic Considerations

Ultrasonography will allow anatomic definition of the location of clinically apparent swellings along the course of the jugular vein and their relationship

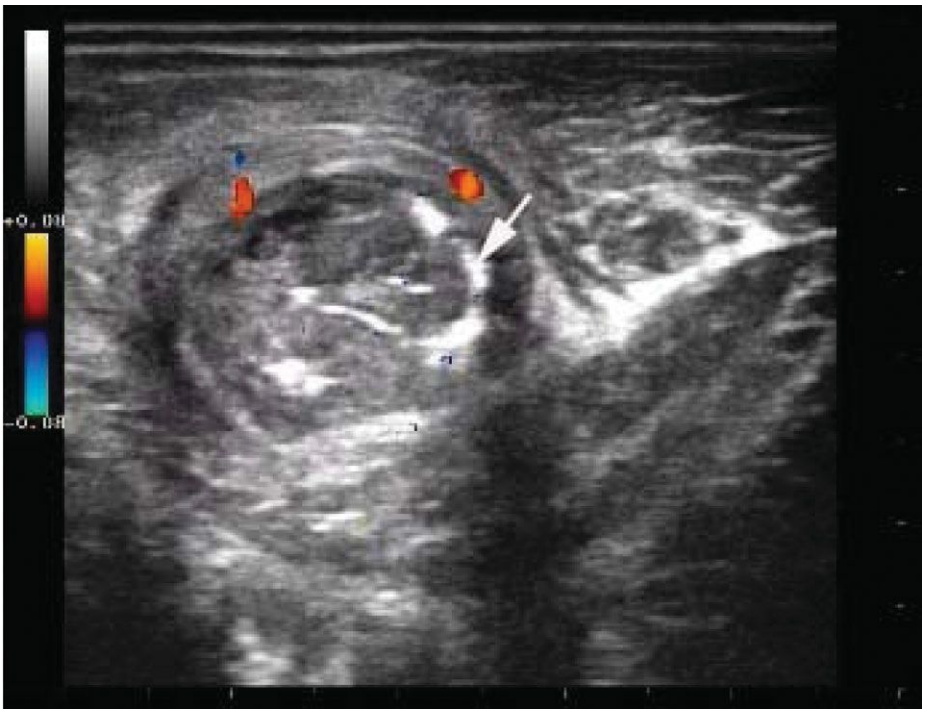


Fig. 8. Color flow Doppler ultrasound image of septic jugular thrombophlebitis with laminar echogenic material (arrow), consistent with suppurative exudate.

to the structures caudal to the mandible (parotid salivary gland, branches of the carotid artery) and from the underlying main carotid artery, esophagus, and trachea along the course of the neck.

3. Results

Normal jugular veins appear to be collapsed, with thin echolucent regions of blood flow without some distal occlusion to interrupt blood flow and allow for maximal distension of the vein lumen. Normal distended jugular veins will appear as rounded tubes of echolucent fluid (blood) within the lumen and thin, echogenic walls with distinct margins of the endothelial surface (Fig. 1). The blood column within a normal vein that has been occluded will, on some occasions, begin to swirl and provide a variably echogenic laminar appearance (Fig. 2).

In the author's practice, swelling or pain around the intravenous catheter insertion site are considered indications for ultrasound imaging of the jugular vein. In addition, distension of the vein or its proximal branches with or without unilateral facial edema or trauma to the neck region might also be indications for scanning the jugular vein.

Common problems that can be identified with ultrasound imaging of the jugular vein include detection of perivascular inflammation, localized or extensive thrombophlebitis, differentiation of septic thrombophlebitis from thrombosis alone, and early detection of catheter-associated thrombosis.

Perivascular inflammation (Fig. 3)—associated with intravenous injection or catheter placement—can result in swelling and thickening of the perivascular tissues, whereas the lumen of the jugular vein remains unaffected. This finding would usually indicate that topical local therapy—anti-inflammatory and/or topical antimicrobial agents—might be sufficient in resolving the thickening. Occasionally, an aberrant injection of material into the adjacent perivascular tissues or surrounding musculature might occur, and this can be demonstrated and monitored with the use of ultrasound.

Varying degrees of thrombosis within the jugular vein lumen (Fig. 4) can be identified, from focal/regional involvement to more progressive thrombosis with complete luminal obstruction that can extend along the entire course of the jugular vein within the neck. A thrombus will appear as a variably echogenic, variably shaped solid material within the lumen of the vein. Often, the thrombus will appear to be attached to the endothelial surface of the vein at a site of catheter entry or as a sheath along an indwelling intravenous catheter or attached to a site of previous injection or catheterization. The margins may appear irregular and indistinct ("fuzzy"), as with recent or active thrombus formation, or smooth with thrombi of longer duration in which blood flow has formed the margins. Occasionally, a jugular thrombus of longer duration will have a cone shape to its distal margins. Longitudinal images (Fig. 5) are useful for determining the linear involvement along the course of the jugular vein. Acute thrombosis can undergo repair by fibrinolysis to varying degrees of recanalization, or it can evolve to organization into chronic fibrous resolution with complete obstruction of the vein lumen (Fig. 6).

Thrombosis can occur with repeated intravascular injections, iatrogenic trauma on attempting to inject the jugular vein of a fractious horse, or after injection by an unskilled individual. Thrombosis has also been reported to occur as a sequel to other sites of infection, unrelated to venipuncture or catheterization.[2] Once thrombosis of the jugular vein has been identified, ultrasound imaging can be useful in monitoring the clinical progression or resolution of the lesion.

In addition to detection of thrombosis, ultrasound is also useful for detecting changes consistent with septic thrombophlebitis as well as determining the location and extent of inflammatory exudate within the vein. Septic thrombophlebitis is usually observed as varying regions of densely echogenic material within the thrombus (Figs. 7 and 8). Finding this echogenic focus can allow for ultrasound-guided aspiration to obtain a sample of the exudate for bacterial culture and sensitivity and possibly drainage of the exudate.

4. Discussion

Ultrasound imaging is a useful tool for evaluation of a variety of changes along the jugular vein. It can aid in differentiation of the anatomic location of swellings in the jugular groove—whether the change is within the jugular vein or from involvement of the perivascular tissues. Commonly differentiated changes include perivascular inflammation, jugular thrombosis over the spectrum of acute-to-chronic and focal-to-extensive involvement of the vein, and detection of changes consistent with septic exudate within the thrombus or in the perivascular tissues. Early detection and intervention for thrombosis, whether from injections or associated with intravenous catheter placement, can improve clinical outcomes and may help to reduce costs of medical management. Ultrasound imaging is vital in the detection of sites of sepsis within the venous thrombus. The technique for ultrasound imaging of the jugular vein is relatively easy and involves the use of readily available ultrasound equipment in most equine practices.

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Review of Innovative Ultrasound Techniques for the Diagnosis of Musculoskeletal Injury

Natasha M. Werpy, DVM, Diplomate ACVR*; and Laura Axiak, DVM

Ultrasound examination with the use of standard ultrasound and angle contrast (on- and off-beam angles) ultrasound techniques, as well as imaging the non–weight bearing limb or changing limb position, can contribute valuable information to the diagnosis of musculoskeletal injury. In addition, these techniques may facilitate visualization of an injury by use of ultrasound after magnetic resonance imaging examination. Therefore, the lesion can subsequently be monitored with ultrasound. Authors' address: 9418 SW 67th Drive, Gainesville, FL 32608; e-mail: equinedxim@yahoo.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Multiple imaging modalities are available to aid in the diagnosis of tendon and ligament injury.[1] Often, a combination of modalities is used to fully characterize musculoskeletal injury. With the advancement and increasing availability of computed tomography (CT) and magnetic resonance imaging (MRI), diagnoses are commonly made through the use of advanced imaging after negative ultrasonographic or radiographic studies are performed. Retrospectively, with knowledge of the location and character of the lesion, many injuries can subsequently be identified through the use of ultrasonography or radiography. This process contributes to the development of improved imaging skills, and, in certain cases, the diagnosis can be subsequently achieved with the use of ultrasound and/or radiographs without the benefit of advanced imaging. CT and MRI may require general anesthesia and a significant financial investment from the client. Ultrasonography is readily accessible, cost-effective, and can easily be performed in the field. These qualities make it a highly advantageous imaging modality and facilitate its use for sequential examinations. Sequential examinations are extremely useful to assess lesions over time and healing responses on the basis of administered therapeutics. However, ultrasound is extremely operator-dependent, relying on the training, skill, and operator experience to obtain diagnostic images. Educated ultrasonographers understand how the ultrasound beam propagates in normal tissue as compared with abnormal tissue. They are able to differentiate injury from artifact and have a practical knowledge of the limitations of ultrasound as an imaging modality.[2] The purpose of this review is to describe the use of angle contrast (on- and off-beam angles) ultrasound techniques, to discuss the interpretation of these images, and to demonstrate the benefits of comparing images acquired with the limb in weight bearing and non–weight bearing positions. These ultrasound techniques will be presented in combination with other modalities to provide a better appreciation of ultrasonographic capabilities and how they can be used for the diagnosis of musculoskeletal injury.

NOTES

Anatomic Variation

The most reliably identified tendon and ligament injuries through the use of ultrasound include enlargement, enthesopathies, and hypoechogenicity indicating inflammation, edema, and/or fiber disruption. These injuries can be described by the structure involved, their exact locations, the extent of injury, and amount of damage. Anatomic knowledge of the structures being examined in both the transverse and longitudinal planes is essential for the diagnosis of these injuries and to recognize artifacts. A successful ultrasonographer is familiar with the normal anatomic appearance (size, shape, position, and textural quality) of each structure examined as well as any anatomic variations.[3]

Tendons and ligaments are of similar composition and differ in their structural orientation. The fibers are more uniformly distributed in tendons and are more multi-directional in ligaments.[4] Normal anatomic variation in the fiber orientation within ligaments causes non-uniform echogenicity on ultrasound examination, such as in the collateral ligaments of the distal interphalangeal joint (Fig. 1).[5,6] This variation in fiber orientation also causes magic angle artifact within ligaments on MR images.[5,7] When regions of decreased echogenicity are identified in collateral ligaments with multi-directional fibers, multiple ultrasound beam angles are then used at the same level to create echogenicity in the different fiber bundles (Fig. 2). In addition, comparison to the contralateral limb can be helpful when attempting to differentiate normal anatomic variation from a clinically significant abnormality.

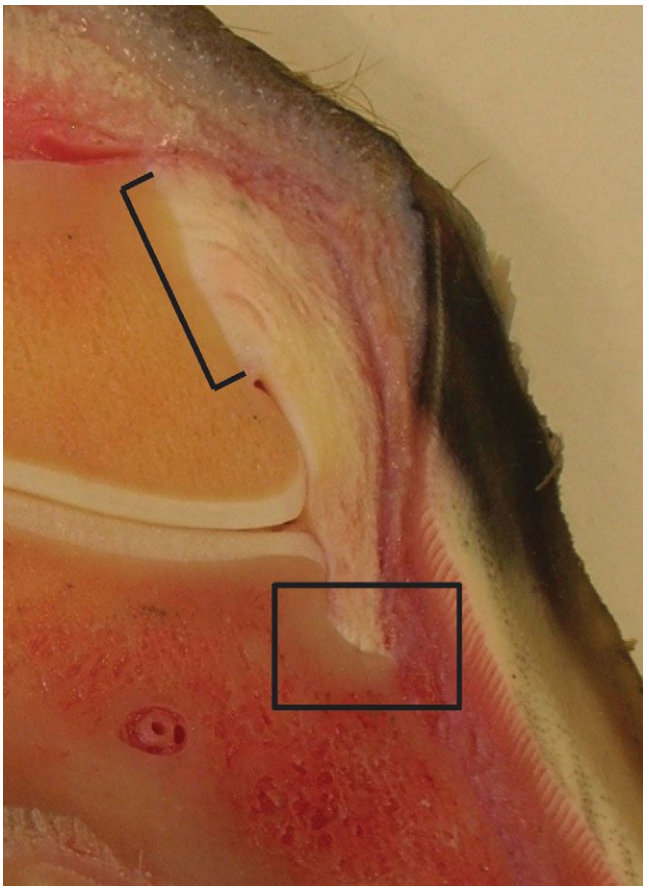


Fig. 1. Frontal plane section of the foot at the level of the distal interphalangeal joint collateral ligament. The varying orientation of fibers in the collateral ligament of the distal interphalangeal can be seen from the origin (bracket) on the middle phalanx and as the ligament extends distally to the insertion (box) at the fossae of the distal phalanx. In addition, there is variation in the fiber orientation at the level of the middle phalanx (within the bracket). These anatomic characteristics affecting the appearance of this ligament are imaged with ultrasound, MRI, and CT.

Identification of Lesions After Advanced Imaging

In most cases, once a lesion has been identified through the use of advanced imaging, it can readily be determined if the lesion should be visible through the use of traditional imaging modalities. This process increases our knowledge of anatomy and identifies structures or artifacts that can prevent visualization of a lesion when traditional imaging modalities are used. Retrospectively evaluating these lesions with the use of ultrasonography leads to the development of new skills and techniques. In certain cases, this process allows the diagnosis of these lesions going forward, without the benefit of advanced imaging. Several innovative ultrasound techniques have been validated through the use of advanced imaging and correlating the anatomic appearance of structures with gross and histologic evaluation.

Anisotropy: Understanding the Influence of Ultrasound Beam Angle

Fiber disruption and fluid accumulation in tendons and ligaments can be apparent with ultrasound. In contrast, other types of fiber injury can remain echogenic on ultrasound images despite clinically significant abnormalities. This discrepancy in the diagnostic capability of ultrasound during certain stages or types of injury may result in an ultrasound appearance that is not markedly different from normal, which can make certain types of injury challenging to diagnose with the use of this modality. Tissue echogenicity is determined by the acoustic properties (acoustic impedances) of that tissue. Variations in the acoustic properties and the resulting changes in echogenicity are used to differentiate normal and injured tissues.[8]

The term "anisotropic" in regard to ultrasonography simply means that the tissue exhibits different acoustic impedances when imaged at different angles. This principle is illustrated when imaging the flexor tendons in the transverse plane. When the probe angle is adjusted proximally or distally to obtain maximum echogenicity, the anisotropic properties of the tendons are demonstrated (Fig. 3). Obtaining a quality image in tissues of varying acoustic impedances is dependent on the ability of

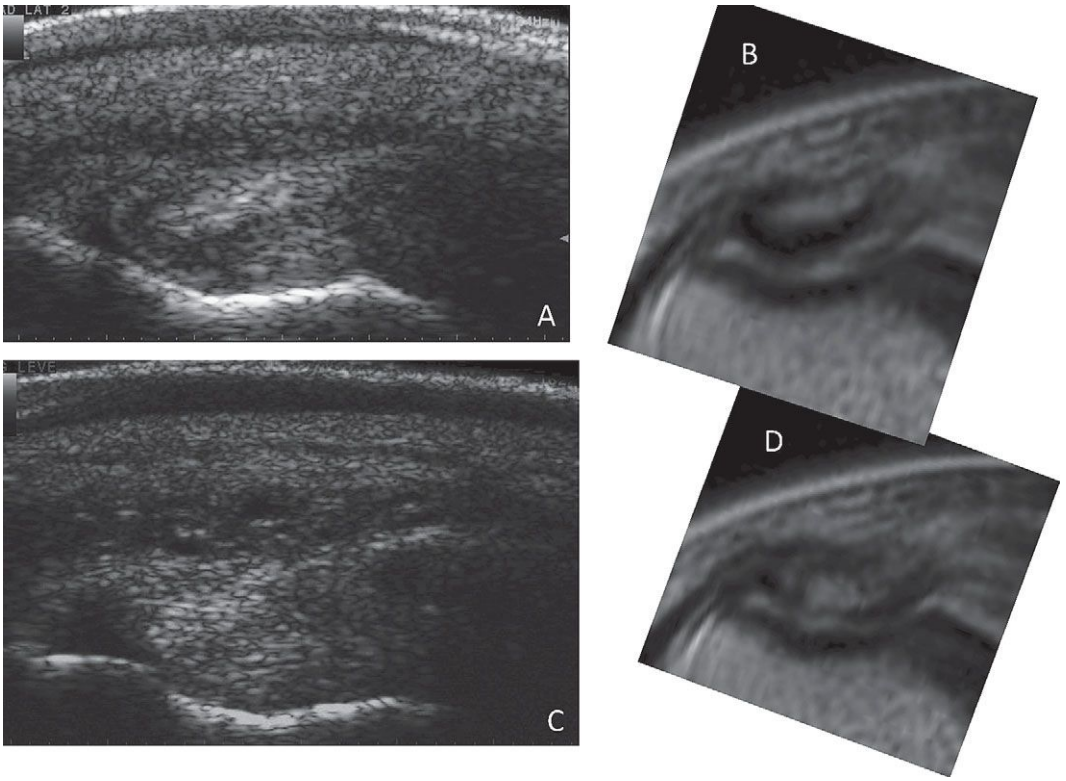


Fig. 2. Transverse ultrasound images (A, C) of a collateral ligament of the distal interphalangeal joint at the level of the middle phalanx with two different beam orientations and the corresponding MR images at the same level (B, D). On the ultrasound images, the central aspect of the ligament changes in echogenicity on the basis of the ultrasound beam angle. The appearance is representative of bundles of fibers with different orientations. This difference in fiber bundle orientation also results in the changes in signal intensity present on the MR images when the position of the limb is altered. The recognition of this ultrasound appearance was the result of the initial identification of the different fiber bundle orientation on MRI. Therefore, MRI was used to develop a more accurate method for evaluation of ultrasound images of the collateral ligaments of the distal interphalangeal joint at this level. Dorsal is to the left of each image.

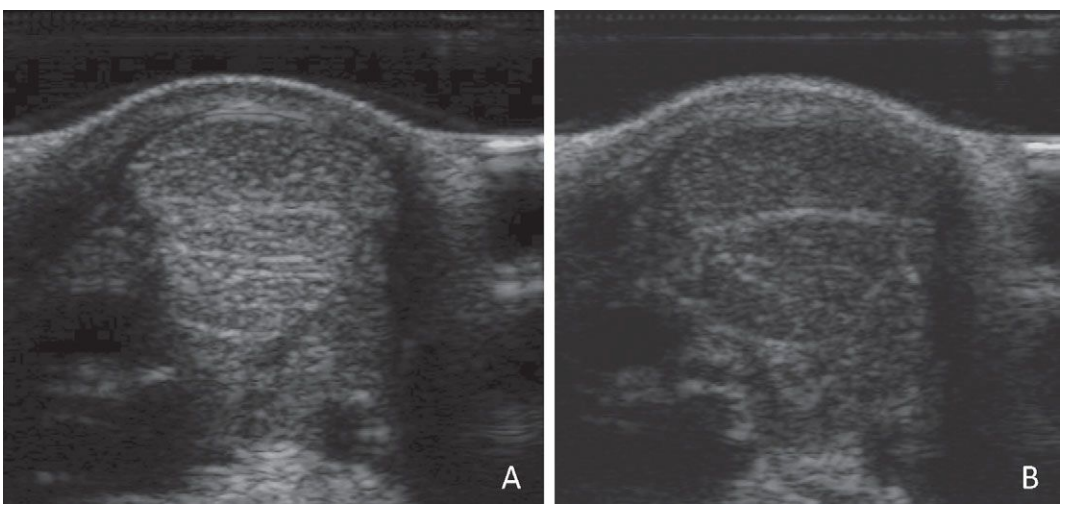


Fig. 3. Transverse ultrasound images of the superficial and deep digital flexor tendons at the level of the metacarpus that demonstrate the principle of anisotropy, or the dependency of echogenicity on beam angle. A, Ultrasound beam is perpendicular to the tendons creating diffuse echogenicity within the tendon. B, Ultrasound beam is off-angle or oblique incidence to the tendons. In this image, the echogenicity of the tendons are reduced compared with that in A. However, the tendon margins remain echogenic. Medial is to the left of each image.

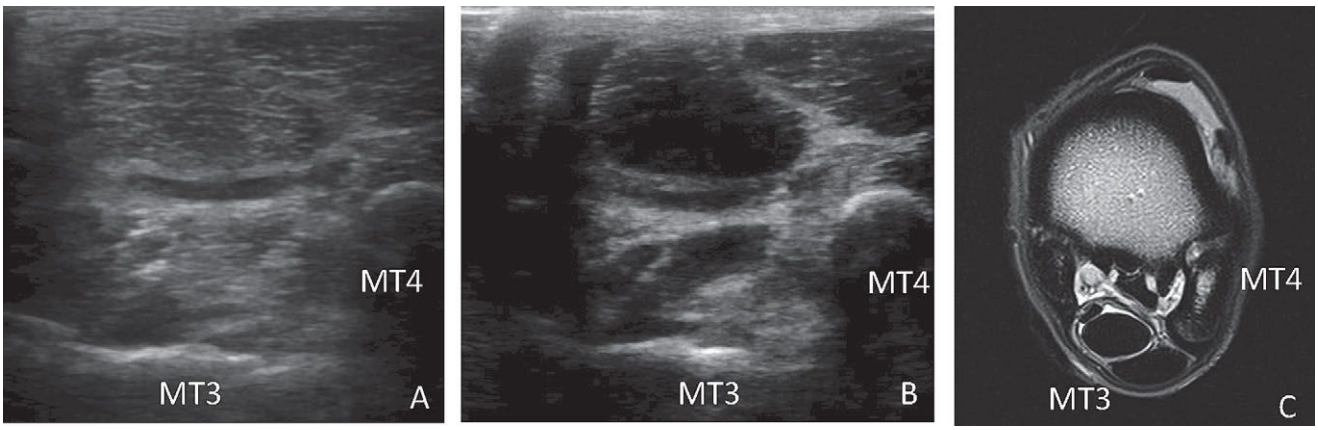


Fig. 4. Transverse ultrasound images (A, B) of the hind proximal suspensory ligament that were acquired with the horse resting on its toe. The beam was directed toward the plantar margin of the third metatarsal bone by use of on-angle (A) and off-angle (B) ultrasound beam orientations. C, Corresponding MR image at the same level. Note the adipose tissue and muscle bundles remain echogenic regardless of beam angle. Similar to that shown in Fig. 3, the ligament fibers are echogenic when the ultrasound beam is on-angle or perpendicular to the long axis of the tendon and are subsequently reduced in echogenicity when the ultrasound beam is off-angle. The peripheral margin of the ligament is visible when the ultrasound beam is off-angle.

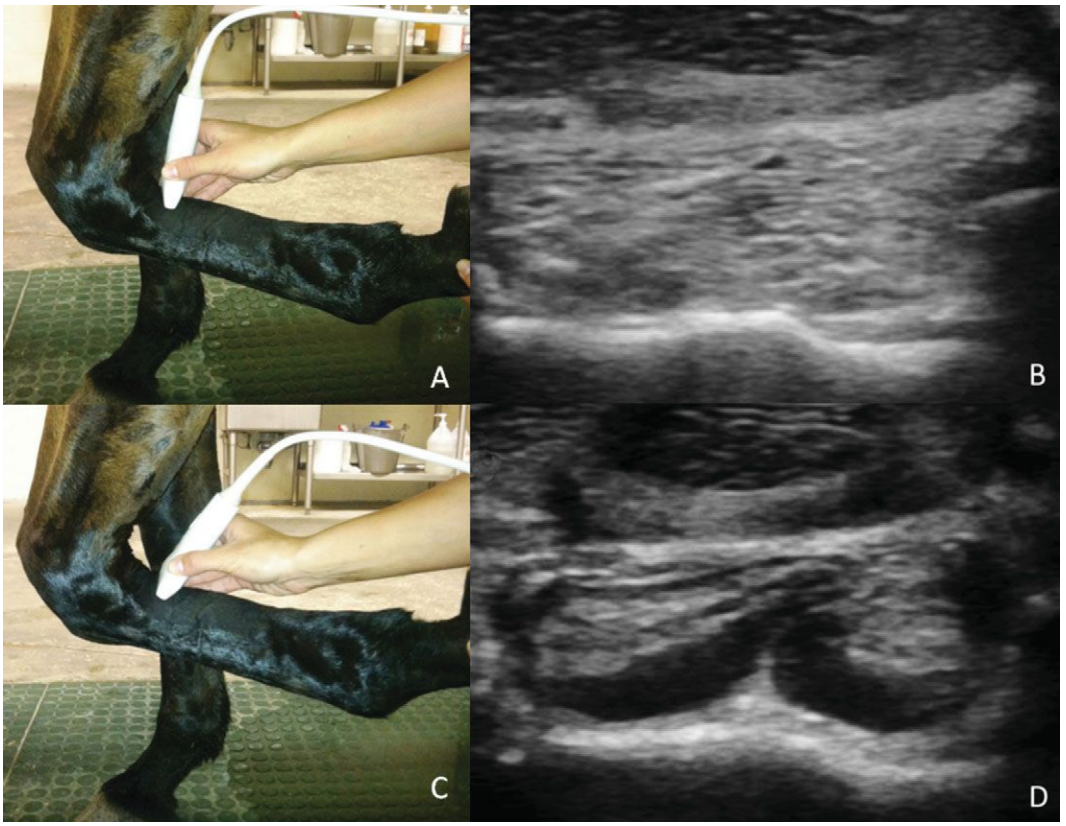


Fig. 5. Probe position and resulting ultrasound images for angle contrast (on- and off-beam angles) ultrasound technique when imaging the proximal suspensory ligament in the forelimb. A, Ultrasound beam is perpendicular to the proximal suspensory ligament in the metacarpus. B, Corresponding transverse ultrasound image of the suspensory ligament by use of this perpendicular beam angle. C, Ultrasound beam is oblique incidence to the proximal suspensory ligament. A change in hand position with angle change is required to achieve this image. D, Corresponding transverse ultrasound image of the suspensory ligament with this off-angle technique. Note the hypoechogenicity in the ligament fibers of image D and the hyperechogenicity of the muscle and fat bundles in the suspensory ligament regardless of beam angle (B, D).

the ultrasonographer to appropriately adjust the machine and settings (transducer, frequency, depth, focal zones, and gain). In addition, altering the direction of the beam angle is necessary to assess

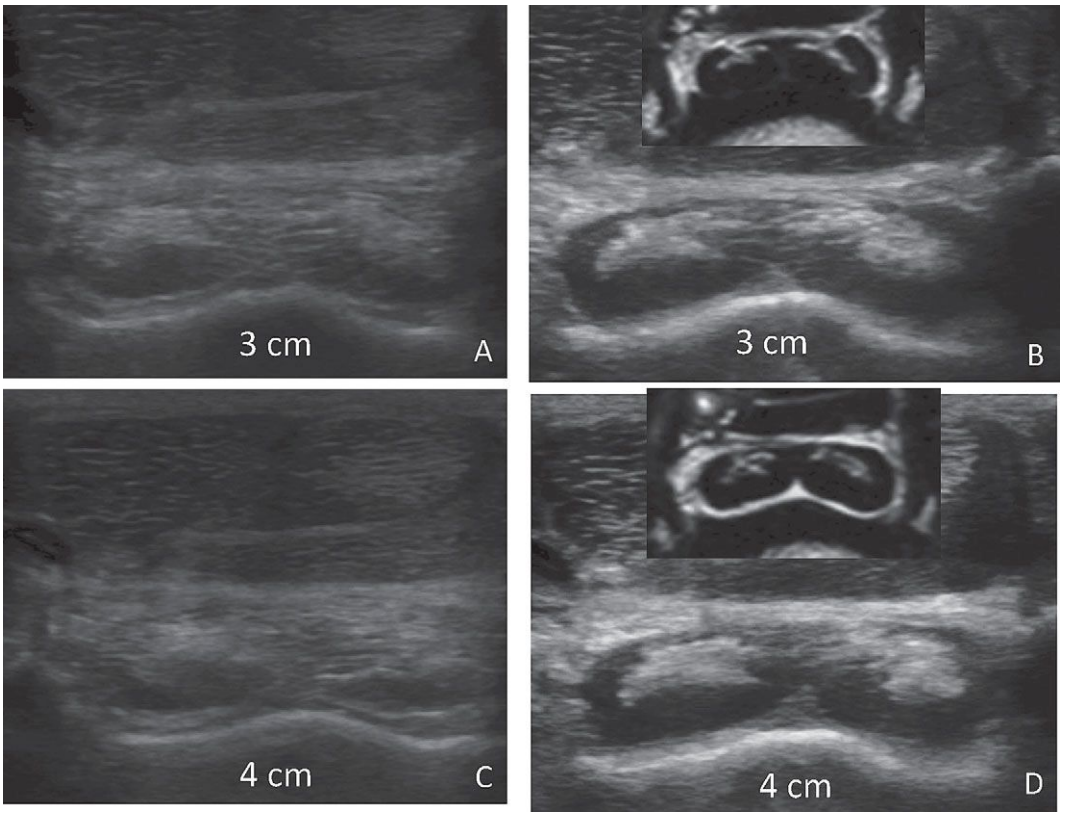


Fig. 6. Transverse ultrasound images of the proximal suspensory ligament in the forelimb. Ultrasound beam is perpendicular to the proximal suspensory ligament at 3 cm (A) and 4 cm (C) distal to the accessory carpal bone. Corresponding transverse ultrasound images at 3 cm (B) and 4 cm (D) distal to the accessory carpal bone with the ultrasound beam at an oblique incidence angle and MR images at the same level overlying these images. Note the hypoechogenicity in the ligament fibers (B, D) and hyperechogenicity of the muscle and fat in the suspensory ligament (A–D). There is decreased echogenicity in the dorsal aspect of the suspensory ligaments (A, C) with the ultrasound beam perpendicular to the longitudinal axis of the ligament. This appearance varies among horses with normal suspensory ligaments, and in this horse it is more marked than is typical. This appearance should not be confused with injury and may be the result of relaxation artifact. However, the dorsal aspect of the ligament is most frequently affected by injuries. Therefore, the dorsal aspect of the ligament should be carefully evaluated, and regions of decreased echogenicity should be assessed in conjunction with lobe size, shape, and margins along with comparison to the opposite limb.

the normal anatomy and to identify pathologic change. On the basis of the principles of anisotropy, the off-angle or oblique-incidence ultrasound technique can be used in structures with complicated anatomy to further elucidate the nature of the tissue and any associated anatomic characteristics as well as to aid in the diagnosis of pathologic change.[9] This technique and how it can be used to identify normal anatomy and diagnose musculoskeletal injury is discussed below.

Standard Ultrasound Technique

Ultrasound has traditionally been the imaging modality of choice for diagnosing most soft-tissue injuries because it is non-invasive, cost-effective, and easily accessible. The standard ultrasound technique has been described for the equine distal limb in scholarly veterinary publications and textbooks.[1,10–16] The majority of musculoskeletal imaging in the equine distal limb is performed with a linear transducer.[17] The ultrasound probe is placed on the palmar or plantar surface of the limb with the beam oriented perpendicular to the longitudinal axis of the tendons or ligaments of interest with the limb in a weight bearing position. In addition, transverse images are obtained. The size, shape, margins, and echogenicity of the structures should be evaluated and a measurement system, with the use of distance (cm) or zones, should be used to obtain images at regular intervals. Comparison to the contralateral limb is a critical step in most cases, even those with obvious injury in the primary limb. This system will facilitate sequential examinations. In certain cases, the medial and lateral extent of the tendons and ligaments cannot be imaged in the same field of view, and the probe should be directed toward the medial and lateral aspects of the structure to obtain peripheral margin images.

The standard ultrasound technique (single-beam angle with the limb in a weight bearing position)

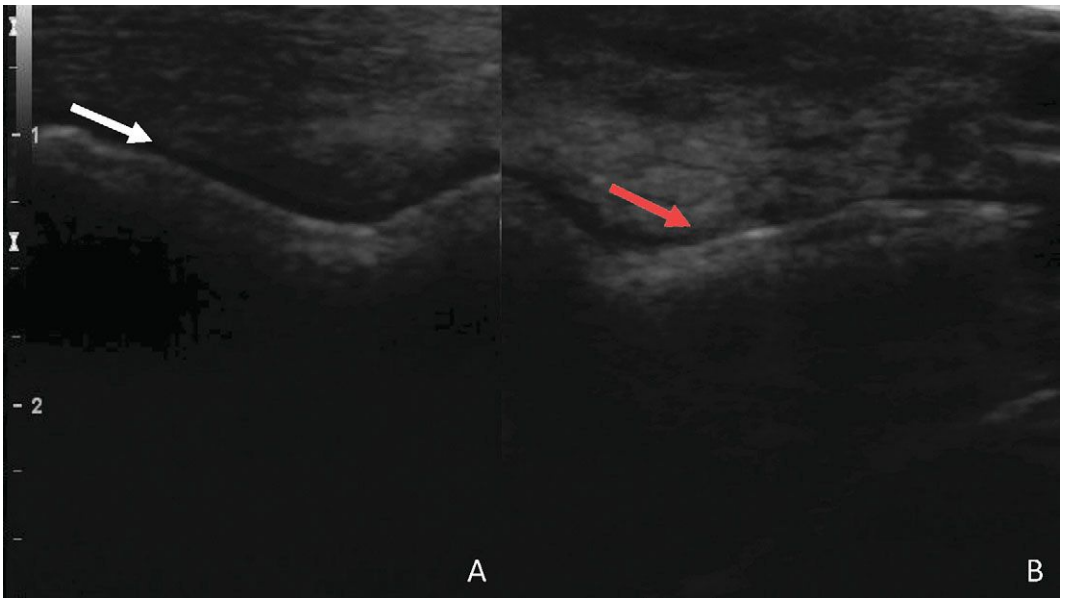


Fig. 7. Transverse ultrasound images of the third metacarpal bone with the fetlock flexed. A, The dorsal aspect of the lateral condyle of the third metacarpal bone has a clearly defined hypoechogenic line of articular cartilage (white arrow) with a smooth osseous margin. B, The medial condyle of the third metacarpal bone has an irregular articular surface (red arrow) and osseous margin. In addition, the region of the articular cartilage is echogenic, indicating cartilage abnormality such as fibrosis or degeneration. These findings were not evident with the limb in a weight bearing position.

does not always demonstrate important anatomic features of tendons and ligaments. This technique was selected to create maximum echogenicity in the structure, to prevent decreased echogenicity caused by variations in beam angle (anisotropy) that could be mistaken for a lesion. In addition, this technique was designed to prevent relaxation artifact from causing false decreases in echogenicity that could be mistaken for a lesion. However, decreased tension on certain structures may actually increase the conspicuity of lesions. The angle contrast ultrasound technique uses perpendicular and oblique beam (on- and off-beam) angles to identify bundles of fibers with different orientations. This technique, along with changes in limb position and weight bearing, can be used to identify abnormalities that are less apparent or not visible with the standard technique.

Angle Contrast Ultrasound Technique (On- and Off-Beam Angles)

The angle contrast (on- and off-beam angles) ultrasound technique is typically used after examination with the standard ultrasound technique. To perform the angle contrast ultrasound technique, the ultrasound beam is placed at perpendicular and oblique angles of incidence relative to the longitudinal axis of the tendon or ligament fibers. To achieve an oblique incidence image, the ultrasound beam is first positioned perpendicular to the tendon or ligament to create maximum echogenicity, and this image is saved and compared with an oblique incidence image at the same level (Fig. 4). To create the oblique incidence image, the probe angle is then changed by moving the probe cable proximally or distally, depending on the structure being imaged and the specific region of interest (Fig. 5). The least amount of angle that results in decreased echogenicity of the tendon or ligament fibers should be used. The shape, size, and location of the imaged structure and any other anatomic landmarks, such as osseous margins, in the oblique incidence image should be compared with the original perpendicular beam angle image. The shape, size, and location of the soft tissue and osseous structures should match in the two different images to ensure that similar levels of the tendon or ligament are being evaluated and can then be compared. The angle contrast ultrasound technique has been incorporated as part of the standard examination in all musculoskeletal examinations by the author.

This technique has proven to be effective when imaging the suspensory ligament through the use of ultrasound. It facilitates differentiating regions of fibers from adipose tissue and muscle (Fig. 6).[9] Anisotropic properties of tendon and ligament fibers differ from that of adipose tissue and muscle. Muscle echogenicity is much less dependent on beam angle compared with tendon and ligament fibers. Adipose tissue echogenicity is not dependent on beam angle. Regions of adipose tissue, and, to a lesser extent, muscle, will remain echogenic regardless of beam angle. In contrast, tendon and ligament fibers will become hypoechogenic when the beam angle is not perpendicular to the longitudinal

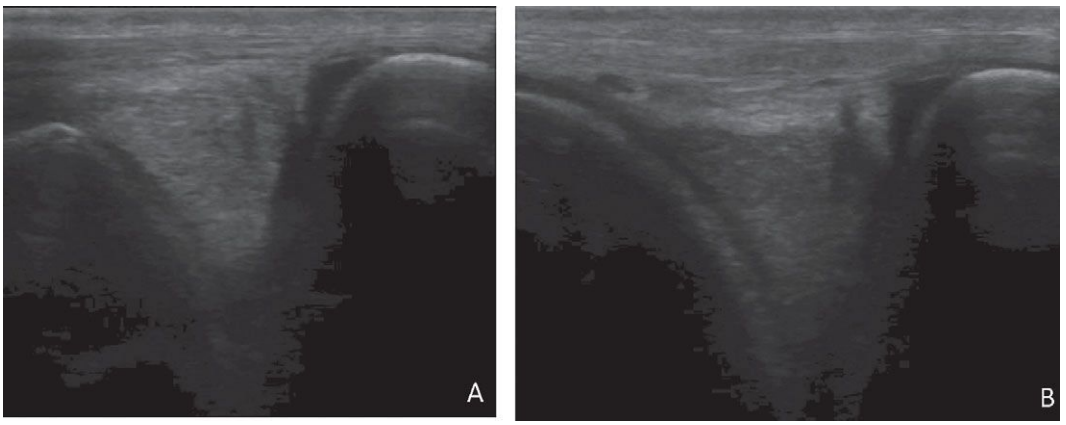


Fig. 8. Transverse ultrasound images of the cranial aspect of the medial meniscus. A, Weight bearing image shows two regions of decreased echogenicity in the distal aspect of the medial meniscus with an irregular tibial margin. B, Non–weight bearing image shows enlargement and coalescence of the previously identified regions of abnormality with a further decrease in echogenicity consistent with presence of fluid and fiber loss, indicating a tear in the medial meniscus.

axis of the tendon or ligament fibers. By use of variations in the ultrasound beam angle, regions of scarring and injury in soft-tissue structures can be identified. Scarring is echogenic regardless of ultrasound beam angle. Areas of fiber disruption are anechoic regardless of beam angle. Therefore, comparing the echogenicity of soft tissues structures with on-beam angle (perpendicular) and off-beam

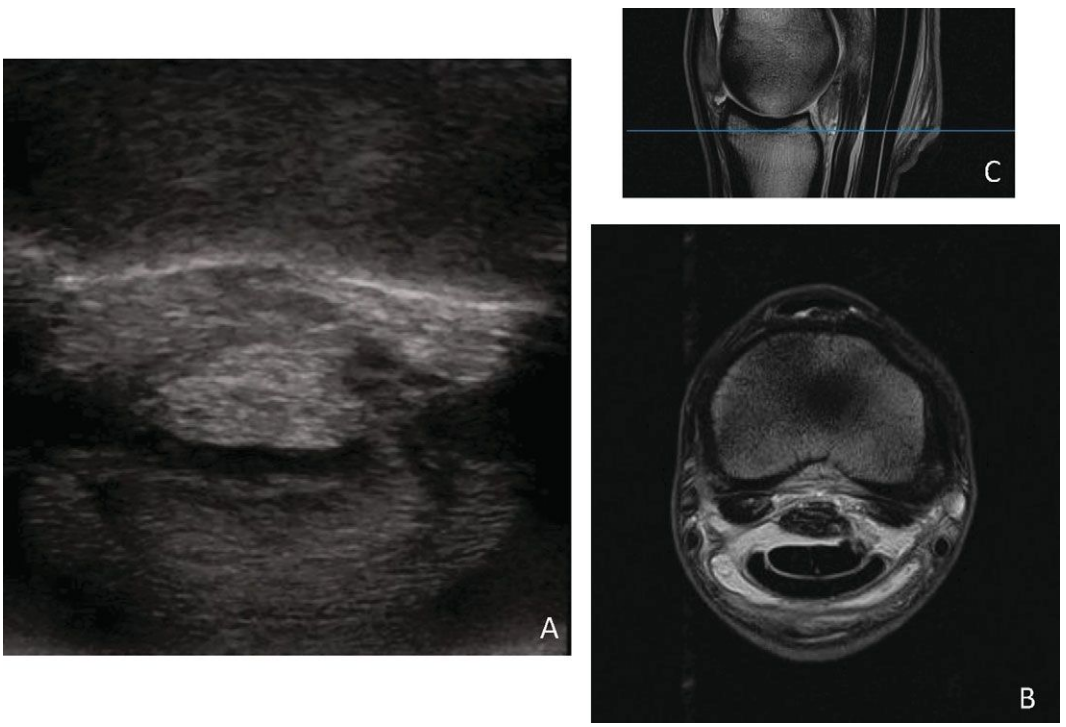


Fig. 9. Ultrasound (A) and MR (B, C) images of an 11-year-old Dutch Warmblood mare that presented for right forelimb lameness localized to the digital sheath. No significant soft tissue lesions were identified on initial ultrasound examination. High-field MRI was performed under general anesthesia of the left fore fetlock region and revealed abnormalities in the deep digital flexor tendon and straight sesamoidean ligament with associated synovial proliferation. A, On retrospective ultrasound examination, these abnormalities could be identified after flexion of the limb and manual displacement of the ergot. B, MR image at the level of the injuries. C, Localizer image denoting the slice position relative to the ergot. Note that tissue manipulation of the ergot is necessary to fully examine the tendons at the level of the fetlock. Medial is to the left of each image.

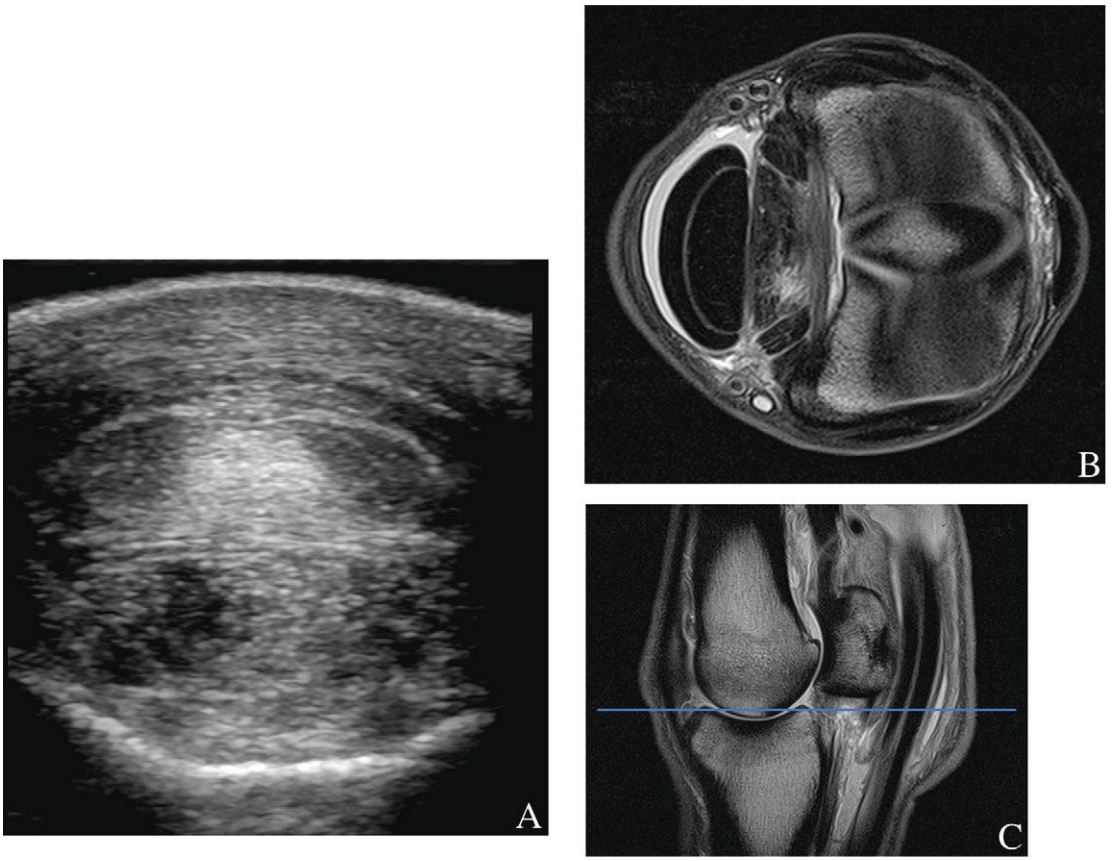


Fig. 10. A, Transverse ultrasound image of the straight sesamoidean ligament at the level of the junction with the intersesamoidean ligament. B, Corresponding magnetic resonance image at the same level. C, Localizer image. This lesion appeared smaller when imaged with the limb in a weight bearing position and was more difficult to visualize. With the limb in a non–weight bearing position, the lesion size was similar when comparing ultrasound and MR images and the lesion was readily identified.

angle (oblique) ultrasound beam orientations can be used to characterize regions of pathologic change from normal anatomic variation.[14]

Limb Position, Tissue Manipulation, and Probe Pressure

Limb position can affect visualization of certain structures. When examining the palmar or plantar soft tissues at the level of the pastern and the foot, have the horse standing squarely on all four limbs and then place the foot of interest caudal from this square stance to facilitate examination. Visualization of the medial aspect of the elbow with ultrasound can be greatly improved with the limb extended. The degree of extension is variable. The location of the lesion or area of interest within this region of the elbow will affect the degree of extension that is necessary. In contrast, examination of the medial aspect of the stifle is best performed with the limb placed squarely underneath the body. When the limb is positioned forward or behind, the vertical visualization of certain structures of the stifle is limited.

Evaluating the limb in weight bearing and non–weight bearing positions can provide additional information and aid the diagnosis of pathologic change. In certain anatomic regions, standard and angle contrast ultrasound techniques can be combined with changes in weight bearing and tissue manipulation to achieve more information. This is especially true when imaging the front and hind limb suspensory ligaments. Placing the limb in a non–weight bearing position has many advantages. The decreased tension in the flexor tendons allows manipulation of their position. They can be manipulated to create a wider skin surface for the ultrasound probe, increasing visualization of the medial and lateral aspects of the ligaments (Fig. 4). Vasculature that may have been creating artifact with the limb in a weight bearing position can often be manipulated to decrease the amount of artifact. In addition, this process decreases the depth between the probe and the suspensory ligament, which often allows use of a higher frequency that increases image resolution.

For ultrasonographic examination of the proximal metacarpal region, the limb is placed in a non–weight bearing position with the lower leg suspended and minimal carpal flexion while the metatarsal region can be examined with the horse

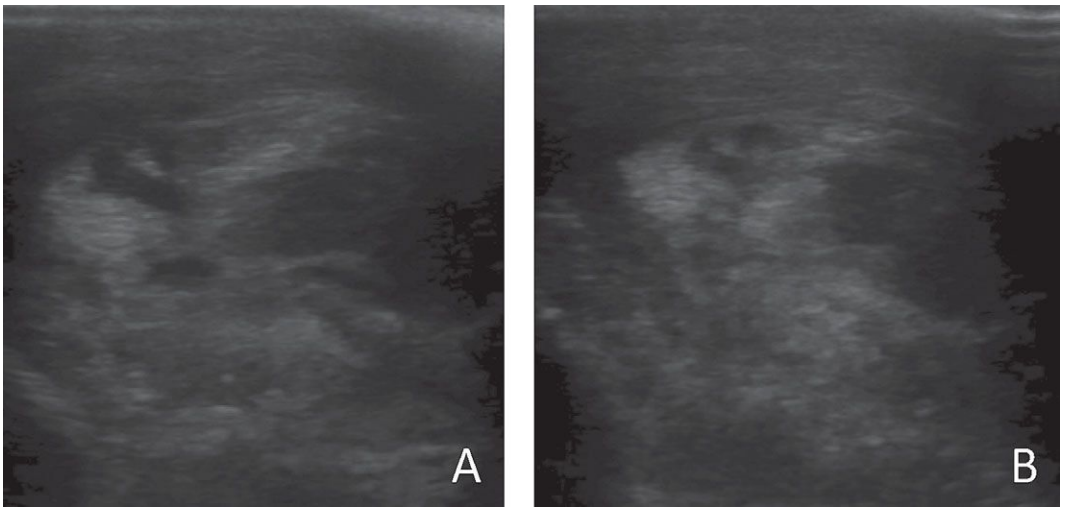


Fig. 11. Transverse ultrasound images of a tear in the lateral aspect of the superficial digital flexor tendon both acquired with the limb in a non–weight bearing position. A, The ultrasound probe is positioned such that it is in contact with the skin surface. However, no pressure is applied to the probe and the tear is quite evident. B, With pressure on the ultrasound probe, the tear in the superficial digital flexor tendon appears smaller, is less conspicuous, and does not obviously extend through the plantar margin. This injury was less evident with the limb in a weight bearing position.

resting on the toe, in most cases. Once images of the proximal metacarpus have been obtained, the carpus can be flexed further to maintain a more comfortable position. When imaging the proximal metatarsal region, placing the limb of interest in a non–weight bearing position (resting on the toe) can allow flexor tendon manipulation. However, increased limb flexion results in greater tendon laxity, which can be beneficial when tendon manipulation cannot be adequately performed with the horse resting on its toe.

A complete ultrasonographic examination of joints often requires imaging the joint in flexion after examination of the joint in a weight bearing position. When examining the fetlocks and carpi, joint flexion allows visualization of regions of the joint that are not accessible with ultrasound when the limb is in a weight bearing position. This position aids in identification of the dorsal articular surfaces of the medial and lateral condyles of the third metacarpal bone in the fetlock (Fig. 7). This examination is an excellent adjunct to radiographs, especially if an abnormality is suspected on the basis of radiographic appearance. In the stifle joint, flexion of the limb is required to examine the cranial tibial meniscal ligaments and to identify certain types of meniscal tears. This can often be achieved with the horse resting on its toe. However, visualization of the distal margin of the medial femoral condyle and the distal aspect of the cranial cruciate ligament is best achieved with more pronounced joint flexion. In addition, small meniscal tears may become more apparent, revealing the maximum extent of their margins with greater joint flexion. After evaluation with the horse resting on its toe, it is best to reevaluate any findings with more pronounced flexion of the joint. This technique is used to determine if the findings change with different limb positions or degree of flexion. Examination of the stifle joint with the limb in weight bearing and non–weight bearing positions allows protrusion of the meniscus to be more easily identified. In many cases, the meniscus will be further displaced outside the joint margins with the limb in a weight bearing position and then will return within the joint space with the limb in a non–weight bearing position. Ultrasonographic examination can be used to reliably identify the menisci, collateral ligaments, cranial meniscal ligaments, and the distal portion of the cranial cruciate ligaments.[18]

In general, a lack of tension in soft-tissue structures can increase the visibility of lesions, especially when adjacent fluid can enter regions of fiber disruption. Although this has been shown repeatedly when evaluating meniscal tears in the stifle (Fig. 8), reduced pressure can also aid in the diagnosis of soft-tissue injury in other anatomic regions. The benefit of this technique is somewhat related to lesion configuration. However, it appears to increase the conspicuity of many different types of injuries being diagnosed with ultrasound. Longitudinal splits and margin tears are substantially affected by this technique. This premise has developed from comparing ultrasound and MRI images of lesions. Although there are certain types of injury that can be identified with MRI that will not be evident on ultrasound images, other lesions, especially those with fiber disruption, should be evident with ultrasound if the structure is accessible. When MRI lesions with fiber disruption are identified, they may appear smaller when imaged with ultrasound. This can occur when the periphery of the lesion is

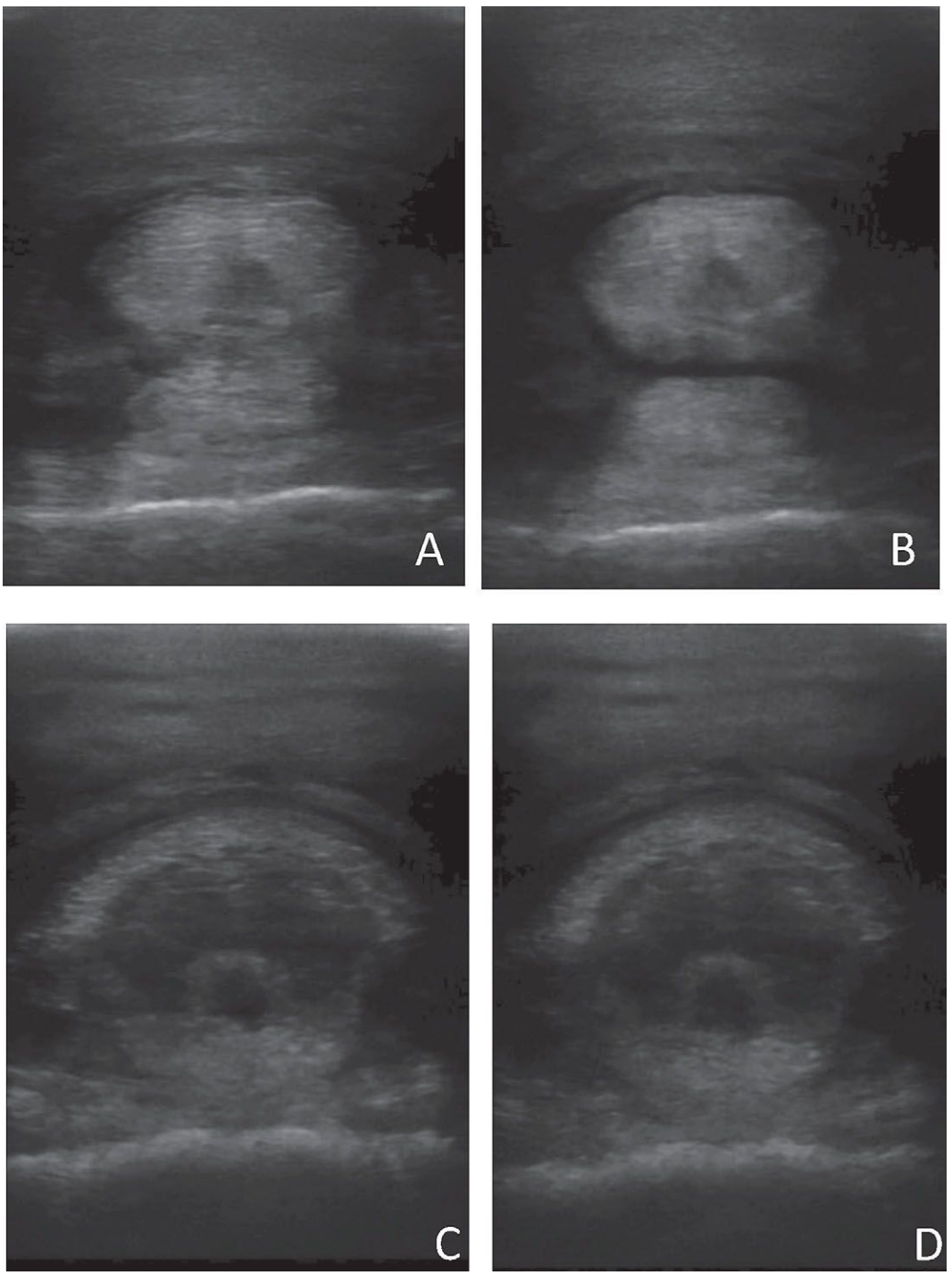


Fig. 12. Transverse ultrasound images at the level of the pastern made with the limb in a non–weight bearing position. Possible adhesions exist between the deep digital flexor tendon and the straight sesamoidean ligament in the left forelimb distally (A, B) and more proximally (C, D). Ballottement can be used to determine if adhesions are present. Digital pressure on the sheath can displace structures. Fluid completely separates the deep digital flexor tendon and the straight sesamoidean ligament (B) after ballottement and manipulation of the fluid in the digital sheath. However, the deep digital flexor tendon and the straight sesamoidean ligament do not separate in the more proximal image (D) indicating adhesion formation. The proximal images (C, D) of the deep digital flexor tendon are off-angle.

less severely affected and will therefore be more apparent on MR images when compared with ultrasound images. However, in certain cases, this discrepancy appears to be the result of differences in tension on the basis of weight bearing. These differences are most apparent when comparing recum-

bent MR images with standing ultrasound images. In these cases, the lesions are more similarly sized and shaped when MR images obtained in a recumbent horse are compared with ultrasound images with the limb in a non–weight bearing position.

Comparison of MRI and ultrasound images also reveals lesions in anatomic regions that are ultrasound accessible. However, additional steps must be taken to adequately visualize these regions with ultrasound if these regions have not been part of the standard ultrasound examination previously. The palmar or plantar fetlock under the ergot is one of these regions. At the level of the ergot, there are many important anatomic structures on midline, such as the straight and intersesamoidean ligaments and the cruciate ligaments. This region can be obscured by the ergot and is more challenging because of the curvature of the limb and ligaments at this level. Complete examination of the region can require both flexion and tissue manipulation (Fig. 9). Flexion results in relaxation of the palmar or plantar soft tissues, whereas manipulation of the ergot allows visualization of these midline structures. Lesions of the straight sesamoidean ligament at the junction with the intersesamoidean ligament are frequently identified retrospectively after MRI with the use of this technique (Fig. 10). Regions that may not have been specifically imaged with previous ultrasound include fibers of the hind suspensory ligament that extend proximally to the fourth tarsal bone. This area demonstrates injury on MRI images and is ultrasound accessible. In this case, adjusting the proximal anatomic limits of a hind suspensory ligament ultrasound study will facilitate identification of lesions in this region.

As previously discussed, limb position and tension on soft-tissue structures can affect the visibility of lesions. In addition, probe pressure can also markedly affect the visibility of certain lesions. Changes in the amount of probe pressure at the region of interest can be used to increase the conspicuity of certain lesions (Fig. 11). Alterations in probe pressure in combination with tissue manipulation are a benefit, especially when evaluating the digital sheath. With the limb in a non–weight bearing position, digital pressure can be used to force fluid into different regions of the sheath to aid in determining the presence or absence of adhesion formation (Fig. 12).

Summary and Conclusions

Although ultrasound has limitations compared with advanced imaging, the continued development of innovative techniques will aid the diagnosis of musculoskeletal injury in horses. Advanced imaging provides the best method for the development of these techniques and furthers our educational process about imaging and the pathologic changes associated with musculoskeletal injury.

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How to Inject the Medial Femorotibial Joint Recess Under Ultrasound Guidance

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1. Introduction

Injection of the medial femorotibial (MFT) joint is frequently a requisite for the diagnosis and management of equine lameness. The technique is important for the localization of lameness through diagnostic analgesia[1] and the determination of the significance of pathology when identified on other diagnostic tests (imaging or arthroscopy). Furthermore, joint injection is an inherent component of lameness management through the administration of intra-articular medications ranging from chondroprotective agents, anti-inflammatories, and, more recently, biological products. Although several different injection techniques have been described, each method has some disadvantages. To circumvent these disadvantages, the author has developed and will describe a technique for ultrasound-guided injection of the MFT joint that can be performed solo.

Ultrasonographic examination reveals that the size and depth of the MFT joint recess varies.[a] Minimal effusion or fibrosis may result in a smaller recess. Placement of a needle properly within the recess may not allow fluid to become visible at the needle hub because proliferative synovial membrane may plug the needle or the needle size may be too small relative to the viscosity of the synovial fluid. If fluid is noted, the attachment of the needle to the syringe followed by injection does not guarantee proper deposition of the medication within the recess. Movement of the needle too deeply results in deposition of the product beneath the synovial membrane and too superficially into the subcutus.

Recently, a detailed description of an alternative technique for injection of the MFT joint and relevant anatomy has been presented.[2] This technique utilized a standard 1.5-inch, 20-gauge needle to inject the MFT joint recess. The advantages of the technique is that injecting the MFT joint recess, which is located proximal to the meniscus, decreases the potential of needle contact with cartilage or the medial meniscus. The disadvantages are (1) it is a blind technique relying on anatomical landmarks that may vary with limb positioning and (2) the technique is dependent on noting fluid to be present at the hub of the needle before injection.

Validation of a cranial injection technique with the use of cadaver limbs was recently published[3] that compared a cranial approach with a medial approach to the MFT joint. However, the former was successful in approximately 50% of cases, whereas the latter was successful in only 93% when no effusion was present.

NOTES

The technique for ultrasonographic examination of the MFT joint has been well described.[3–6] Ultrasound examination allows determination of the location, size, and depth of the MFT joint recess. Therefore, we used ultrasound monitoring to prevent inadvertent injection of the medial recess of the femoropatellar joint.

This study describes the technique for solo ultrasound-guided injection of the MFT joint recess and overviews its use in 147 injections of 77 clinical cases.

2. Materials and Methods

Seventy-seven horses undergoing this technique were tracked prospectively. Data were collected on type of injection (diagnostic, therapeutic), dosage and volume administered, frequency of injection, and any complications.

Technique

The site for injection of the MFT joint recess is proximal to the medial meniscus, cranial to the medial collateral ligament, and caudal to the medial patellar ligament.

The tail was wrapped, and the area was aseptically prepared. Clipping was usually unnecessary in this area because of the sparsity of hair. The ultrasound machine was positioned caudally for convenient viewing. We routinely used a 7.5-MHz linear transducer and prepared it with acoustic gel and a sterile probe cover. A sterile syringe was filled with the anesthetic, contrast, or medication to be used for the intra-articular injection, and a new 20- or 22-gauge needle was aseptically preplaced onto the syringe.

When this procedure was used for the purpose of desensitizing the MFT joint during a lameness examination, immobilization of the hind limb was frequently accomplished by lifting the ipsilateral forelimb. The horse was distracted at the time of needle insertion by having the owner feed the horse some grain or horse treats. Most horses acclimatized to having their stifles handled when given food during the scrubbing process. This form of restraint was preferred to a twitch, which was only used in the horse that was not food-motivated and/or remained unruly. Horses that were difficult to jog received an extremely low intravenous dose of detomidine HCL[b] (0.002 mg/kg). This allowed for more consistent jogging and less apprehension during the blocking procedure. Warm alcohol was applied to the injection site and used as the external acoustic medium between the probe, within its sterile sheath, and the skin. The ultrasound probe was placed longitudinally over the medial recess (Fig. 1) by use of the three anatomical landmarks previously mentioned as references. Once the recess was identified on the screen (Fig. 2) the probe was then rotated 90° into a transverse position (Fig. 3). The probe could then be moved slightly proximally or distally to ensure positioning over the most distended part of the recess (Fig. 4). A subcutaneous 2-mL "bleb" of 2% lidocaine HCL[c] was injected adjacent to the cranial margin of the ultrasound probe (Fig. 5) with the use of a 27-gauge needle. This

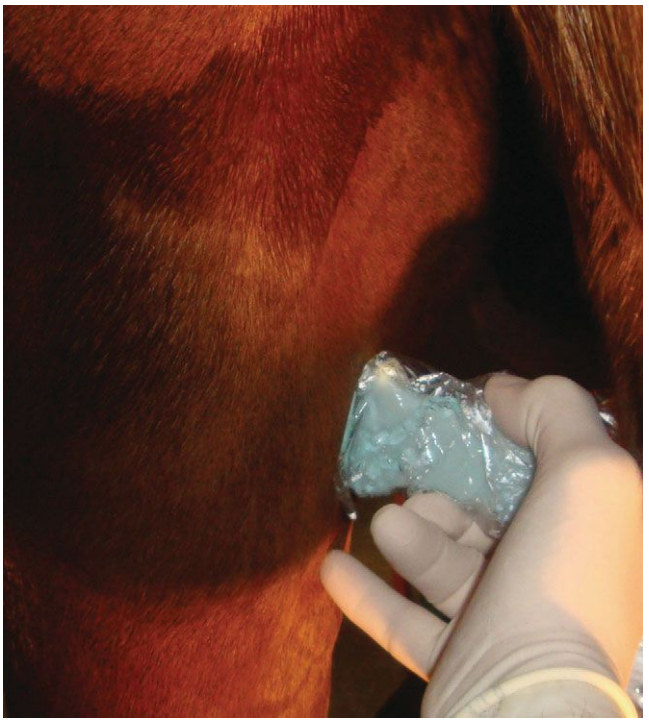


Fig. 1. Step 1: Longitudinal positioning of the ultrasound probe and centering it on the MFT joint recess.

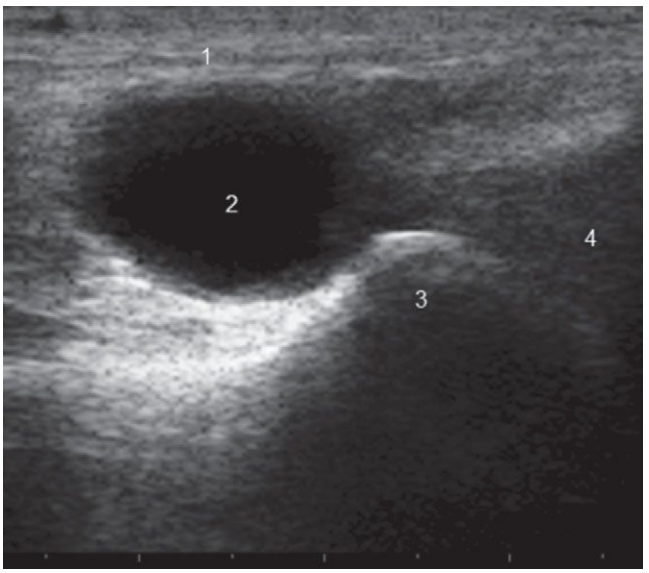


Fig. 2. Longitudinal reference sonogram of the MFT joint recess through the use of the craniomedial approach pictured in Fig. 1. Proximal is to the left and distal to the right. 1, Femoral fascia; 2, MFT joint recess; 3, femoral condyle; 4, medial meniscus.



Fig. 3. Step 2: Rotate the ultrasound probe 90° into a transverse position, staying centered over the MFT joint recess.



Fig. 5. Step 3: Inject a 1- to 2-mL bleb of local anesthetic cranial and adjacent to the probe.

"bleb" was used as a landmark (Fig. 6) for replacement of the probe once the local anesthetic had taken effect and decreased any further discomfort that could arise from introduction of the needle used for injection. The syringe with the 22-gauge needle attached was slowly introduced through the skin (Fig. 7) at the cranial margin of the ultrasound probe. The needle was pushed through the femoral fascia into the recess, and the plunger was depressed (Fig. 8). The contents of the syringe were observed to enter the recess (Fig. 9). The syringe and needle were then withdrawn.

When the procedure was used to administer medications or for a contrast study of the MFT joint, the horse was sedated intravenously with detomidine HCL[b] (0.01 mg/kg) and butorphanol tartrate[d] (0.01 mg/kg). Because food is not an incentive to a sedated horse, the level of sedation was tested before injection by carefully palpating the outer sheath or

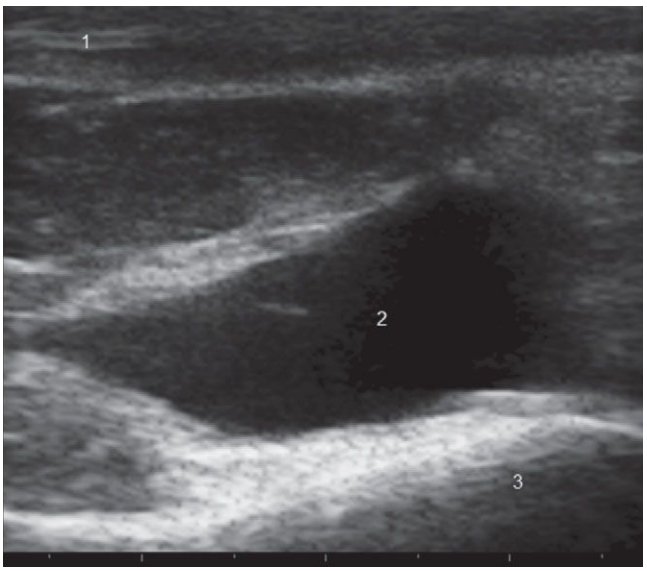


Fig. 4. Transverse reference sonogram of the MFT joint recess through the use of the craniomedial approach pictured in Fig. 3. Cranial is to the left and caudal to the right. 1, Femoral fascia; 2, MFT joint recess; 3, femoral condyle.



Fig. 6. Step 4: Replace the probe transversely with the skin bleb used as a landmark.

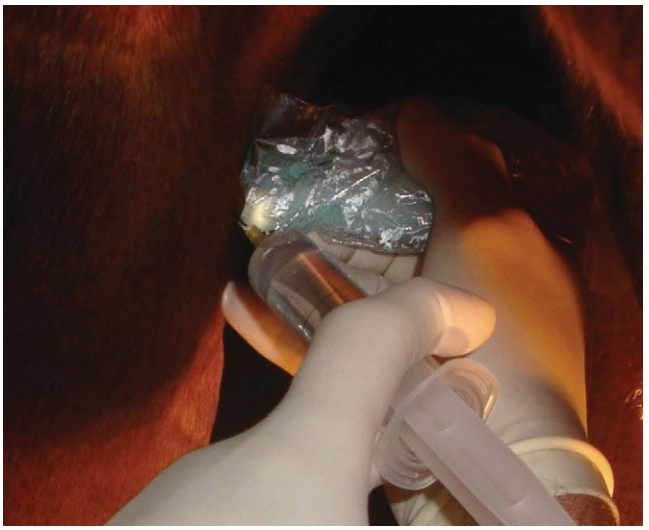


Fig. 7. Step 5: While maintaining a parallel alignment between the syringe and ultrasound probe, slowly drive the syringe needle combination caudally through the skin bleb at approximately 30° when using a linear probe.

mammary area. Additional detomidine HCL[b] (0.005 mg/kg) was given if required. Local anesthesia was frequently not necessary when this level of sedation was used. The technique was similar except that a slightly larger needle (20-gauge) was used to allow for easier injection of more viscous products. If the area was not desensitized, putting slight pressure on the cranial aspect of the probe at the time of needle insertion helped to prevent a surprise reaction from the horse.



Fig. 8. Step 6: Injection is commenced after entry through the synovial membrane.

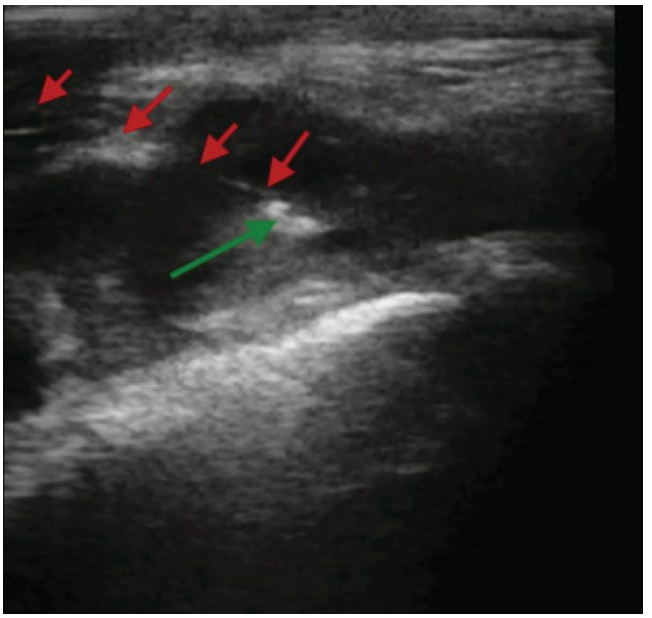


Fig. 9. Transverse sonogram of the MFT joint recess. The needle is observed within the MFT joint recess (red arrows) and turbulent echogenic material (green arrow) is noted being ejected from the needle tip into the fossa. Cranial is to the left and caudal to the right.

The operator resisted the temptation of becoming "mesmerized" by the screen and focused on maintaining a parallel alignment between the syringe and probe. Malalignment resulted in losing sight of the needle, which also could occur by simply having a wet needle that is not sufficiently echogenic. Injection of a small test volume allowed visualization of fluid swirling and movement of echogenic gas/medication within the recess. The echogenicity of products that were not very echogenic was enhanced by adding a small volume of air to the contents of the syringe when preparing the injections.

The technique is versatile and can be performed with a microconvex or linear probe. A rectal probe is slightly more difficult to hold but has the advantage that no chord projects into a male horse's sheath.

3. Results

One hundred forty-seven ultrasound-guided injections of the MFT joints were performed on 77 horses over a 3-year period. The signalment included seven Thoroughbreds, 12 Standardbreds, nine Quarter Horses, and 49 Warmbloods, including crosses. There were 59 geldings, 16 mares, and two stallions. The median age was 8 years, with a range from 2 to 18, and the average age was 8.4 years.

The time period between repeat injections into the same joint varied from 1 day to 3 years. The left MFT joint was injected in 49 horses and the right MFT joint was injected in 20 horses. Fifty-four



Fig. 10. Contrast study of the MFT joint. Note the distended MFT joint recess and no communication with the femoropatellar joint in this horse.

horses had bilateral injections. Seventy-seven MFT joints received a single injection and 25 MFT joints received multiple injections. Twenty-nine horses had additional ultrasound-guided injections of the femoropatellar (FP) and/or lateral femorotibial (FT) joint in conjunction with the MFT joint(s).

Injectable products used were therapeutic and diagnostic. Therapeutic agents included corticosteroids,[e,f,g,h] chondroprotective agents,[i] and biological agents.[j] Diagnostic products included local anesthetics[c,k] and contrast media.[l] Total volumes and products were variable. The total volume injected ranged from 3 to 20 mL. Combined products never exceeded a volume of 15 mL, whereas single diagnostic agents were injected at volumes up to 20 mL.

We found that ultrasound-guided needle placement and injection allowed monitoring of the needle position and accurate drug deposition. Last, the technique was not dependent on the presence of effusion or on fluid acquisition; therefore, a smaller needle could be used, which is less painful and less traumatic.

Insertion of the needle with the syringe already attached was not problematic because the horse was either sedated or had the insertion site desensitized with local anesthetic. Inadvertent movement resulted in a temporary delay in administration, needle withdrawal, or positional change but no needle breakage. Movement was rare if the described restraint procedures were followed. No infections occurred in this study. A slight learning curve was necessary to depress the plunger of the syringe with the left hand for a right-handed operator when injecting the horse's right stifle. Sitting on a rolling mechanic stool allows one's hands to remain steady and maintain more consistent alignment of needle and probe. Confirmation of the medication being injected into the recess was immediately noted by visualizing the medication exiting the needle on the ultrasound screen.

4. Discussion

Reasons for injecting the MFT joint include desensitization for the purpose of lameness diagnostics, therapeutic administration of medications, or contrast studies. Contrast studies were recently initiated to see if there is communication between the femoropatellar and MFT joint for the purpose of selective administration of therapeutic medications such as interleukin-1 receptor antagonist protein[j] (Fig. 10).

This technique is similar to the blind technique previously described,[1] with the addition of ultrasound monitoring with the use of an ultrasound probe placed caudal to the insertion site. This ultrasound-guided technique was developed because of inconsistency or difficulty with previous techniques. Fluid is not always visible or obtainable when a blind technique is used. Obtaining fluid does not always provide assurance that the MFT joint has been penetrated because the medial recess of a distended femoropatellar joint can lie adjacent to the MFT joint recess and may be inadvertently penetrated (Fig. 11).

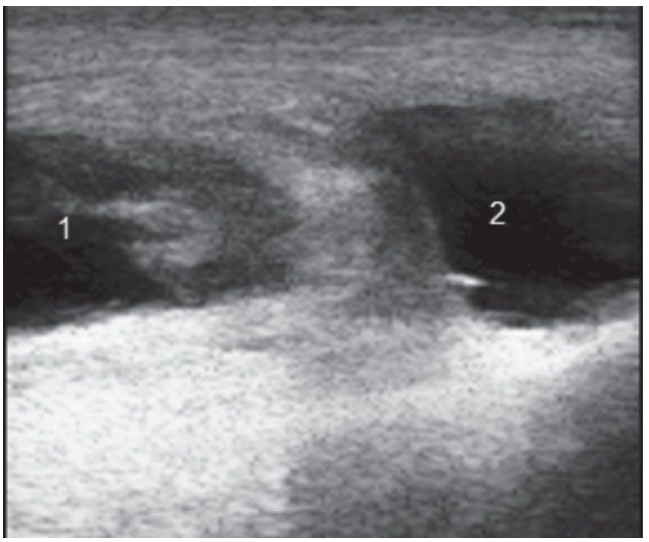


Fig. 11. Transverse sonogram shows the close proximity of (1) the medial recess of the femoropatellar joint and (2) the MFT joint recess. Cranial is to the left and caudal to the right.

Ultrasound-guided injections allow for accurate needle placement and drug deposition. They are frequently performed by two operators, one holding the probe and the other guiding the needle into the beam (ultrasound field of view). The spatial limitations of the medial aspect of the horse's stifle does not allow for convenient maneuverability of two operators; therefore this technique for a solo operator was developed to overcome that obstacle.

Injection time was not recorded in this study but subjectively appears to be less when ultrasound guidance is used. The injection time was quite rapid with ultrasound guidance because there was no delay associated with waiting for fluid to appear at the hub or incorrect manipulation of the needle into the wrong plane or direction. On one bilateral case, the time taken from application of the probe to completion of the injection was less than 30 seconds per side.

Simultaneous injection and ultrasound monitoring requires strict attention to maintaining the needle in the ultrasound beam. If the needle tip disappears from view, injection of a small amount of medication usually allows for rediscovery. Leaving the needle attached allows for more sterility because the open hub of the needle is not exposed or manipulated. The majority of the population were geldings, and two stallions were included. Sheath or scrotal contamination of the back of the hand holding the ultrasound probe can occur, but the syringe hand remains guarded, and contamination of the injection site was not a complication.

Because the needle is pre-attached, the whole process of watching the needle enter the recess and the administration of its contents can be accomplished very rapidly. With the needle trajectory being constantly visible, redirection is easily accomplished. Clients enjoy visualizing the process and appreciate the benefits of all upper-limb ultrasound-guided injections. This technique has become extremely valuable because it is simple and ensures consistent intrasynovial injection of the MFT joint recess.

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[a]Denoix J.M. Personal communication, 2010.

[b]Dormosedan, Pfizer Animal Health, Pfizer Canada Inc, Kirkland, QC H9J 2M5, Canada.

[c]Xylocaine, AstraZeneca Canada Inc, 1004 Middlegate Road, Mississauga, ON L4Y 1M4, Canada.

[d]Torbugesic, Pfizer Animal Health, Pfizer Canada Inc, Kirkland, QC H9J 2M5, Canada.

[e]Betaject, Sandoz Canada, 110, de Lauzon, Boucherville, QC J4B 1E6, Canada.

[f]Depo-Medrol Sterile Aqueous Suspension, Pfizer Animal Health, Pfizer Canada Inc, Kirkland, QC H9J 2M5, Canada.

[g]Kenalog*-10, Bristol-Myers Squibb Canada, 2344 Alfred-Nobel Boulevard, Suite 300, Montreal, QC H4S 0A4, Canada.

[h]Predef 2X, Pfizer Animal Health, Pfizer Canada Inc, Kirkland, QC H9J 2M5, Canada.

[i]MAP-5, Bioniche Animal Health Canada Inc, PO Box 1570 Belleville, ON K8N 5J2, Canada.

[j]Arthrex IRAP II System, Advanced Veterinary Products Canada, 5500 Wharf Street, Sechelt, BC V0N 3A0, Canada.

[k]Carbocaine-v 2%, Pfizer Animal Health, Pfizer Canada Inc, Kirkland, QC H9J 2M5, Canada.

[l]Omnipaque, GE Healthcare Canada Inc, 2300 Meadowvale Boulevard, Mississauga, ON L5N 5P9, Canada.

Ultrasonographic Appearance of Normal and Injured Lateral Patellar Ligaments in the Equine Stifle

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Lateral patellar ligament injuries are uncommon but significant injuries in horses. Knowledge of normal ultrasonographic variations is important to differentiate normal from injured lateral patellar ligaments. Authors' addresses: William R. Pritchard Veterinary Medical Teaching Hospital (Gottlieb) and Department of Surgical and Radiological Sciences (Whitcomb, Vaughan, Galuppo, Spriet), School of Veterinary Medicine, University of California, Davis, CA 95616; e-mail: rachel.f.gottlieb@gmail.com. †Corresponding author; *presenting author (current address: Northwest Equine Performance, 25230 S Eldorado Road, Mulino, OR 97042). © 2013 AAEP.

1. Introduction

Ultrasound is widely used in horses with lameness localized to the stifle; however, limited information is available regarding the appearance of the normal and injured lateral patellar ligament (LPL).

2. Materials and Methods

Twelve horses without stifle lameness underwent ultrasound examination of both LPLs, and ultrasonographic features were recorded. Eighteen horses with LPL injury were identified from cases presenting for stifle ultrasound from 1999 to 2011.

3. Results

The normal appearance of the LPL changes from the patella to the tibial tuberosity. The LPL is poorly defined at its origin, becomes flattened and somewhat bilobed over the lateral trochlear ridge, and then assumes a large oval-triangular shape at the joint level, where echogenicity and fiber pattern are variable as the result of invaginations extending from its deep margins. The LPL is tapered and has striations at its tibial insertion. Eighteen LPL injuries were identified in horses of multiple breeds and uses. All were acute in nature. Twelve presented with wounds. Severe lameness (Grades 4 to 5/5) was present in 11 of 18 horses. Radiography showed fractures of the tibial tuberosity ($n = 6$), patella ($n = 4$), and lateral trochlear ridge ($n = 1$). Ultrasonographic lesions were graded as severe in 78% of cases. The mid-insertional portion of the ligament was most often affected, and fractures directly involved the LPL in nine horses. Three horses were euthanized because of severe concurrent injury. Five were treated for osteomyelitis and one for synovial sepsis. Recheck ultrasound in four horses showed a stable to slightly improved appearance. Eight horses returned to their previous use, two were retired, two were lost to follow-up, and three remain in rehabilitation.

Research Abstract

NOTES

4. Discussion

Normal variations in shape, echogenicity, and fiber pattern of the LPL are important considerations to prevent false-positive diagnoses during stifle ultrasound exams. LPL injuries were often severe and associated with craniolateral stifle trauma. Prognosis varied from fair to good in horses with primary LPL injury.

Computed Tomography Arthrography for the Diagnosis of Equine Femorotibial Lameness in 137 Horses: 2007 to 2012

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Erik H.J. Bergman, DVM, Diplomate ECAR

Computed tomography arthrography provides a more complete diagnosis and a better understanding of lameness arising from the equine femorotibial joint. Authors' addresses: University of California, Davis, One Shields Avenue, Davis, CA 95616 (Puchalski); Lingehoeve Veldstraat 3, Lienden 4033 AK, The Netherlands (Bergman); e-mail: smpuchalski@ucdavis.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Stifle lameness, associated with the femorotibial joints, is an important problem in sport horses. A specific or complete diagnosis is difficult with the use of diagnostic techniques such as radiography, ultrasound, nuclear scintigraphy, and arthroscopy. Computed tomography (CT) arthrography has been proposed as a useful technique to evaluate this complex joint.

2. Materials and Methods

Clinical, ultrasound, radiography, nuclear scintigraphy ($n = 17$), and postmortem data ($n = 19$) from 137 lame horses undergoing CT arthrography of 141 stifle joints from 2006 to 2013 were gathered and evaluated.

3. Results

There were 111 of 137 Warmbloods. The mean age was 8 years, and mean lameness score was 2.1 of 5. The majority had a >10-week history. Abnormalities of the meniscotibial ligaments and entheses were identified in 102 of 141, cruciate ligaments in 78 of 141, and menisci in 57 of 141. Abnormalities of the femorotibial joint and its margins were present in 73 of 141. Lesions of any component of the meniscus apparatus were identified in 105 of 141. Twenty-five of 34 stifles with cranial and 34 of 59 stifles with caudal cruciate ligament injury had concurrent injury of the medial meniscus apparatus. Negative ultrasound and radiograph studies were associated with caudal cruciate and caudal meniscotibial ligament injuries. Gross pathology confirmed lesions in 19 stifles.

4. Discussion

CT arthrography aids in the diagnosis and understanding of femorotibial joint lameness. Lesion combinations may be important when complete diagnostic evaluation of the stifle is not possible because solitary lesions of any component of the stifle is rare.

Research Abstract

NOTES

How to Perform a Transrectal Ultrasound Examination of the Lumbosacral and Sacroiliac Joints

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1. Introduction

There is increasing interest in pathology of the lumbosacral and sacroiliac joints giving rise to stiffness and/or lameness and decreased performance in equine sports medicine.[1–3] Pain arising from these regions can be problematic alone or in conjunction with lameness arising from other sites (thoracolumbar spine, hind limbs, or forelimbs).[4] Localization of pain to this region is critically important through clinical assessment, diagnostic anesthesia, and imaging.

In general, diagnostic imaging of the axial skeleton and pelvis is difficult to perform and to interpret. Radiography is infrequently performed. To obtain good-quality diagnostic radiographs, general anesthesia, a high-output radiographic generator, and special techniques must be performed.[5,6] Variation in the size and shape of the sacroiliac joints and sacral wings and caudal sacral osteophytes are common[7,8]; special techniques for taking radiographs have allowed for identification of these structures and the inter-transverse joints.[5] These authors urge caution in the interpretation of lesions identified on radiography in the absence of other diagnostic imaging and clinical examination. Nuclear scintigraphy is an important component of work-up for sacroiliac region pain, but limitations exist. Several reports[9,10] exist detailing the anatomy and technique findings in normal horses[11,12] and findings in lame horses.[13] Patient motion, camera positioning, and muscle asymmetry can cause errors in interpretation. In normal horses, the appearance of the sacroiliac region varies with age but is generally symmetric. In horses with sacroiliac problems, it is more difficult to distinguish the tubera sacrale from the sacroiliac joint than in normal horses, and, in horses with lameness, there is more asymmetry detected.[12,13]

Techniques for percutaneous and transrectal ultrasound examination have been described, and

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their use is increasingly common.[2,14–19] Ultrasound is very useful for the evaluation of the joint margins and lumbosacral intervertebral disc.[14] Similar to the radiographic anatomy of the region, variability exists in the appearance of the tubera sacrale, dorsal sacroiliac ligaments, thoracolumbar fasciae, and the lumbosacral joint.[3,19] Artifacts of acquisition and interpretation can occur with improper technique in the ultrasound examination, and knowledge of reference images and the regional anatomy is paramount in accurate interpretation of diagnostic imaging studies of this region. All diagnostic imaging techniques should also be interpreted in light of the anamnesis and the static and dynamic clinical examination.

The purpose of this "How-to" presentation is to provide a review of the technique for transrectal ultrasound with reference images and ultrasound images of examples of pathology of the lumbosacral junction, lumbosacral intertransverse joints, and sacroiliac joints. This presentation will review imaging findings in 231 horses presenting to Lingehoeve Diergeneeskunde, Equine Referral Hospital, The Netherlands, for evaluation of stiffness or poor performance in 2012.

2. Materials and Methods

The diagnostic imaging picture archiving and communications system (PACS) of Lingehoeve Diergeneeskunde was searched for all horses undergoing transrectal ultrasound during 2012. Age and breed data were collected. All horses had a complete clinical evaluation for poor performance. After anamnesis and static and dynamic examination, a percutaneous examination of the lumbar spine and a transrectal examination of the sacroiliac and lumbosacral junction were performed.

Transrectal ultrasound examination was performed with the use of the technique described by Denoix.[20] and illustrated in Fig. 1. A 5- to 10-MHz micro-convex intra-operative ultrasound probe was used per rectum. All horses were sedated with an α-2 agonist and restrained in stocks. The rectum was cleaned, and copious lubricant was introduced into the rectum. A stand-off pad was not used. Anti-spasmodic agents were used infrequently (nine horses). The ultrasound images are oriented so that ventral is to the bottom of the image display screen, and, when appropriate, cranial is to the left.

Technique

Step 1: Lumbosacral Disc

Transrectally, the aorta and its bifurcation (normally at the level of the fifth lumbar vertebra) can be palpated. Place the ultrasound probe in a median plane, caudal to the aorta and vena cava (bifurcation normally at the level of L5). The L6 and S1 vertebrae can be recognized by the well-defined hyperechoic shadowing ventral margins, meeting at approximately a 140° to 150° angle. At this site, the lumbosacral (LS) disc, the ventral longitudinal ligament, and, occasionally, the dorsal longitudinal ligament, can be identified (Fig. 2). At this location, scan the entire disc by moving the probe left and right, maintaining a paramedian orientation of the probe.

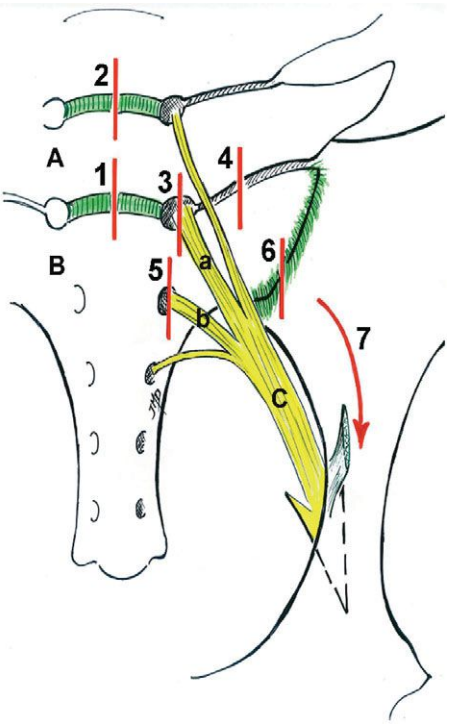


Fig. 1. Schematic drawing by Denoix of the ventral aspect of the lumbosacral region illustrates the stepwise procedure of transrectal evaluation of the lumbosacral and sacroiliac joints. Red lines indicate probe orientation. A, Sixth lumbar vertebra; B, sacrum; C, sciatic nerve; a, L6 nerve root; b, S1 nerve root; 1, lumbosacral intervertebral joint; 2, L5–6 intervertebral joint; 3, lumbosacral intervertebral foramen; 4, lumbosacral intertransverse joint; 5, S1–2 intervertebral foramen; 6, sacroiliac joint; 7, direction of imaging the psoas minor tendon.

Step 2: Intervertebral Disc and Vertebral Bodies of L5–6 and L4–5

From the LS disc, move the ultrasound probe in a cranial direction while maintaining the median plane orientation. As the probe is moved forward, maintain visualization of the ventral vertebral margin. The L5–6 disc is just dorsal and is usually caudal to the aortic bifurcation. The probe is moved cranially to identify the L4–5 disc space dorsal to the aorta.

Step 3: Lumbosacral Intervertebral Foramen

Identify the LS disc space. Move the probe in a lateral direction, maintaining a paramedian imaging plane. As the probe is moved laterally, look for a smoothly demarcated defect in the bone surface that represents the intervertebral foramen. Once

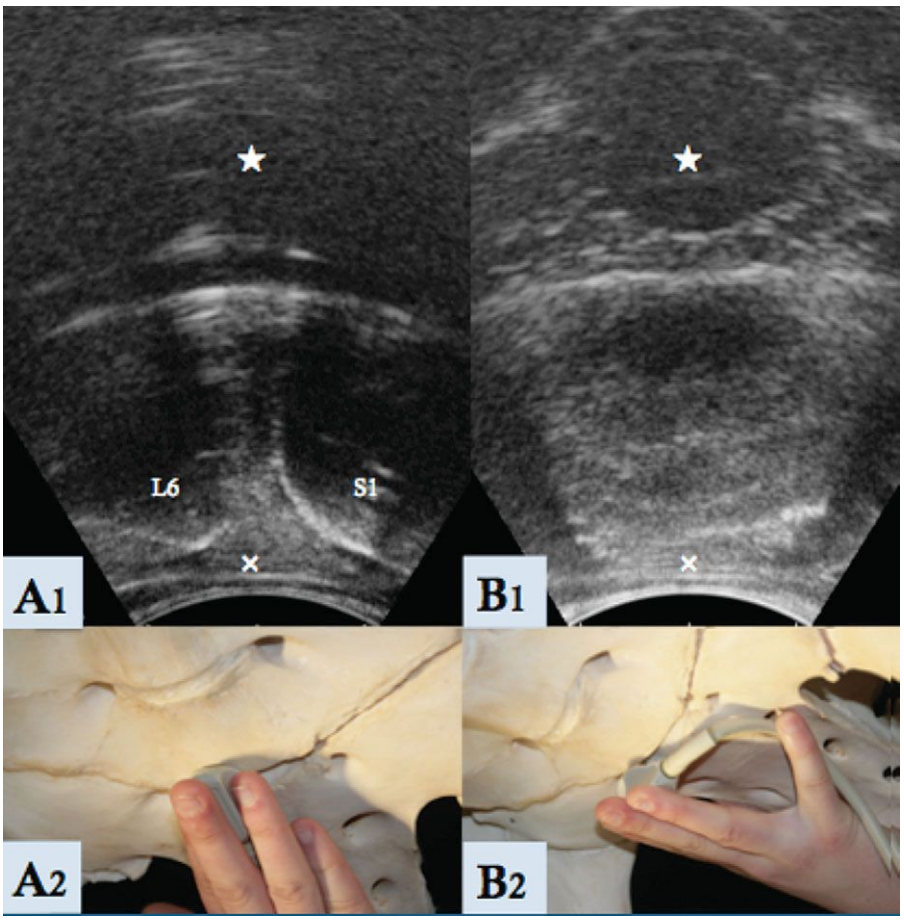


Fig. 2. These images depict the desired ultrasound image and probe position for evaluation of the lumbosacral joint, including the LS disc, in a median plane (A1 and A2) and a transverse plane (B1 and B2). This represents Step 1 of the examination. Stars mark the spinal cord; X marks the ventral longitudinal ligament.

the defect is identified, look for the L6 nerve root that can be identified as a bundle of long, parallel, hyperechoic fibers. When the foramen is first identified, the imaging plane will be oblique to the nerve, and probe manipulation is needed to orient the ultrasound probe parallel to the nerve root to char-

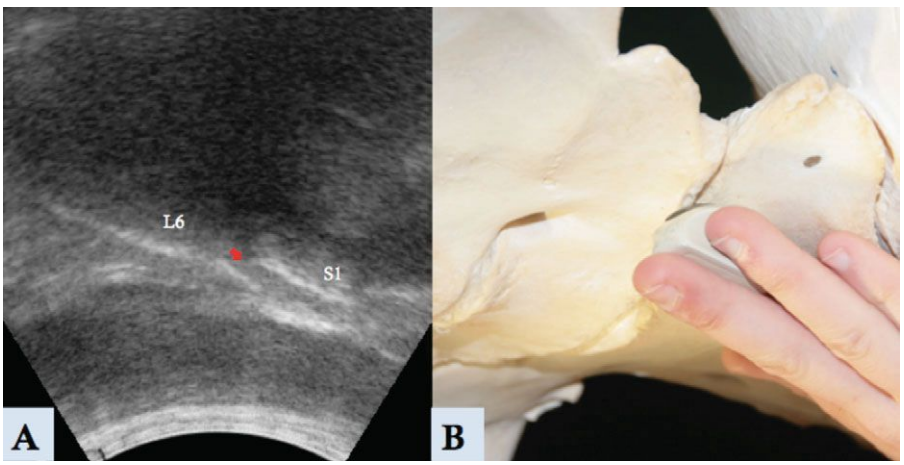


Fig. 3. This image depicts a paramedian plane of the lumbosacral intertransverse joint and the associated probe position along the ventral aspect of the sacrum. This represents Step 4 of the examination. Arrow demarcates the joint space.

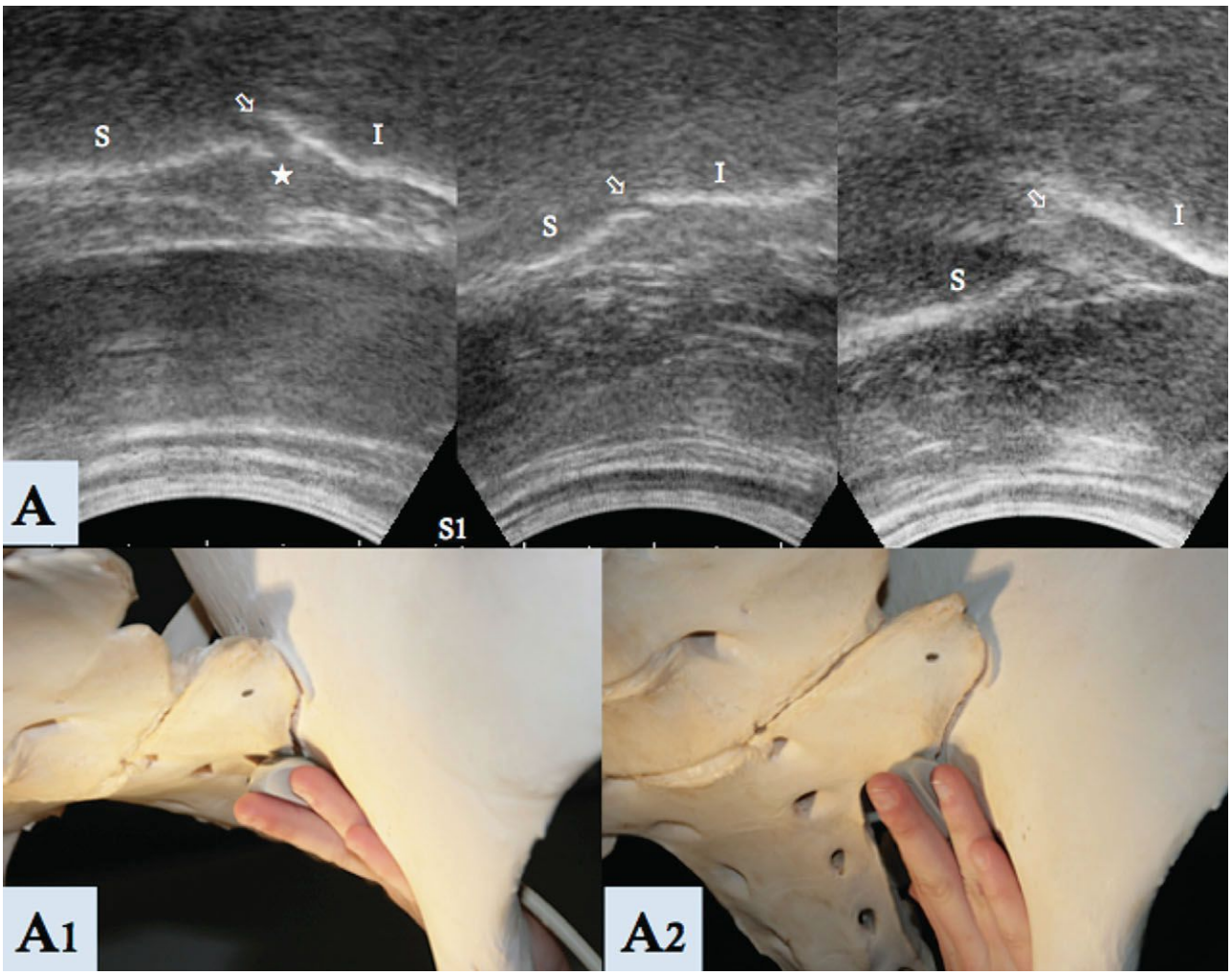


Fig. 4. These images depict longitudinal sections through three different sacroiliac joints (A) and the associated probe position (A1 and A2). Star marks the ventral sacroiliac ligament. This represents Step 6 of the examination. Arrow marks the joint space.

acterize this structure as it passes through the foramen.

Step 4: Lumbosacral Intervertebral Intertransverse Joints

From the LS intervertebral foramen, continue to move in a lateral direction to cross the LS intertransverse joint. The joint is identified as a small defect in the bone surface (Fig. 3). Center the surface defect (joint space) in the ultrasound image and move the probe in a medial to lateral direction while maintaining a paramedian probe orientation. This allows for evaluation of the joint margins.

Step 5: S1–2 Intervertebral Foramen

Return to the L6 nerve root at the LS intervertebral foramen (see Step 3). Move the entire probe in a caudal direction, maintaining the same probe orientation to identify the S1–2 intervertebral foramen and the S1 nerve root. The imaging characteristics are similar to the LS intervertebral foramen and the L6 nerve root. The operator must maintain awareness of their orientation and location relative to the described landmarks in order to insure accurate identification of these structures.

Step 6: Sacroiliac Joint

From the S1 nerve root, move the entire probe in a lateral direction, maintaining a paramedian probe orientation. The ventral surface of the sacral wing has a convex contour, allowing it to be identified. As the probe is moved along the convexity of the sacrum, a large artery, the caudal gluteal artery, will be identified. Immediately dorsal to the caudal gluteal artery, the convex surface of the sacral wing will form a junction with another hyperechoic shadowing but flat bone surface. This junction represents the sacroiliac joint. The caudal gluteal artery is used as an acoustic window for evaluation of the sacroiliac joint margins. Additionally, the ventral sacroiliac ligament can be identified, providing an additional landmark for the sacroiliac joint. The

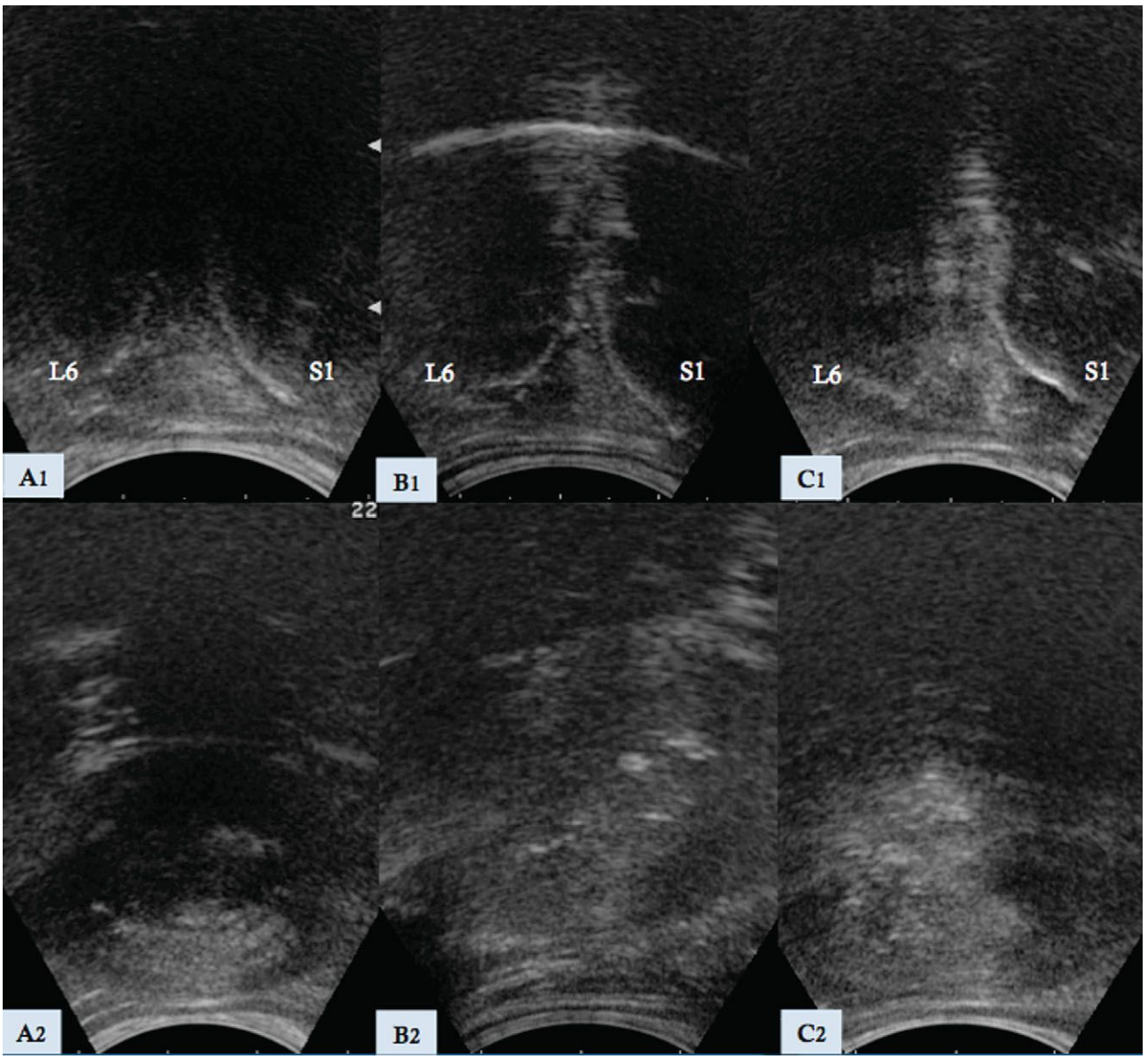


Fig. 5. These composite images depict three different grades of alterations in echogenicity and homogenicity in the lumbosacral disc. Median plane images are displayed along the top and transverse images along the bottom. A1 and A2, Mild change in echogenicity and homogenicity; B1 and B2, moderate; C1 and C2, severe.

ventral sacroiliac ligament long axis is oblique to the paramedian imaging plane and perpendicular to the joint margin; therefore, probe manipulation is required to optimize images of this ligament.

Make longitudinal images of the caudomedial margin of the sacroiliac joint including the ventral sacroiliac ligament (Fig. 4).

Optional: Step 7: Psoas Minor Tendon

Move the entire probe in a lateral and ventral direction along the ventral concavity of the ilium. The psoas minor tendon is encountered and can be identified by the typical imaging characteristics of a tendon (long, parallel hyperechoic fibers). This structure can be tracked by means of ultrasound to its attachment on the ilium.

Step 8

Repeat for the contralateral side. Using the same imaging landmarks identify the contralateral intervertebral forminae, lumbosacral intertransverse joint, sacroiliac joint, and ventral sacroiliac ligament. The psoas minor tendon and its insertion on the ilium can also be visualized at this site.

Ultrasound images were reviewed on a dedicated workstation with DICOM viewing software (eFilm, Merge Healthcare), including digital calipers. One author reviewed the images of the lumbosacral disc,

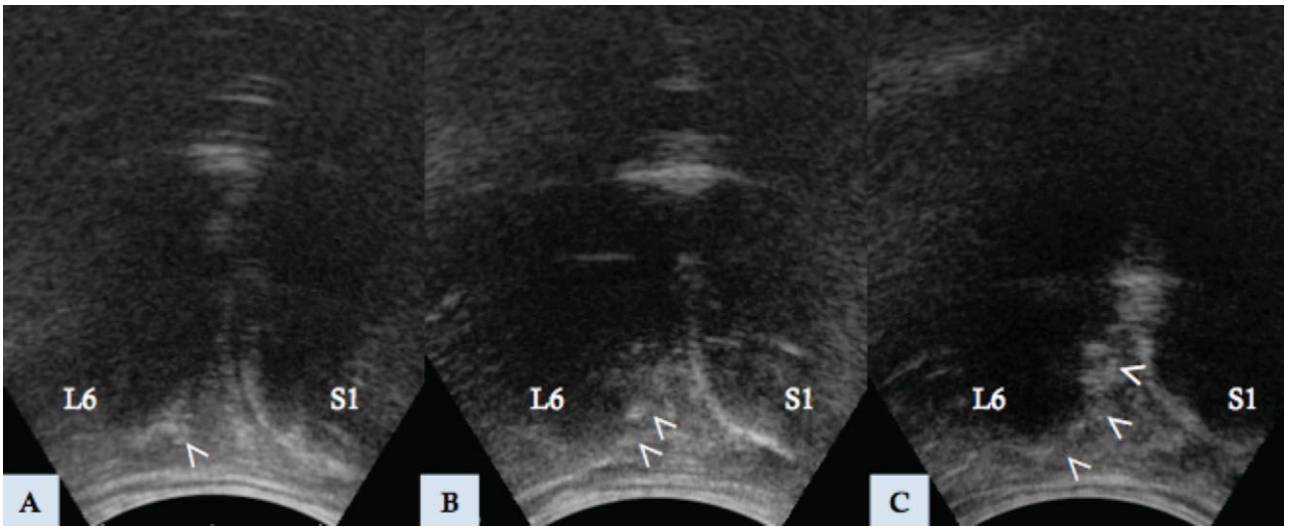


Fig. 6. These composite images depict three different grades demonstrating alterations in the lumbosacral joint vertebral margins. Images are made in a median plane. Alteration in margination is demarcated by the arrowheads. A, Focal, mild change; B, regional, moderate change; C, severe change along the majority of the visible margin of L6.

intertransverse joint, and sacroiliac joint, retrospectively (H.J.B.). To grade the following structures, a subset of reference images were identified and graded as normal, mild, moderate, or severe by two authors (H.J.B. and S.M.P.). On the basis of the subset of reference images, the grading of the remaining images was performed. The LS disc echogenicity and homogeneity were considered together, and the disc was graded (normal, mild, moderate, severe). The LS disc grading was mild if the disc has localized regions of increased echogenicity or changes from homogenous to mildly heterogeneous, moderate if there was increased echogenicity and the disc appeared heterogeneous, and severe if the disc was hyperechoic and heterogeneous (Fig. 5). The vertebral margins at the LS intervertebral space were characterized for changes in shape and margination (normal, mild, moderate, severe). They were considered mildly abnormal if there was focal irregularity or shape change, moderate if the irregular margination involved most but not all of the vertebral border, and severe if the margin was irregular and there was shape change indicating bone modeling (Fig. 6). The intervertebral space was measured from the caudoventral margin of L6 to the cranioventral margin of S1. The degree of ventral bulging of the LS disc was measured in the fashion described by Nagy et al[3] (Fig. 7). The left and right sacroiliac joints were considered separately. Bone proliferation as identified by increased size and irregular margination at the joint margin was characterized for the ilium and the sacrum (normal, mild, moderate, and severe) (Fig. 8 and Fig. 9). The ventral sacroiliac ligament was noted as normal or abnormal if it was thickened and heterogeneous). The lumbosacral intertransverse joints were also characterized as normal or abnormal if proliferation was identified. Descriptive statistics were calculated.

3. Results

Two hundred thirty-one horses were identified. The breed distribution included 199 Warmblood horses and 32 other breeds. The mean age was 8.8 years (standard deviation = 3.7). The median age

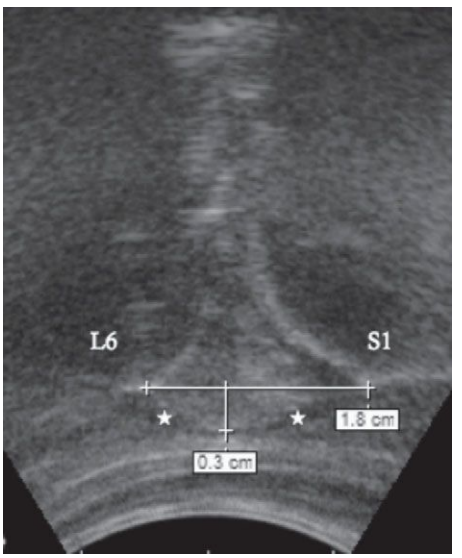


Fig. 7. This median plane image of the lumbosacral joint demonstrates the measurements. Measurement A (1.8 cm) was made from the caudoventral margin of L6 to the cranioventral margin of S1. Measurement B (0.3 cm) was made by drawing a line perpendicular to measurement A to the ventral margin of the intervertebral disc. Stars mark the ventral longitudinal ligament.

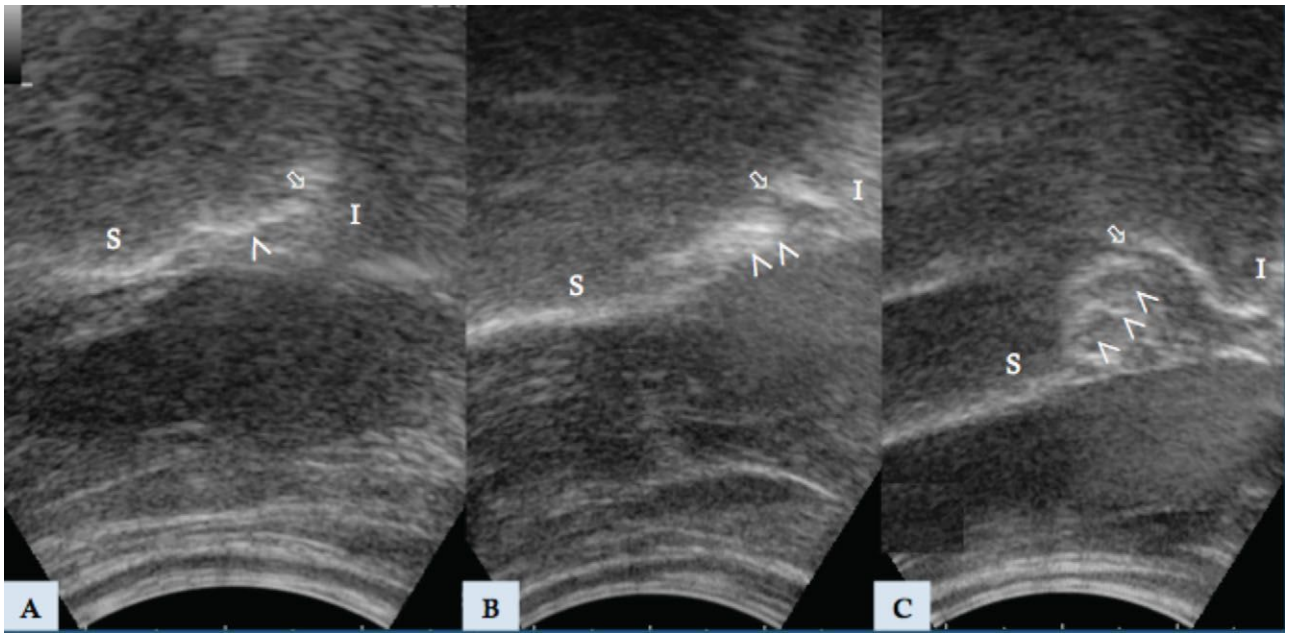


Fig. 8. These composite images depict three different grades of sacral margin proliferation. The images are made in a paramedian plane. S indicates sacrum; I, ilium; joint space is demarcated by the open arrow. Arrowheads identify bone proliferation that is mild in A, moderate in B, and severe in C on the sacrum at the level of the sacroiliac joint.

was 8, with a range of 3 to 22 years. In 227 horses, poor performance was attributed at least in part to abnormalities of the lumbosacral or sacroiliac joints. Four horses were asymptomatic, and transrectal ultrasound was performed as a part of pre-purchase examination.

Images of 228 lumbosacral disc spaces were reviewed. There was sacralization of the LS space in three horses (~1%). Three horses were excluded because diagnostic images were not saved to the PACS. Because of data presented previously by Nagy and Dyson, normal and mild were considered

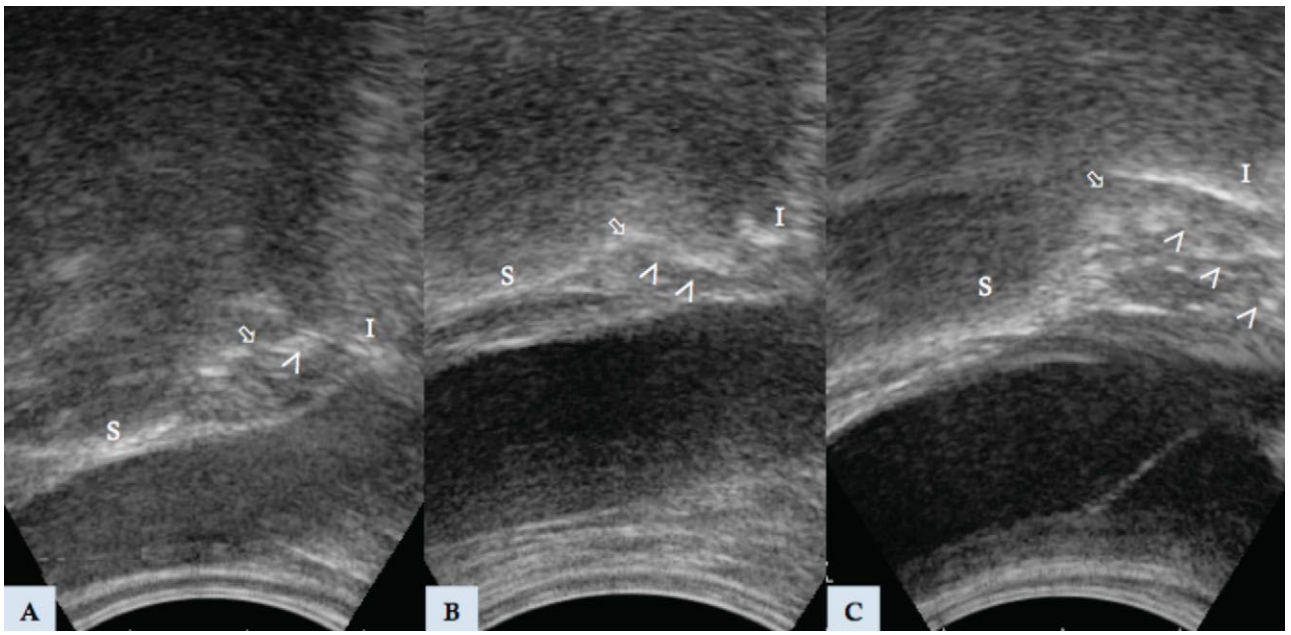


Fig. 9. These composite images depict three different grades of ilium margin proliferation. The images are made in a paramedian plane. S indicates sacrum; I, ilium; joint space is demarcated by the open arrow. Arrowheads identify bone proliferation that is mild in A, moderate in B, and severe in C on the ilium at the level of the sacroiliac joint.

Table 1. Lumbosacral Disc

Echogenicity	Number (n = 225)		Percentage
Normal	120	186	82
Mild	66		
Moderate	35		16
Severe	4		2

Table 3. Lumbosacral Disc Space Measurements

LS Disc	Measurement A	Measurement B
Mean (SD)	15.4 mm (2.5)	1.7 mm (0.95)
Median (range)	15 (8–22)	2 (0–3)

together for the lumbosacral disc space descriptive characteristics. The results for ultrasound findings of the lumbosacral disc and vertebral margins are presented in Table 1 and Table 2, respectively. Measurements of the lumbosacral disc space and LS disc bulging are presented in Table 3. Abnormalities of the lumbosacral intertransverse joints were infrequent, with 38 of 462 (8%) identified.

Sacroiliac joint abnormalities were separated into joint margin abnormalities on the ilium and on the sacrum; the results are presented in Table 4. Left and right were considered separately, and, of the total abnormal sacroiliac joints (n = 173), abnormalities of the left were identified in 71 and abnormalities of the right were identified in 102. The ventral sacroiliac ligament was characterized as abnormal (thickened and heterogeneous) in 113 of 461 (24%) joints. Similar to the intertransverse joints, when considering moderate and severe abnormalities of the sacroiliac joints, abnormalities of higher severity were more frequently identified on the right 71 of 113 than the 42 of 113.

4. Discussion

The objective of this report was to provide a review of the procedure for transrectal ultrasound evaluation technique of the lumbosacral and sacroiliac joints and to provide reference images from a population of horses with clinical signs of lumbosacral and/or sacroiliac dysfunction. For each of the anatomic sites evaluated, this population of horses had variability in the severity of the abnormal findings.

It remains difficult to determine the clinical significance of alterations in echogenicity and margination at the lumbosacral joint. In this group of horses, there was variability in the echogenicity of the LS disc and the margination of the endplates of L6 and S1, but the majority of horses were categorized as normal or mildly abnormal. A moderate proportion of horses were described as moderate to severely abnormal. Previous reports detailing the ultrasound evaluation of this region in horses without clinical signs of lumbosacral disease demonstrated that there can be variability in the echogenicity and margination at this site, but marked change was not present.[3] Although it is impossible to make a direct comparison of the two studies, it is likely that horses in this population with moderate or severe ultrasound changes have clinically significant imaging findings. Moderate to severe changes at the lumbosacral junction are identified in clinically abnormal horses. It must be underlined that subclinical manifestations with incidence on the horse behavior, locomotion, or performance do exist.

Anomalous anatomy was rare in this population. Historic literature has discussed variability in the anatomic configuration of the pelvis and lumbosacral region,[5] and sacralization of the lumbar vertebra has been reported previously.[2,5] Ultrasound evaluation is limited to the ventral aspect of the osseous structures, and the identification of anomalies is dependent on the anomaly causing overall shape change of the vertebra or loss of the intervertebral disc space, visible from the ventral surface.

Lumbosacral intertransverse joint pathology was also very uncommon in this population. Ultrasound evaluation of these joints is limited to the ventral joint margin. Postmortem evaluation of Thoroughbred racehorses showed that the entire population had degenerative changes of the intertransverse joints.[21] The low incidence of intertransverse joint pathology relative to this previous report may be caused by the differences in patient population or possibly the limitations of transrectal ultrasound as compared with ex vivo postmortem evaluation. Lumbosacral intertransverse joint pathology has been observed with a higher prevalence in racing Standardbred trotters.

The sacroiliac joint abnormalities of the majority of horses in this study showed normal or mild changes. Nevertheless, slightly greater than one

Table 2. Lumbosacral Disc Space Vertebral Margins

Vertebral Margin	Number (n = 225)		Percentage
Normal	146	207	92
Mild	61		
Moderate	12		5
Severe	6		3

Table 4. Sacroiliac Joint Margin Bone Proliferation

		Number		Percentage
Sacrum (n = 462)	Normal	187	289	63
	Mild	102		
	Moderate	120		26
	Severe	53		11
Ilium (n = 462)	Normal	136	311	67
	Mild	175		
	Moderate	119		26
	Severe	32		7

third of horses in this group with clinical signs had moderate to severe sacroiliac changes. Ultrasound evaluation of the joint margins allows for the identification of periarticular proliferative new bone (osteophytes), as expected in this or any other joint with joint disease. Variability in the appearance of the sacroiliac joint is expected in normal populations; however, many publications agree that abnormalities of increasing severity are increasingly likely to be clinically significant.[6,10,12,18,20] Scintigraphic abnormalities are often identified in horses with hind limb lameness; however, care should be taken in the interpretation of these findings because overlap exists between horses with sacroiliac disease confirmed by diagnostic anesthesia and horses with other causes of hind limb lameness.[4,12,13] It is likely that a similar corollary exists for ultrasound evaluation of the same region, whereby overlap exists between clinically significant and clinically silent pathologic change. However, sacroiliac pathologic change has been a common finding in postmortem studies, which may indicate that this truly represents a common pathology.[7,8,21] By contrast, it is also important to recall that ultrasound of the caudomedial margin of the sacroiliac joint may represent a small window relative to overall size of the joint and associated soft tissue structures.

In summary, transrectal ultrasound of the sacroiliac and lumbosacral joints is a key component the evaluation of horses with lumbosacral and sacroiliac stiffness or poor performance. The technique requires a knowledge of the anatomy, the ability to perform ultrasound, good equipment, and knowledge surrounding the identification and interpretation of abnormalities. Transrectal ultrasound should be used in conjunction with anamnesis, complete clinical examination, and percutaneous ultrasound evaluation of the lumbar facet joints. This technique can be used to guide diagnostic and treatment decisions. Although it is a potentially useful technique for prepurchase examination evaluation, caution should be exercised in image interpretation until a broader body of knowledge exists.

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Ultrasonographic Findings in 42 Horses With Cecal Lymphadenopathy

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Cecal lymphadenopathy is an uncommon but important ultrasonographic finding seen in some horses with neoplastic, infectious, or inflammatory diseases of the abdomen. Authors' address: University of California, Davis, School of Veterinary Medicine, One Shields Avenue, Davis, CA 95616; e-mail: mevaughan@ucdavis.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Mesenteric lymphadenopathy is commonly found on postmortem examination in horses with infectious, inflammatory, or neoplastic disease but has been rarely reported as an ultrasound finding. Mesenteric lymph nodes are not often visualized by ultrasound because of their location deep within the abdomen. The cecal mesentery, however, is located adjacent to the body wall in the right flank region and can be readily visualized in most horses. The purpose of this study is to describe ultrasonographic findings associated with cecal lymphadenopathy with clinical diagnosis and outcome.

2. Materials and Methods

Records were reviewed for horses presenting for abdominal ultrasound from August 1999 to July 2007. Cases were included if multiple cecal lymph nodes were visible adjacent to the lateral cecal artery and vein.

3. Results

Forty-two horses (age 3 months to 26 years) with cecal lymphadenopathy were identified. Horses presented for weight loss, fever, anorexia, lethargy, colic, and diarrhea. Additional ultrasound abnormalities included thickened intestine (90%), abdominal masses (29%), and abdominal abscessation (9.5%). Etiologies were categorized as inflammatory ($n = 16$), neoplastic ($n = 14$), or infectious ($n = 12$) on the basis of available clinicopathologic data. Twenty-one horses (50%) were euthanized or died within 6 months of diagnosis, 13 were improved, and eight were lost to follow-up.

4. Discussion

The cecal mesentery is readily imaged in the right flank and caudoventral abdomen and should be evaluated as part of a complete abdominal ultrasound examination. Cecal lymphadenopathy was often associated with additional important ultrasonographic abnormalities. Such information was helpful and often warranted further diagnostic procedures such as biopsy, aspiration, or surgery for definitive diagnosis.

Research Abstract

NOTES

From Hoof Testers to MRI: The Evolution of Distal Limb Lameness in Sporthorse Practice

Brendan Furlong, MVB, MRCVS

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1. Introduction

The art of lameness diagnosis has undergone an exciting evolution during the time that I have been involved in sporthorse medicine. The driving forces behind the progression can be grouped into two main categories. First are the scientific and technological advancements in the field of diagnostic imaging, and second, the breeds and uses of the horses that are present in the equine world today.

Back in the 1970s and prior, the majority of large animal practitioners were faced with a completely different horse demographic. Warmbloods were not on the horizon yet, and performance horses and "sports medicine" were only in the embryonic stages. A farm call would present the equine veterinarian with racehorses (Standardbreds or Thoroughbreds), broodmares (Standardbreds or Thoroughbreds), Quarter horses, or riding horses, which were usually some sort of light-boned type. Horse shows at that time did not place the same demands on the horses as the shows of today; there was no year-round show schedule. There were no Winter Equestrian Festival and summer Horse Shows in the Sun (HITS) circuits, and therefore the needs of the owners, riders, and trainers were very different. As the nature of the horse shows evolved, along with the horse pedigree, so did the need for better diagnostics in the field of musculoskeletal injury.

Any individual graduating from veterinary school as late as the 1970s would approach lameness diagnosis in a very straightforward way. Observation of the horse by means of hands, eyes, ears, and manipulations of the limb was the most sensitive means of noting gait abnormality, and this was obviously highly subject to individual experience and skills in this area. Hoof testers, basic nerve blocks, and primitive radiology equipment were also used as adjunctive means to try to better define the horse's issue, but, again, this was dependent on the clinical examination. Advanced imaging modalities have revolutionized the diagnosis and management of distal limb lameness in sporthorse medicine. The ability to have an accurate anatomic and physiologic diagnosis has allowed for horses to enjoy longer careers at the upper levels and for successful management at the twilight periods of their careers at the lower levels to teach our young riders coming up the competition ranks.

In my 30+ years of experience, the majority of lameness that I encounter is in the forelimbs—roughly 75%—and, of that, 75% is referred to the distal limb. The high numbers of injuries found in this area, especially the foot, coupled with sometimes perplexing and inaccurate peripheral nerve analgesia results in this region, have necessitated the use of advanced imaging modalities in this area.

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In our practice, the use of the standing MRI and nuclear scintigraphy has allowed for rapid diagnosis and treatment that were not achievable with traditional means.

In my early practice years, hoof testers were the most popular tool for trying to understand lameness of the foot. If a horse was negative to hoof testers but continued to be foot-sore regardless of treatment, he was turned out until soundness was achieved. Radiographs would be used to try to identify a fracture, arthritis, significant navicular bone changes, or significant degenerative changes at the attachments of the soft tissues (and to surmise injury to those structures). Today, the quality of radiographs is greatly improved but still only lends a small window into the foot. Hoof testers are used to determine the presence of an abscess or hot nail, but a mildly positive or negative response to the testers is not the end of the diagnostic process. Superior-quality ultrasound equipment is available to allow imaging through the frog to visualize the distal aspect of the navicular bone and associated soft tissue structures. However, this technique offers a limited window into the complex anatomy of the foot. In addition, it cannot be used to detect fluid in bone, and, because of the limited window, deep digital flexor tendon injuries that are not on midline or close to midline will not be detected. Ultrasound is also less sensitive than MRI for certain fiber abnormalities and cannot be used to image the distal aspect of the collateral ligaments of the coffin joint.

With increased use of MRI in sporthorse practice, I have been able to identify bone bruising, early soft tissue sprains, and significant injuries to the previously inaccessible areas of the foot (ie, distal aspect of the collateral ligaments of the coffin joints). A horse with a mild injury can have a targeted treatment, and the question of "compete or not" with acute lameness can easily be answered. Recommendation of denerving surgery to the foot for certain patients that have pain referred to the foot can be made more confidently if MRI can confirm healthy soft tissue structures and monitor them after surgery to ensure the health of the foot and therefore the safety of horse and rider as they continue to compete. Hence, many of these upper-level horses can be useful and continue their successful careers. Of course, not every case has a happy ending; it is never easy to explain to an owner that their young sporthorse prospect with intermittent lameness has a flexor surface erosion that was not evident in their pre-purchase examination radiographs. However, having a definitive diagnosis to allow for accurate prognosis in such situations can save time and money in the long run. Resources and time can be allocated toward a new horse or changing the career for the injured horse rather than investing in a case that ultimately carries a poor prognosis for long-term soundness at upper-level work (and disappointment and frustration for the owner/trainer).

Coupled with a good lameness work-up and history, nuclear scintigraphy paired with MRI has also been paramount in advancing the diagnosis of distal limb lameness even above the foot. Horses with confounding blocking patterns are often referred to nuclear scintigraphy, which can direct areas of interest to be explored via MR imaging. Consider a horse with multiple limb lameness that has multiple "hot spots" in his distal limbs: enostosis-like lesions were visualized on standing MRI. Horses that block to palmar digital or abaxial sesamoid analgesia may have increased radiopharmaceutical uptake in the fetlock, and MRI will demonstrate bony cysts, subchondral bone injury, or edema not visible on traditional radiographs. There is also a substantial population of horses that block to a "high suspensory" with negative or questionable radiographic and ultrasound findings—or horses that block lower, but distal limb imaging is negative for pathology. These horses often have increased radiopharmaceutical uptake at the level of the origin of the suspensory ligament, and placing the MR coil at the area directed by the bone scan often identifies regions of proximal suspensory injury with metacarpal/tarsal involvement. These lesions dictate a different treatment and prognosis than those with soft tissue injury only, and, with the use of the results of the advanced imaging modalities, ultrasound can be used with more confidence to monitor these injuries after definitive diagnosis. Indeed, MRI has made us better ultrasonographers.

It is an exciting time to be in the field of sports medicine, with the continual advances of diagnostic imaging and our ability to interpret these images. However, it is prudent to remember the importance of a thorough history and clinical examination and to understand the goals for the horse in question. No image can substitute for good "horse sense" as it applies to making decisions for our equine athletes. It is the author's opinion that a team approach to complicated lameness that coordinates comprehensive soundness evaluation, judicious application of appropriate imaging modalities, and clear communication between the veterinarians, farrier, owners, riders, and trainers results in the most successful outcome in these cases.

Diagnostic Approaches to Lameness of the Proximal Metacarpus and Carpal Sheath

Christopher (Kit) Miller, DVM

Diagnosis of lameness caused by lesions of the distal radius, proximal metacarpus, and carpal tendon sheath often requires an extensive clinical examination, including careful palpation, examination of the horse in hand and under saddle, and regional anesthesia. Lesions are often difficult to identify with the use of conventional imaging modalities. Author's address: Miller & Associates, 120 Nichols Road, Brewster, NY 10509; e-mail: kit.miller.dvm@miller-dvm.com. © 2013 AAEP.

1. Introduction

The distal radius, proximal metacarpus, and distal forearm, including the carpal tendon sheath and its enclosed soft-tissue structures, is a frequent and often confusing source of performance-limiting lameness in the show horse. Lameness referable to this area can be difficult to identify and to image accurately. Diagnosis of these conditions is dependent on a thorough clinical examination, including palpation and diagnostic anesthesia, and the horse must be examined on multiple surfaces both in hand and under saddle. Radiography is often equivocal, and treatment decisions frequently depend on the results and interpretation of the clinical and ultrasonographic examinations.

2. Anatomy

The carpal canal, which contains the carpal tendon sheath, is formed by the accessory carpal bone and associated ligaments laterally, the accessory ligament of the superficial digital flexor tendon (ALSDF) medially, the palmar carpal ligament dorsally, with the palmar border formed by the flexor retinaculum and palmar metacarpal fascia.[1,2] The carpal sheath contains both the superficial digital flexor tendon (SDFT) and the deep digital flexor tendon (DDFT) and is immediately adjacent to the distal palmar radius. The anatomical location of these structures is important to understand when trying to decipher the results of individual peripheral nerve and intrathecal blocks.

3. Imaging

Conventional imaging (radiology and ultrasound) of the distal radius proximal metacarpus and especially the carpal tendon sheath can be difficult to interpret. Whereas soft-tissue injuries to the SDFT or suspensory ligament in the proximal metacarpus are often easily diagnosed with simple palpation and routine ultrasound examination, soft-tissue injuries in the region of the distal radius can be difficult to identify. It is possible to obtain accurate ultrasound images of the soft-tissue structures within the carpal tendon sheath, however, and interpretation of the images requires a thorough knowledge of the gross anatomy. Ultrasound images of the DDFT and SDFT, along with its accessory ligament, within the carpal sheath, are most easily obtained with the ultrasound transducer positioned caudo-medially over the region of the

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sheath and axial to the accessory carpal bone.[3] Images of the SDF and DDF muscles along with their musculotendinous junctions in the distal antebrachium can be obtained with the probe placed at the back of the forelimb.[4] The presence of muscle fibers interspersed with the superficial and deep tendons at the musculotendinous junctions makes image interpretation of these structures particularly difficult. It is helpful to routinely image both front limbs simultaneously to help make an accurate determination.

Radiographs are often inconsistent at providing a diagnosis. Some bony lesions at the back of the radius, such as osteochondromas, are consistently identified radiographically. Osteochondromas are cartilage-capped outgrowths from the palmar radial cortex and consist of a mixture of cartilage and bone. They are typically located proximal to the physeal scar on the caudomedial radius. Osteochondromas can impinge on the DDFT within the carpal sheath, resulting in carpal sheath effusion and lameness.[5] Other bony abnormalities such as physeal remnant spikes, located at the level of the physis on the caudal radius, or enthesiophytes on cortex of the proximal palmar metacarpal bone can be very difficult to image because of their small size and superimposition of other bony structures.

4. Diagnostic Approach

Acute lameness related to the proximal metacarpus and carpal sheath appears to be quite common in show jumpers and hunters. A typical history is the horse that exercises quite vigorously, either in the ring or on the lunge line, without apparent incident, and becomes acutely and often markedly unsound. Frequently, the lameness may not become apparent for more than 24 hours. In the author's opinion, this is a classic "high suspensory" history. Frequently, the affected limb is unremarkable to physical examination. Occasionally, there is mild venous congestion in the proximal, medial metacarpus region, but generally the limbs are often surprisingly normal on palpation, given the magnitude of the lameness. It is important to rule out the lower limb as the source of lameness with systematic nerve blocks before anesthetizing the structures of the proximal metacarpus. As with palpation, images of the affected proximal metacarpus are often unimpressive. In our practice, horses with this type of history and clinical symptoms are treated symptomatically with ice, nonsteroidal anti-inflammatories, and rest, and they are rechecked in approximately 10 days. Some horses improve dramatically in that time period. At the 10-day re-check, if the horse jogs soundly with no palpable abnormality and no demonstrable lesion on ultrasound, we will typically start light exercise. This usually consists of walking the horse under tack for an additional 10 days followed by tack-walking with short, straight-line trotting intervals for another 10 days. If the horse remains normal on clinical and ultrasound evaluation at that time (30 days after injury), it is gradually returned to normal exercise. For horses that show any lameness during this progression, we return it to walking-only exercise for a 2-week period and try again. In horses with lesions of the proximal suspensory that can be identified on ultrasound examination, we begin the exercise progression once the horse is sound and the lesion has resolved.

In the show hunters and jumpers, it is important to differentiate whether lameness localized to the proximal metacarpus is caused by proximal superficial flexor tendonitis or injury to other soft-tissue structures. Proximal SDFT injury is often a progressive, degenerative condition, which occurs more frequently in the older show hunters and jumpers.[6] Affected individuals are often observed to have a subtle forelimb gait asymmetry when first ridden, which resolves quickly as the animal warms up. In the author's opinion, these horses are often uncomfortable at the canter, and, in the early stages, the lameness may be transient or indiscernible at the trot. There may be a brief period of lameness after vigorous exercise, which resolves spontaneously. Swelling of the proximal SDFT may be difficult to identify because the affected tendon may be constricted within the carpal sheath, making effusion uncommon. However, the soft-tissue swelling may be present if the injury extends distally into the proximal metacarpus. Because proximal SDF tendonitis is an important rule-out in the older show horse, diagnostic blocking patterns are an important consideration. Anesthesia of the lateral palmar nerve on the axial surface or at the base of the accessory carpal bone will frequently anesthetize both a proximal suspensory desmitis and a proximal SDF tendonitis. The lateral palmar nerve block is performed by injection of a small volume of anesthetic solution into the longitudinal groove along the medial aspect of the accessory carpal bone in its distal one-third. The needle is advanced until it contacts the carpal bone, and the anesthetic solution is deposited.[7] It is important to image all of the soft-tissue structures in the proximal metacarpus in horses whose lameness resolves with the lateral palmar nerve block, most particularly the SDFT and the origin of the suspensory ligament. Practitioners who diagnose proximal suspensory desmitis with local infiltration of the suspensory origin are at risk of missing a case of proximal SDF tendonitis if they do not also anesthetize the lateral palmar nerve, because local infiltration of the suspensory origin may not effectively anesthetize the proximal SDFT. Furthermore, if the SDF tendonitis extends proximally into the carpal canal, anesthesia of the lateral palmar nerve may result in improvement but not complete abolition of the lameness. It is particularly important, then, to consider the horse's age, history, and clinical presentation in making this diagnosis.

Lameness of the carpal tendon sheath may also be a performance-limiting condition in show horses, particularly in the jumpers and equitation horses. Acute injury to soft-tissue structures within the sheath, such as the SDFT and DDFT, frequently causes a significant and easily identified carpal tendon sheath effusion. The ALSDFT and palmar radial physis and cortex are peripheral to the sheath; therefore, carpal tendon sheath effusion in these cases is more variable. Diagnosis of lameness related to structures within the carpal sheath often requires an extensive clinical examination. Affected horses may be much more lame when examined with a rider than in hand, and the lameness may be somewhat transient. In addition, the results of diagnostic anesthesia can be somewhat confusing in that the lameness may be markedly improved though not completely abolished. Lameness caused by SDFT or DDFT injury within the carpal tendon sheath should resolve or nearly resolve after anesthesia of the carpal tendon sheath because the affected structures are contained within the sheath. Results of intrathecal anesthesia are somewhat more variable for horses whose lameness is caused by pathology of the distal radius or ALSDFT because sections of these structures are outside of the sheath.

Injury to the SDF can also occur in the distal antebrachium at the musculotendinous junction. This condition is similar to proximal SDF tendonitis because it tends to occur in older individuals. In the author's opinion, this type of lameness is often acute in onset and frequently occurs when an older show horse stumbles and becomes severely lame. The flexor muscles may be rigid and visibly or palpably thickened in affected horses. Severely affected animals may stand with the carpus in flexion and may be resistant to full weight-bearing. Because the injury involves the flexor muscle, it may be difficult to identify on initial ultrasound, though the echogenicity of the muscle tissue will change significantly over time and can be tracked with serial ultrasound examinations of the superficial flexor muscles at the back of the radius.

Carpal joint lameness is an infrequent diagnosis in our practice. In the author's opinion, many show horses compete successfully with carpal joint pathology, particularly to the dorsal aspect of the intercarpal and radiocarpal joints. Injury to the palmar aspect of these same joints occurs after direct trauma and is frequently of much greater consequence. Jumping horses will occasionally fall, with a rail caught at the back of their carpus, and traumatize or fracture their accessory carpal bone. These injuries may be performance-limiting or even performance-ending, depending on the extent of the trauma.

Diagnosis of lameness in the proximal metacarpus and carpal sheath is often difficult and time-consuming. A complete and thorough clinical examination, including careful palpation, examination of the horse in variable settings, and systematic diagnostic anesthesia, are essential. The affected structures have overlapping anatomic location and innervations, and conventional imaging is frequently suggestive rather than definitive. Being systematic and thorough, it is possible to accurately identify many of these conditions. Many can be managed with combinations of physical therapy and medication, both intrathecal and systemic. For horses with injury to tendons or ligaments within the carpal tendon sheath, tenoscopy often has a favorable outcome. Tenoscopy allows accurate visualization of the structures within the sheath, and surgical debridement of lesions is often important for complete resolution of the lameness. For horses with progressive, degenerative injury, particularly to the proximal SDF tendon and SDF muscle, the rate of injury recurrence is quite high. Many of these horses will have their show careers prolonged if they are accurately diagnosed when the condition is in its early stages.

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Distal Limb Lameness in the Sport Horse: A Clinical Approach to Diagnosis

Richard D. Mitchell, DVM, MRCVS

Diagnosis of lameness caused by lesions of the distal limb requires a thorough clinical examination, including careful palpation, manipulative tests, hoof tester evaluation, examination of the horse in hand and under saddle, and regional anesthesia. Specific causative lesions may require technical imaging modalities to fully evaluate. Author's address: Fairfield Equine Associates, PC, 32 Barnabas Road, Newtown, CT 06470; e-mail: rmitch2074@aol.com. © 2013 AAEP.

1. Introduction

Today's high-level sport horse represents a significant investment of time and money. High-level show jumpers, dressage horses, and Western performance horses often take several years to train to their upper levels, and injuries or illness can substantially affect the time to produce a winning horse. Owners and trainers are concerned that these horses receive the best possible care without excess expenses and down time. The veterinary care of such horses should take a more aggressive approach to lameness diagnostics of the horse in training, not simply one of attending to lameness after the horse is no longer able to train. Recognition of potentially serious distal limb lameness early on may prevent significant loss of training and competition time as well as extending the horse's career.

2. Anatomy and Risk Factors

For the purposes of this discussion, the author will address the anatomical region from the fetlock distally. The bony structures involve the three metacarpals, (MC2, MC3, MC4 or medial splint bone, cannon bone, and lateral splint bone, respectively), the proximal medial and lateral sesamoids, the proximal phalanx (P1, long pastern bone), the middle phalanx (P2, short pastern bone), the distal phalanx (P3, coffin bone), and the distal sesamoid (navicular bone). Each of the articulations is supported by collateral ligamentous structures and fibrous joint capsules. The distal sesamoidean ligaments (as the extension of the suspensory apparatus) and the flexor tendons attach along the palmar aspect of the pastern and foot and provide support. The dorsal digital extensor attaches along the dorsal aspect, ultimately inserting on the extensor process of the distal phalanx.

The distal limb plays a significant role in adaptation to footing and shock absorption during locomotion. The corium parietis, composed of the epidermal and dermal lamellae, function as a ligament suspending P3 within the hoof wall. There is considerable movement of P3 within the hoof wall on landing and propulsion because of this function. The digital cushion also functions in shock absorption.[1–3] Foot balance, both medial/lateral and dorsal/palmar, can have a profound effect on the excursion of joints and the stress on soft tissues of the distal limb.[1,4] Footing surfaces that are very

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hard, excessively soft, or unstable can result in aberrant motion and stress that may result in injury. The demands of high-level sports result in more concussion as well as greater extension and flexion than casual exercise. Fatigue may not be as significant a factor in injuries of the sport horse compared with racing horses, but chronic repetitive trauma is thought to be a factor. The types of injuries seen in the sport horse, racing horse, or pleasure horse may vary considerably as to intensity and potential chronicity.[5,6]

3. History and Clinical Signs

Distal limb lameness may present in a peracute fashion with extreme discomfort such with a P3 fracture or foot abscess, but more often, signs may be more subtle with the horse having a subtle lameness that changes somewhat with work. "Gee doc, he feels tight in his shoulders," is a frequent comment for more insidious forms of early front distal limb lameness. Distal hind limb issues may mimic more commonly thought-of conditions such as distal tarsitis or proximal suspensory desmopathy in that the horse is "weak" behind and may "warm out" of the lameness. Some heat or swelling may be perceived in the distal limb, but often this is not the case. The current competitive environment in North America often allows for nonsteroidal anti-inflammatory drug (NSAID) use in competing horses; horses with mild distal limb pain are able to compete comfortably. However, when Monday morning rolls around and the effects of the NSAIDS are gone, the horse is suddenly more uncomfortable than before the event. This presents a good opportunity to investigate the case.

Horses may present with a unilateral lameness of varying degrees (0–5/5) or appear bilaterally lame, especially if circled or ridden, which will be discussed later. Some lameness may be very subtle and will require special efforts to make it more apparent for an objective diagnosis. Many horses vary in their degree of lameness, improving profoundly with a few days of rest. Such mild lameness issues may be best re-examined after a day or two of exercise.

4. Physical Examination

This author is of the opinion that it is essential to have periodic veterinary inspections of the training sport horse to catch subtle lameness and performance problems before they become serious clinical issues. Although many trainers and owners are very adept at catching subtle soundness and health related issues, many things may be overlooked by the individual who sees the horse every day. Periodic veterinary inspection should include a general health check and a thorough lameness evaluation. The frequency of these exams will be somewhat dependent on the age and activity of the horse, but two to four times yearly should be a minimum. Horses with successfully managed orthopedic disease may need to be checked more often.

A typical lameness examination should first involve a thorough visual exam of the horse, taking notice of body symmetry and muscle structure. Observing the horse in its stall may give the examiner some clues regarding the horse's level of comfort. Watching the horse step out of its stall may give big clues about chronic lameness issues. After the visual inspection, a palpation examination should be performed. The author uses a modification of an "acupuncture" examination that allows for complete palpation of the horse while eliciting responses from potentially painful areas. Careful inspection of the feet for balance and symmetry should be performed. Flexion manipulations can then be performed in a "passive" sense (not asking the horse to walk or jog away) while noting any resistance or painful responses. It may be appropriate to use hoof testers at this point before starting any exercise. Next, the horse can be moved in hand at a walk and trot, taking note of any obvious lameness or unusual foot flight or limb motion. This is best performed on hard footing if available. It is useful to see the horse walk in circles as well as on a straight line. Likewise, jogging the horse in circles as well as in a straight line may provide much more insight related to the horse's level of comfort. If the horse is too fractious to lunge in circles, jogging in hand on circles may provide an acceptable alternative. The author frequently gives a small dose of sedative (detomidine hydrochloride, 1.5 mg total dose)[a] to evaluate lameness if the horse is not well-behaved. Watching the horse move in straight lines and circles on both hard and soft footing can be immensely valuable in detecting lameness.

The next step is to perform flexion tests of all limbs with the horse then walking or trotting away. These may give clues to soreness that is not otherwise evident. It may be worth pursuing some positive flexion responses or readily apparent lameness at this point while simply recording some others for future reference. Keep in mind that a positive distal limb flexion may indicate a problem in any number of places, from the distal interphalangeal joint (DIPJ) to even a suspensory ligament issue. It is a tool for regionalization and augmentation for observation purposes.

"Wedge" tests are sometimes useful to aid in localization of the source of lameness. A reverse wedge that stretches the deep digital flexor tendon (DDFT) may increase a lameness related to that structure; a wedge placed laterally under the foot may stretch the medial collateral ligament of the DIPJ and increase lameness if that structure is injured. The latter effect is because of compression on the same side as the wedge and rotation of the DIPJ on the contralateral side that stretches the collateral ligament on that side.[1]

Care should be taken to not overexert the very lame horse (>3/5) until a good sense of the nature of

the problem is recognized. A detailed record of observations should be maintained for each examination. After flexion tests, it is advisable to observe the horse work on a lunge line and under saddle. Some horses are difficult to lunge and may pose a hazard for injury to horse and handler. These horses should be lightly sedated or simply watched under tack. Many lameness conditions are not otherwise apparent until a rider is aboard and/or the horse is asked to do more work.

The riding examination may give the veterinarian a great deal of information not otherwise apparent during the in-hand exam. Rider weight may change the balance of the horse in such a way as to augment lameness. Weight on the back may be the source of discomfort and may be demonstrated by a change in the shape of the back, height of head carriage, shortening of stride, or disobedience. The horse should be asked to perform the various gaits while under saddle and carefully observed for changes in the level of lameness with each gait, transitions from one gait to another, and at directional changes. Distal limb lamenesses of the lower limb are often exacerbated by circling, especially on hard footing.[5] Directional changes may enhance this effect and most significantly at the initiation of the change in direction.

It is very important to consider more than just the "limbs" when searching for the answer to lameness and performance problems. Axial skeletal issues may arise that can produce serious limitation in function and may cause diminished function that mimics distal limb lameness. Sacroiliac strain and lower lumbar pain are frequently encountered in jumping and dressage horses. Back pain may sometimes manifest as lameness in a hind limb. Neck pain related to muscle or ligament strain may make the horse unwilling to work in a specifically desired manner. Osteoarthritis of the cervical synovial facets may produce front limb lameness and should be taken into account during the physical examination.

At this stage of the examination, the examiner should have a good sense of the severity of any lameness and may proceed with further diagnostics such as nerve blocks. It should be noted that if the degree of lameness suggests that further damage may be incurred by exercise or exercise with regions desensitized, the author will go straight to radiography or ultrasound in select cases.[6] Diagnostic nerve blocks are the author's next diagnostic procedure of choice after the physical examination as long as they are deemed safe, relative to the severity of the lameness. Severely lame horses may be at risk because of limb instability and could suffer more damage if blocked and exercised in some fashion. Many lameness conditions look alike and flex alike; however, they are not of the same origin. If the lameness is of sufficient grade to produce a clear contrast and has not been reduced by exercise, for example, warming up, the examiner would be wise to pursue the lameness with diagnostic nerve blocks. Once localized, further physical examination, radiographs, and diagnostic ultrasound may further elucidate the nature of the problem. In some cases, physical examination and diagnostic imaging may be sufficient to clearly define the diagnosis, and, in such cases, nerve blocks may not be required, allowing the veterinarian to proceed with appropriate therapy.[7] More complicated cases may require more advance imaging techniques such as nuclear scintigraphy, magnetic resonance imaging (MRI), or computed tomography (CT).

5. Diagnostic Anesthesia

Distal limb regional anesthetic techniques are straightforward to perform and applicable for use, except for the most fractious of horses. Mild sedation of the more difficult horse may aide the process and still permits a working examination, as previously mentioned. The distal digital nerve block (posterior digital block) should be the initial block performed just proximal to the level of the collateral cartilage.[8] The use of a smaller amount of anesthetic agent (mepivicaine 2%)[b] such as 1.5 to 2 mL over each nerve, in the author's experience, will provide a reliable anesthetic effect at 5 to 8 minutes, with less likelihood of affecting more proximal structures through proximal diffusion. Checking the foot with hoof testers before and after the block will provide information about its efficacy. One may wish to block only one nerve at a time if hoof testers indicate significant asymmetrical pain; however, failure of this block does not eliminate the possibility of an asymmetrical cause, in this author's experience. The sole region, podotrochlear apparatus, and DIPJ may be anesthetized with this block most commonly. Occasionally some improvement of proximal interphalangeal joint (PIPJ)-related lameness may be produced by this block, probably because of the close proximity of the large palmar pouch of that joint.

Lack of response to the distal digital nerve block does not rule out the foot as a source of lameness because the distal limb may be incompletely blocked. In the author's experience, common conditions such as a severe hoof abscess sometimes require more proximal anesthesia. There are multiple options for anesthetic techniques at this point. One may elect to simply move more proximally to the proximal digital nerve block or basi-sesamoid block just distal to the fetlock joint. This block may effectively anesthetize the more dorsal structures of the digit that are sometimes missed with the distal digital nerve block; however, more proximal regions such as the fetlock joint and associated anatomy may be anesthetized.[9] Intra-articular anesthesia of the DIPJ with 5 mL of anesthetic agent may render the horse sound when the distal digital block did not have this effect. This is certainly more confining to the foot but not specific to the DIPJ because the distal aspect of the deep digital

flexor, navicular impar ligament, and navicular bone may be anesthetized as well. It has been reported that horses demonstrating MRI evidence of DDFT lesions do not reliably improve with palmar digital anesthesia, but 68% are improved with anesthesia of the distal interphalangeal joint.[10]

It has been proposed that lameness related to the distal DDFT may be diagnosed with an intrathecal block of the distal digital tendon sheath (3–5 mL), and certainly this may be true, but it must be kept in mind that this anesthetic technique can affect other structures on the palmar pastern such as the straight distal sesamoidean ligament and middle scutum.[11]

Anesthesia of the navicular bursa may be performed accurately with the use of radiographic or ultrasound guidance in a compliant patient. Three milliliters of anesthetic agent is sufficient to establish a block. The author prefers to perform this block with the limb held up by an assistant using a previously described navicular position technique[12] and confirmed by radiographic imaging. The author believes that this block is certainly specific to the foot and is a good indicator of problems in the podotrochlear apparatus.

The PIPJ may be selectively anesthetized also with either dorsal or palmar approaches, the latter being the author's preference. The large palmar pouch of the PIPJ reliably yields synovial fluid, and 5 mL of anesthetic will effectively block the joint in 5 to 8 minutes, in the author's opinion. The approach is oblique and appears to avoid the DDFT and digital sheath.

Ring blocks of the proximal pastern are probably the most inaccurate, and, in the author's experience, are subject to the most "drift" proximally and are rarely used by this author for diagnostics.

Proximal to the fetlock, the low volar or low four-point block is useful to regionalize the fetlock joint as the source of the lameness; however, this block may begin to drift proximally in as little as 10 minutes, in the author's experience. The author believes that intra-articular fetlock anesthesia, with the use of 5 mL of solution, is effective in assessing pain from the synovium and fibrous capsule and, in some cases, the immediate subchondral bone. Deeper subchondral lesions may not respond to this block.

6. Differential Diagnosis

Having established that the site of the lameness is the distal limb, the next step is to consider the differential diagnosis for the various issues in this region and the imaging techniques that can further establish a diagnosis. The most important imaging technique is simply thorough observation. Removal of the shoe certainly is the first of these techniques and may reveal a great deal. Foot symmetry, obvious deformity, and readily apparent pathology may establish a diagnosis without technical imaging. It is probably not indicated to immediately move to radiographic imaging of an acute abscess or heel bruise, but, if these prove refractory to treatment, that may not be the case. A profoundly effusive fetlock that is palpably uncomfortable and painful to flexion should probably be imaged with radiography and ultrasound regardless of other steps taken.

Careful assessment of flexion responses, nerve block results, and more thorough physical examination may reveal information concerning conditions of the hoof capsule and sole, along with some issues more proximally in the digit, but it is most likely that some form of technical imaging will need to be used to identify the origin of the lameness. The more common issues to consider are below:

- Solar bruising, abscesses, keratoma
- Sheared heel syndrome
- P3 trauma/inflammation or rotation (positive, negative, and possible laminar tearing)
- Desmopathy of the DIPJ collateral ligaments
- Synovitis of the DIPJ
- Navicular bone/bursal inflammation/navicular impar desmopathy
- DDFT tenosynovitis and tendinopathy (including dorsal fibrillation, core lesions, and enthesiopathy in the more distal aspect)
- Collateral sesamoidean and chondrocoronal desmopathy
- Desmopathy of the distal digital annular ligament
- Inflammation of the pastern joint (PIPJ) and associated collateral and palmar ligaments
- Trauma to the middle scutum
- Desmopathy of the various distal sesamoidean ligaments
- Fetlock synovitis and osteoarthritis
- Proximal sesamoiditis and suspensory branch insertional enthesiopathies
- Subchondral bone trauma/inflammation at any of the articulations
- Distal metacarpal fractures

Some of these conditions may be a straightforward visual/physical diagnosis, but many—such as osteoarthritis, soft tissue injury, and subchondral bone inflammation—will require some degree of technical imaging (such as MRI) to elucidate. Most often the above-mentioned conditions occur in various combinations related to function, foot balance, and sport discipline.

7. Imaging

Radiography remains the gold standard for imaging of the equine distal limb. Fractures, osteoarthritis, enthesiopathies, P3 rotation and malposition, advanced subchondral bone changes, and dystrophic mineralization are frequently detected in this manner. Many soft-tissue abnormalities may escape detection with radiography, but some subtle signs of

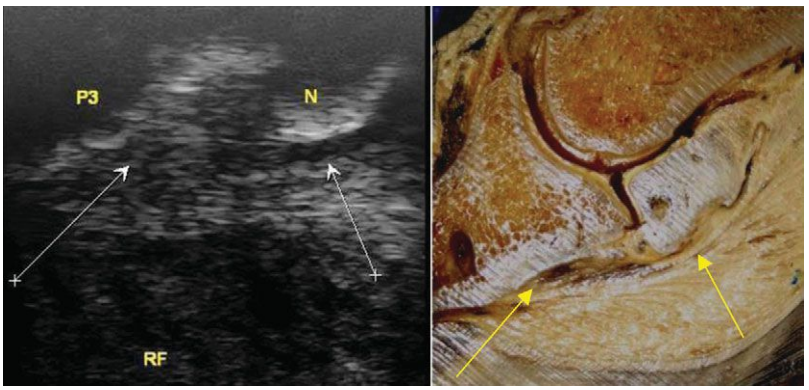


Fig. 1. Sonographic transcunean view and comparative anatomy of the DDFT coursing over the navicular flexor surface (right arrows in each image) and the insertion on P3 (left arrows in each).

problems may be evident because modern digital radiograph has improved overall image quality.

Ultrasound can be the next step in evaluating problems of the distal limb. Good knowledge of the anatomy is required to properly evaluate the structures, but most of the tendinous and ligamentous structures may be readily imaged. Collateral ligament desmopathies and flexor tendon injuries are easily evaluated with a 7.5–12-MHz linear transducer. The palmar fetlock and pastern, dorsal fetlocks and pastern, and dorsal coronet may be examined. The palmar aspect of the foot, with some trimming and soaking preparation of the frog, may be examined by means of a transcunean approach to visualize the distal DDFT, the navicular bone, and the insertion of the DDFT on P3 (Fig. 1).[13] The proximal aspect of the navicular bone, the bursa, and collateral sesamoidean ligaments may be visualized with an 8–10-MHz, microconvex probe placed between the bulbs of the heel. It should be mentioned that the distal insertion of the DIPJ collateral ligaments are beyond the view of the ultrasound probe because the hoof wall blocks the view.

Nuclear scintigraphy is very useful in further regionalizing the source of the lameness. It can be very helpful in determining what other diagnostics or imaging modalities might be best used. Areas of increased radioisotope uptake (IRU) are characteristic to some structures in working horses and knowledge of this is required,[14] but this imaging modality can be extremely helpful in evaluating the significance of the findings of other modalities such as radiography and ultrasound. A suspicious-looking navicular bone on radiography that demonstrates dramatic IRU on scintigraphy is thought to have more clinical significance in arriving at a diagnosis of navicular bone inflammation (Fig. 2).

In recent years, MRI has become more available to many equine practitioners for lameness evaluation. Although high-field magnets may produce a more detailed image, they currently require general anesthesia for imaging. The standing units are low-field magnets, and image resolution is not as high, but they are nonetheless capable of providing diagnostic images. A specific region should be identified before the MRI because the ability to image

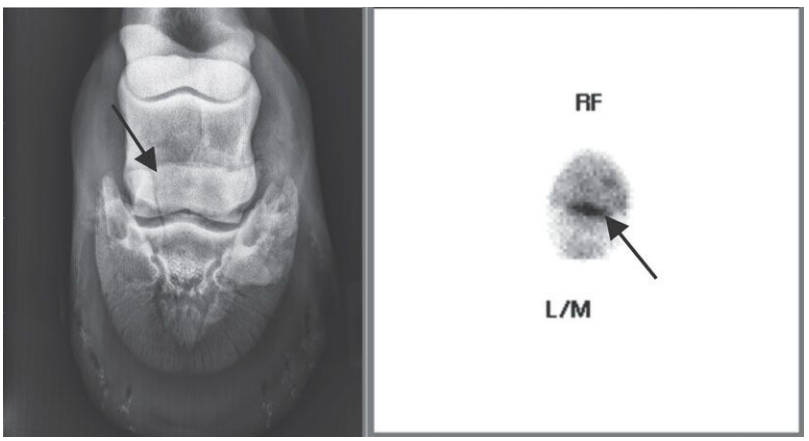


Fig. 2. Radiograph of navicular bone and corresponding solar view scintigraphy (right image) with IRU (arrows indicate the navicular bone in each image).

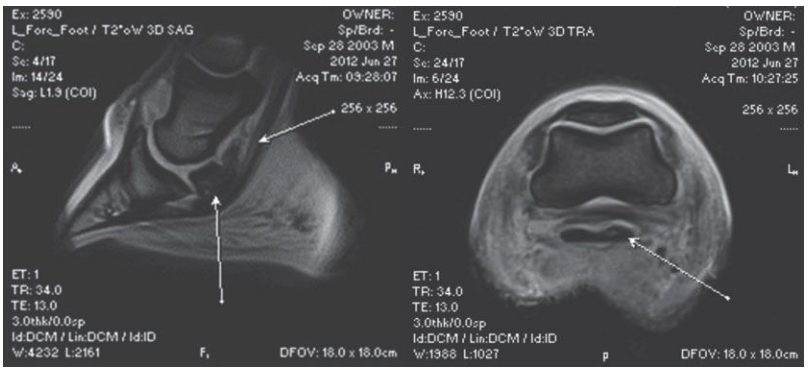


Fig. 3. Magnetic resonance image on left demonstrates mixed hyperintense and hypointense signals (lower arrow) associated with bone edema and sclerosis in the T2*oW series; upper arrow on left image demonstrates a core lesion in the DDFT; single arrow on the right MRI demonstrates a core lesion in the DDFT corresponding to that seen in the left image.

multiple regions is limited. MRI provides increased detail of the anatomy and pathology of the foot over other modalities currently used clinically.[15] Soft tissue issues not otherwise apparent may be readily visible with MRI. Changes in specific sequence signals related to processes such as bone inflammation are often evident with MRI before any appreciable radiographic changes are evident. The advantage in this is that detection of bone trauma and inflammation at an earlier stage may allow for more specific treatment modalities and exercise management (Fig. 3).[16] The use of computed tomography (CT), and especially contrast CT, has a similar application as does MRI in that it may demonstrate soft-tissue injuries as well a bone abnormalities.

The previously discussed imaging modalities are those most often used by this author. Choosing appropriately from this combination of modalities should give the practitioner specific information that increases the significance of physical exam findings and local anesthetic blocks, leading to a reasonable diagnosis.

8. Conclusions

Today's sport horse represents a significant economic and emotional investment for the horse owner. Precise and accurate diagnosis that is done in an efficient and thorough fashion is essential and will be appreciated by the client. The modern private practitioner has many basic skills that should be included in the basic work-up of a lameness case before embarking on advanced-level imaging. Advanced imaging modalities can ultimately lead to a very accurate and time-efficient diagnosis.

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[a]Dormosedan, Pfizer Animal Health, Exton, PA 19341.
[b]Carbocaine-V, Pharmacia and Upjohn Company, Division of Pfizer Inc, New York, NY 10017.

Proximal Metatarsal Lameness in Sports Horses: A Clinical Approach to Diagnosis

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Proximal metatarsal pain is most commonly caused by desmopathy or enthesopathy of the proximal part of the suspensory ligament. Diagnosis of the causes of proximal metatarsal pain has improved markedly with the use of more specific regional analgesic techniques, objective techniques of lameness quantification, and cross-sectional imaging methods. However, even diagnostic anesthesia of the deep branch of the lateral plantar nerve cannot be considered totally specific for the diagnosis of proximal suspensory desmitis. Treatment methods are often unsuccessful, and neurectomy of the deep branch of the lateral plantar nerve may be necessary to regain soundness. Author's address: Clinéquine, Campus Vétérinaire de l'Université de Lyon, VetAgro Sup, 69280 Marcy L'Etoile, France; e-mail: michael.schramme@vetagro-sup.fr. © 2013 AAEP.

1. Introduction

Proximal plantar metatarsal pain can be defined as lameness that improves after anesthesia of the deep branch of the lateral plantar nerve or other forms of subtarsal anesthesia. Proximal plantar metatarsal pain appears to have become the most commonly diagnosed cause of hind limb lameness in sports horses, even more common than distal tarsal joint pain. Recent high-field magnetic resonance (MR) imaging studies of horses with proximal plantar metatarsal pain have indicated that proximal suspensory desmopathy (PSD) and/or enthesopathy was identified as the cause of lameness in 55% to 80% of these horses, whereas in the remaining cases the diagnosis was unrelated to the suspensory ligament.[1,2]

2. Anatomy and Risk Factors

The proximal portion of the hind suspensory ligament is contained within a restricted canal composed of the plantar surface of the third metatarsal bone, the large head of the fourth metatarsal bone, the smaller head of the second metatarsal bone, and the overlying deep lamina of the flexor retinaculum that connects the plantar borders of the second and fourth metatarsal bones. This particular anatomical arrangement has led to suggestions that proximal metatarsal pain may arise from a compartment-like syndrome with compression of the lateral and medial plantar metatarsal nerves rather than from the suspensory ligament itself.[3]

Although the prevalence of PSD in sports horses appears to have increased in recent years, the reasons for this remain poorly understood. Improved recognition not only by veterinarians but also by equestrian professionals certainly has played a role. Modern training demands, regimes, and surfaces may also contribute. Predisposing factors have been identified as dressage training not only at advanced levels but also at lower, non-elite levels (in particular piaffe, passage, and pirouettes),[4] straight hock and hyperextended fetlock conformation,[5] and

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long toe-low heel conformation, especially in combination with a negative palmar angle of the distal phalanx.[5]

3. History and Clinical Signs

A diagnosis of proximal metatarsal pain is based on the history, evaluation of risk factors and conformation, clinical signs, response to diagnostic anesthesia, and imaging.

PSD is frequently though not always associated with a straight hock and hyperextension of the metacarpophalangeal/metatarsophalangeal (MCP/MTP) joint. It is not always clear if this appearance is a primary risk factor or a secondary postural change caused by loss of strength of the stay apparatus in affected horses. The reason for presentation of the horse to a veterinarian may vary from subtle loss of performance to marked, unilateral lameness. Loss of performance during ridden work may present as bilateral stiffness, loss of hind limb impulsion, difficulties in transitions, resistance to lateral exercises, flying changes or canter pirouettes, evasive behavior, or reduced power when jumping. Horses tend to warm out of early injuries fairly quickly. Lameness may be absent or present as mild with an insidious progression or severe with an acute onset. Bilateral lameness is common and can be mild to moderate in degree.

Clinical signs of acute inflammation (swelling, heat, and pain over the affected ligament) may be evident in acute cases but are more frequently absent. The presence of swelling is best identified by palpation in the standing limb. Swelling will result in some loss of the concave profile of the skin on the plantarolateral aspect of the limb, between the plantar border of the lateral splint bone and the lateral margin of the superficial digital flexor tendon in the proximal metatarsal region (Fig. 1). This is a highly specific clinical finding even when mild, and it should always be compared with the same area in the contralateral limb. Pain on palpation is easier to detect in the raised limb, even though the proximal part of the suspensory ligament is difficult to palpate in the hind limb because it is largely covered by the heads of the lateral and medial splint bones and lies deep to the digital flexor tendons. The best technique in this case is to exert pressure on the proximal part of the suspensory ligament by compressing the flexor tendons dorsally. Horses with PSD may also resent pressure between the head of the lateral or medial splint bone and the lateral or medial margin of the superficial digital flexor tendon. Some clinicians believe the Churchill test to be particularly useful in identifying pain in the proximal part of the hind suspensory ligament.

Evaluation of lameness should be performed with the horse trotting in straight lines and lunging in circles both on a hard surface and a soft surface. As with many horses with hind limb lameness, there is often a reduced arc of foot flight, reduced extension of the fetlock joint, and shortening of the cranial phase of the stride. When examining horses with mild lameness caused by proximal plantar metatarsal pain, the use of a wireless, inertial, sensor-based system of lameness quantification[a] can be of tremendous help to quantify the degree of lameness objectively and to characterize the nature of the asymmetry in vertical displacement of the pelvis between strides. Horses with PSD often show push-off rather than impact lameness. The effect of circling and surface variation is less predictable than in horses with forelimb PSD. There is frequently but not always a moderately positive response to both distal and proximal flexion tests. In some horses with PSD, lameness is only obvious under saddle, especially when the rider is sitting on the diagonal of the lame limb.



Fig. 1. Plantarolateral view of both hind limbs of a horse with mild left hind lameness associated with chronic proximal suspensory desmitis. There is loss of the normal concave profile of the skin on the plantarolateral aspect of the left limb, between the plantar border of the lateral splint bone and the lateral margin of the superficial digital flexor tendon (arrows). The normal concave profile in this area is shown in the right hind limb for reference (arrowheads).

4. Diagnostic Anesthesia

There are many different ways of removing sensation from the proximal plantar metatarsal region, in particular from the origin and body of the suspensory ligament. Techniques include the high six-point nerve block, the tibial nerve block, direct infiltration of local anesthetic solution around the origin of the suspensory ligament, and anesthesia of the deep branch of the lateral plantar nerve. This latter technique has a better chance of improving the specificity of diagnostic anesthesia compared with the other techniques.[6] The horse is restrained with a twitch, the affected hind limb is lifted, and the fetlock is supported on the clinician's knee with

the hock flexed at 90° and the third metatarsal bone positioned vertically. The superficial digital flexor tendon (SDFT) is deflected medially, and a 25-mm, 23-gauge needle inserted perpendicular to the skin surface, 15 mm distal to the head of the fourth metatarsal bone, on the plantarolateral surface of the metatarsal region. The needle is advanced in a slightly dorsomedial direction between the fourth metatarsal bone and the lateral border of the SDFT up to the hub, and 3 to 4 mL of mepivacaine is injected without resistance. Occasionally, blood is seen to flow freely from the needle, indicating puncture of the venous portion of the (proximal) deep plantar arch, in which case the needle should be re-directed slightly more dorsolaterally to avoid intravascular injection. The lateral placement of the needle in this technique reduces the risk of inadvertent penetration of the tarsometatarsal joint and the tarsal sheath when compared with other methods of subtarsal anesthesia. However, in up to 20% of horses in which 2.5 mL of mepivacaine was injected at this site, the lateral plantar nerve also appeared to have been desensitized.[7] Therefore, it is always advisable to assess the effect of anesthesia of the distal limb with a low six-point nerve block first, before performing diagnostic anesthesia of the deep branch of the lateral plantar nerve.

Lameness is assessed 10 to 15 minutes after injection. Critical evaluation of the degree of improvement can be performed accurately and objectively with a wireless, inertial, sensor-based system of lameness quantification[a]. This is essential when considering treatment by neurectomy. It is also important when comparing the degree of improvement with that seen after intra-articular anesthesia of the distal tarsal joints. Subtarsal anesthesia may improve tarsometatarsal joint pain and vice versa, but most improvement in suspensory pain is usually seen after anesthesia of the deep branch of the lateral plantar nerve. It has been suggested that pain is less successfully alleviated by anesthesia of the deep branch if enthesopathy of the proximal plantar portion of the metatarsal cortex is present. In these cases, direct deeper infiltration of 2 to 4 mL of mepivacaine at the bone surface may be more effective in abolishing lameness. Anesthesia of the tibial nerve alone eliminates suspensory ligament pain without completely removing sensation from the distal tarsal joints.

5. Differential Diagnosis

Recent high-field MR imaging studies of horses with proximal plantar metatarsal pain have indicated that PSD and/or enthesopathy was identified as the cause of lameness in the majority of them (55–80%), whereas in 20% to 25% of horses, a pathologic process unrelated to the suspensory ligament was documented, and in 10% to 20% of cases, no reason for the lameness could be found in the proximal metatarsal or distal tarsal regions. Lesions that were considered responsible for lameness but were unrelated to the suspensory ligament included osteoarthritis of the distal tarsal joints, osseous cyst-like lesions of the tarsal bones, tarsal bone edema, enthesopathy of the intertarsal ligaments, osseous injury of the third or fourth metatarsal bones, tendinopathy of the deep or superficial digital flexor tendon, and desmopathy of the plantar ligament.[1,2] Other injuries that should be considered in the proximal plantar metatarsal region are stress fractures of the plantar metatarsal cortex and avulsion fractures of the origin of the suspensory ligament. Neuropathy of the deep branch of the lateral plantar nerve may be the cause of pain in horses without imaging abnormalities.

6. Imaging

An accurate imaging diagnosis of proximal metatarsal pain is of great importance because recommended options for management are costly and time-demanding. This diagnosis can be based on radiographic, scintigraphic, sonographic, and MR imaging findings. Radiographic and scintigraphic findings are useful for the detection of bone injuries but frequently nonspecific for PSD. Sonographic assessment of the proximal portion of the suspensory ligament is difficult. High-field MR imaging was recently shown to be the most reliable technique for accurate diagnosis of the causes of proximal metatarsal pain.[1,2]

It is recommended that a complete radiographic examination of the tarsus and proximal metatarsal regions is always performed because distal hock joint pain and PSD may coexist in horses with proximal metatarsal pain. Accurate radiographic assessment of the proximal aspect of the third metatarsal bone for the presence of increased radiopacity or avulsion fractures requires that radiographic views be centered at this level. In the dorsoplantar image of the proximal aspect of the third metatarsal bones of sound horses, there can be a variable amount of increased radiopacity that should not be interpreted as a pathological stress reaction at the origin of the suspensory ligament. In the dorsoplantar image, increased opacity is frequently most obvious laterally. On lateromedial images, remodeling changes may include thickening of the plantar cortex, endosteal new bone, alteration of the trabecular pattern of the proximoplantar aspect, and enthesophyte formation on the plantar aspect of the third metatarsal bone. Although increased radiopacity in this region may be more extensive in horses with chronic PSD, these radiographic findings are frequently not specific. In a recent report, features of PSD in 155 horses showed that 21% of lame limbs had a spur on the dorsoproximal aspect of the third metatarsal bone; 30% had mild, diffusely increased radiopacity proximolaterally in the third metatarsal bone; 3% had focal areas of intensely increased radiopacity; and 6% had low-grade osteoarthritis of the distal tarsal joints.[4]

Ultrasonographic examination of the proximal portion of the suspensory ligament is performed with the use of a ≥7.5-MHz linear-array transducer and must always include comparison with the contralateral limb. A delay of 1 to 2 days after diagnostic anesthesia is useful to avoid imaging artifacts caused by air in the tissues. Alternatively, the ultrasonographic examination may precede the use of nerve blocks. Even with careful attention to detail, ultrasonographic examination of the proximal metatarsal region is a difficult technique. The superficial and deep digital flexor tendons and the inferior check ligament are superimposed over the suspensory ligament. The plantar soft tissue structures all have a different echogenicity, slightly different fiber orientation, and acoustic impedances. Overlying tendons and large vessels also cause refraction and enhancement artifacts, adding to the heterogeneity in the echogenicity of the suspensory ligament itself. The margins of the splint bones interfere with visualization of the lateral and medial borders of the ligament. These factors significantly compromise the ability to interpret the morphology of the proximal portion of the suspensory ligament accurately. Modifications to ultrasonographic techniques that have been suggested to improve accuracy include a plantaromedial position for the ultrasound probe, holding the limb in a non–weight-bearing position, and the use of an off-incidence ultrasound beam.[8] In addition, the use of stand-off pads and convex-array or virtual convex-array transducers may offer a wider field of view on the proximal portion of the suspensory ligament. The suspensory ligament has a more heterogeneous echogenicity than the flexor tendons and a less linear fiber pattern on longitudinal views. At its origin, the echogenicity is most variable and can include normal lateral and medial hypoechoic regions that must be differentiated from pathology. These normal variants are caused by areas of looser connective tissue within the ligament containing fat and vascular elements.[9] Unlike the normal echogenicity variants, pathological changes tend to be asymmetrical and are associated with other changes such as altered linear fiber pattern and enlargement (Fig. 2). The suspensory ligament separates from the plantar metatarsal cortex at 4 cm distal to the level of the tarsometatarsal joint.[10] Distal to this point, the dorsal border of the ligament should be distinct and separated from the underlying metatarsal cortex by a small anechoic gap. It has been suggested that the presence of injury of the proximal part of the suspensory ligament is most commonly recognized by the presence of ultrasonographic enlargement, with poor demarcation of the borders and diffuse reduction of the echogenicity rather than by the presence of focal areas of hypoechogenicity. It should be pointed out that this suggestion is not in accordance with the focal nature of many lesions as described in recent MR imaging studies[1,2] (Fig. 3). An irregular contour of the plantar aspect of the third metatarsal bone may indicate enthesophyte formation, but this finding may not be specific for the presence of PSD. It has also been suggested that there is little use in measuring the cross-sectional area of the suspensory ligament at this level because only the central third of the proximal portion of the ligament can be visualized with the routine ultrasonographic technique. In addition, the cross-sectional area of the suspensory ligament changes markedly in its most proximal 6 cm, the area in which most lesions occur. The cross-sectional area of normal suspensory ligaments was measured on MR images as 0.86 cm^2 at the level of the tarsometatarsal joint, 2.08 cm^2 at 2 cm, 1.81 cm^2 at 4 cm, 1.69 cm^2 at 6 cm, and 1.57 cm^2 at 8 cm distal to the level of the tarsometatarsal joint.[10] Even so, it has been suggested that ultrasonographic cross-sectional area measurements of >1.5 cm^2 are suggestive of PSD.[11]

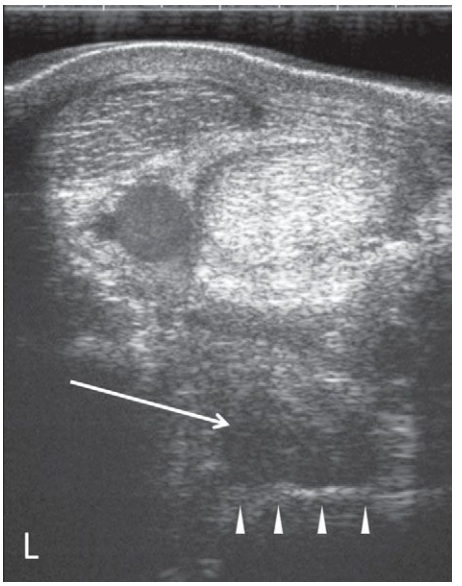


Fig. 2. Transverse ultrasonographic image of the right hind proximal plantar metatarsal region of a horse with severe proximal suspensory desmitis, 2 cm distal to the level of the tarsometatarsal joint. There is a large hypoechoic area (arrow) in the dorsal half of the ligament, adjacent to the ligament's attachment to the plantar cortex of the third metatarsal bone (arrowheads). L indicates the lateral aspect of the limb.

Nuclear scintigraphy of the proximal metatarsal region of sound horses has shown a characteristic distribution of radiopharmaceutical uptake. Maximum uptake occurred at the central to plantar aspect of the proximal metatarsal region in the lateral images, with peak activity over the lateral portion of the proximal metatarsal region on plantar images. In addition, there was a significant difference between left and right proximal metatarsal regions, with higher radiopharmaceutical uptake in the right hind limb.[12] However, nuclear scintigraphy cannot be considered a sensitive tool for the detection of

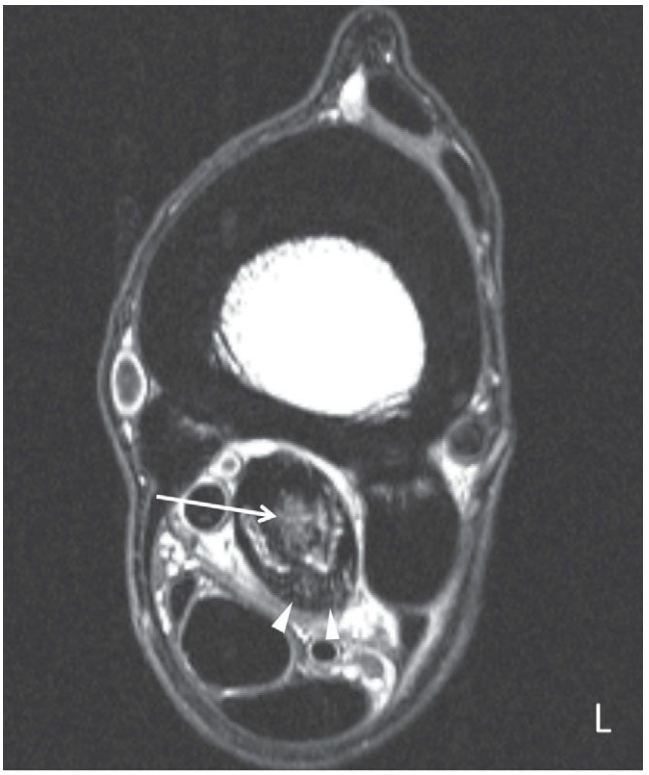


Fig. 3. Transverse proton density MR image of the right hind proximal plantar metatarsal region of a horse with mild to moderate proximal suspensory desmitis, 4 cm distal to the level of the tarsometatarsal joint. There is a small, focal, abnormal, central area of signal hyperintensity area (arrow) in the central collagenous portion of the ligament, interposed between the two normally hyperintense, lateral and medial muscle fiber bundles. There is also mild generalized signal increase in the palmar half of the ligament (arrowheads). L indicates the lateral aspect of the limb.

PSD in the hind limbs of lame horses.[13] Both pool and bone phase images were found to be abnormal in only 12% of horses with ultrasonographic evidence of PSD. Increased radiopharmaceutical uptake was associated with the more severe ultrasonographic lesions. Increased radiopharmaceutical uptake in the proximoplantar aspect of the third metatarsal bone without detectable ultrasonographic or radiographic abnormalities probably represents primary osseous pathology such as stress injury or enthesopathy at the origin of the suspensory ligament rather than PSD *per se*. In a recent study of 155 horses with PSD, there was increased radiopharmaceutical uptake in the proximoplantar aspect of the third metatarsal bone, unilaterally or bilaterally, in 15% of horses.[5]

MR imaging is the best-suited imaging technique for assessment of the proximal portion of the suspensory ligament, thanks to its superior soft tissue resolution and detail. MR imaging is able to visualize inflammatory fluid within bone, tendons, or ligaments when gross pathologic changes have not yet occurred and cross-sectional images allow accurate assessment of the size of the ligament. High-field MR imaging is a more sensitive imaging technique than is ultrasonography for lesions that have bone edema, mild to moderate sclerosis, fiber disruption, or ligament enlargement. Objective comparisons of ultrasonography, MR imaging, and histology have been performed for the proximal part of the suspensory ligament of hind limbs of sound horses.[10,14] MR imaging allowed accurate quantification of suspensory ligament dimensions and accurate identification of tissue bundles containing muscle, adipose, and loose connective tissue throughout the entire length of the origin and body of the suspensory ligament, whereas ultrasonography was unable to distinguish these bundles from surrounding dense collagenous tissue.[10]

High-field MR imaging findings in lame horses indicated that lesions of the proximal part of the suspensory ligament consisted predominantly of focal areas of signal increase that extended on average from 14.2 mm to 50.4 mm distal to the level of the tarsometatarsal joint, with lesion length varying from 4.3 mm to 107 mm. When comparing ultrasonographic with MR imaging findings, ultrasonography was found to have a sensitivity of 66% and a specificity of 31% for the diagnosis of confirmed PSD. Because of the high incidence of false-positive ultrasonographic diagnoses, ultrasonography was considered of limited value for the detection of PSD.[2] In comparison with high-field magnets, low-field MR imaging on standing horses only has a limited ability to show anatomic detail of the proximal portion of the suspensory ligament and to detect soft tissue lesions accurately, mainly because of imaging artifacts caused by movement of the horse. Consequently, the most common primary abnormalities detected with low-field MR imaging in this region are osseous injuries.[15]

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Review of Equine Cutaneous Leishmaniasis: Not Just a Foreign Animal Disease

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Although long considered to be a foreign animal disease, cutaneous leishmaniasis has recently been identified in two horses in Florida with no history of international travel. Both horses were diagnosed with *Leishmania siamensis*, an organism with zoonotic potential. Practitioners in the United States should have this disease on their differential list for horses with ulcers and nodules on the ears, head, and neck. Author's address: University of Florida, Department of Large Animal Clinical Sciences, PO Box 100136, Gainesville, FL 32610; e-mail: sreuss@ufl.edu. © 2013 AAEP.

1. Introduction

Leishmaniasis is a zoonotic disease most well described in people and dogs; however, cutaneous leishmaniasis has been documented in horses around the world. Although cases have been seen in the United States, most of these horses had a history of recent importation from endemic areas. In 2012, the first autochthonous (non–travel-related) case of equine cutaneous leishmaniasis in the United States was published with the discovery of *Leishmania siamensis* in a Morgan mare in Florida.[1] Since that time, a second horse in Florida (also with no history of international travel) has been diagnosed with *L siamensis*.[a] Therefore, leishmaniasis should be included in the differential list for horses in the United States with cutaneous lesions regardless of their travel history or country of origin. The goal of this article is to raise the awareness of this disease amongst equine practitioners by reviewing the etiology, pathogenesis, clinical syndromes, diagnosis, and treatment of leishmaniasis.

2. Etiology/Method of Transmission

Leishmaniasis is caused by the obligate intracellular protozoa of the genus *Leishmania.* In mammals, the parasites reside as intracellular amastigotes within macrophages, where they replicate by binary fission. When infected macrophages rupture, surrounding macrophages phagocytize the amastigotes and become infected. The disease is endemic in tropical and subtropical regions but is spreading with global climate change. There are more than 30 species of *Leishmania* with a complex classification scheme. The species of *Leishmania* vary with region, and different species are often incriminated to cause different disease manifestations. In the Eastern Hemisphere, the "Old World" species *Leishmania donovani, Leishmania infantum, Leishmania tropica*, and *Leishmania aethiopica* are commonly found. In the Western Hemisphere, the "New World" species *Leishmania braziliensis* and *Leishmania mexicana* are frequently found, most commonly in South and Central America. However, *L mexicana* has been reported as the causative organism of some vector-transmitted outbreaks of cutaneous leishmaniasis in people and dogs in Texas.[2] *L siamensis* is a recently described species that had not been previously reported in North America until it was determined to be the etiologic organism in both Florida horse cases.[1] *L siamensis*

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was first described as the cause of autochthonous visceral leishmaniasis in two men in Southern Thailand[3,4] but has also been identified as the cause of equine cutaneous lesions in central Europe[5] and a cow in Switzerland.[6]

In all mammalian species affected with leishmaniasis, the primary mode of transmission is believed to be by various species of sand flies. The promastigote stages of the protozoa are transmitted in the saliva of the female sand fly at the moment of blood feeding.[7] Each species of *Leishmania* is adapted to transmission in certain species of sandflies: *Phlebotomus spp* in the Old World and *Lutzomyia spp* in the New World. The vector in equine transmission in the United States remains unidentified; however, the phlebotomine sand flies *Lutzomyia shannoni* and *Lutzomyia vexator* are found in Florida, where the two autochthonous equine cases were diagnosed.[8] *Lutzomyia shannoni* is known to use both humans and other mammals as hosts, and it has been incriminated as a vector of *L braziliensis* in Central and South America. It has also been shown experimentally to be capable of becoming infected with *L infantum* when feeding on ill *L infantum*-infected dogs.[9] However, it is not known whether *L shannoni* will permit *Leishmania* development into infectious metacyclic parasites. *Leishmania mexicana* is considered endemic in south-central Texas, where the organism is maintained within the ecosystem by small rodents and is presumably spread by a *Lutzomyia spp* (possibly *Lutzomyia diabolica*).[2] Although vector-borne transmission is the primary means of spread between dogs in endemic areas, it is thought that vertical transmission (transplacental and transmammary) is probably the major means of spread in dogs in non-endemic areas of the United States.[10] The frequency of vertical transmission in endemic regions is difficult to determine because of the high likelihood of vector contact. Ticks have been shown experimentally to be capable of transmission in dogs, and horizontal transmission by direct contact with blood has also been shown.[10]

3. Clinical Manifestations

Equine

Cutaneous leishmaniasis has been documented in horses around the world. Lesions are most commonly observed as nodules on the head, pinnae, scrotum, legs, and neck. They may be solitary or multiple, and are often ulcerated (Fig. 1). No other signs are seen, and visceral lesions have not been reported. Equine leishmaniasis was first reported in Argentina in 1927. In South America, *L braziliensis* has been identified as the causative organism in horses.[11,12] In one endemic region of Brazil, 26 horses, donkeys, and mules were examined. Cutaneous lesions were found in 10 animals, and eight animals (30.8% of the examined population) had parasites seen on histopathology of ulcer margins.[13]



Fig. 1. Cutaneous leishmaniasis in the left pinna of a 10-year-old Morgan horse mare in Florida.

In Europe, *L infantum* has been reported as the causative agent of cutaneous leishmaniasis in horses in Germany,[14] Spain,[15] and Portugal.[16] Recently, a report from central Europe identified an organism with 98% identity to *L siamensis* as the cause of cutaneous lesions in four horses.[4] Whereas equine leishmaniasis is documented in Puerto Rico,[17] it is rarely seen in the continental United States. Historically, cutaneous leishmaniasis in the United States was seen in horses with a history of international travel or recent importation. Although autochthonous cases may have been observed[2], the first documented case occurred in 2011.[1] To date, only cutaneous lesions have been documented in horses.

Human

Leishmaniasis is the second leading parasitic cause of death in people, after malaria. There are approximately 1.8 million new cases per year, making this a top priority for the World Health Organization. Three basic syndromes are seen in people: cutaneous, mucocutaneous, and visceral. Cutaneous disease presents as painless nodules, plaques, or ulcers. In general, lesions heal spontaneously in immunocompetent individuals. Mucocutaneous lesions involve the nares, nasal septum, pharynx, lar-

ynx, and genitalia; and they can occur concurrently or 1 to 5 years after resolution of cutaneous lesions. These lesions will not heal spontaneously. Visceral leishmaniasis, otherwise known as "kala azar," is a chronic, insidious disease that may be preceded by cutaneous lesions. Visceral leishmaniasis is usually associated with species of the *Leishmania donovani* complex (*L donovani, L infantum, Leishmania chagasi*) and *L tropica*. Clinical signs include fever, weight loss, anemia, anorexia, cough, diarrhea, and darkening of the skin. Splenomegaly, hepatomegaly, lymphadenopathy, thrombocytopenia, and leukopenia may all be present as the parasite invades the reticuloendothelial system. Visceral disease is fatal unless treated, and treated patients will remain carriers and can recrudesce if immunosuppressed.

Canine

Dogs are the most commonly affected domestic species, with 63% to 80% seropositivity in endemic areas in which they are thought to be the primary reservoir for *L infantum* and maintain the disease in domestic cycles.[10] In endemic areas, all breeds of dogs are affected. Dogs, like humans, can develop cutaneous or visceral disease. Cutaneous lesions can present as nonpruritic exfoliative dermatitis, nodules, ulcers, and long, brittle nails. Visceral disease results in lethargy, weight loss, anorexia, anemia, splenomegaly, epistaxis, hematuria, melena, chronic renal failure, and death. Ocular lesions have also been reported and include conjunctivitis, keratitis, and uveitis. The majority of cases of canine leishmaniasis in the United States have historically involved international travel. However, there have been reports of isolated cases and outbreaks, primarily in foxhounds. In 2000, a New York kennel reported four foxhounds with *L infantum* visceral leishmaniasis without a history of travel. This led to widespread testing, and by 2005, 60 kennels in 22 states and two Canadian provinces were found to have seropositive Foxhounds. Current studies of Foxhound kennels show a 9.8% seroprevalence; however, in high-risk kennels, the percentage of quantitative polymerase chain reaction (qPCR)-positive dogs is 44.8%.[10]

4. Diagnosis

Microscopic evaluation of an impression smear or biopsy of the border surrounding the cutaneous lesion is often the preliminary means of diagnosis. There will be a marked inflammatory response, and numerous intracellular protozoa will be seen within macrophages and occasionally extracellularly. These 1- to 4-μm intracellular organisms have an eccentrically placed, basophilic, round nucleus and a rod-shaped kinetoplast oriented perpendicular to the long axis of the oval nucleus (Fig. 2). This morphology is indicative of the amasitogote form of *Leishmania*. Electron microscopy will allow more detailed examination of the intracellular amastigotes. Immunohistochemistry can be used to confirm the diagnosis of leishmaniasis and has been performed successfully in the horse.[5,14,17]

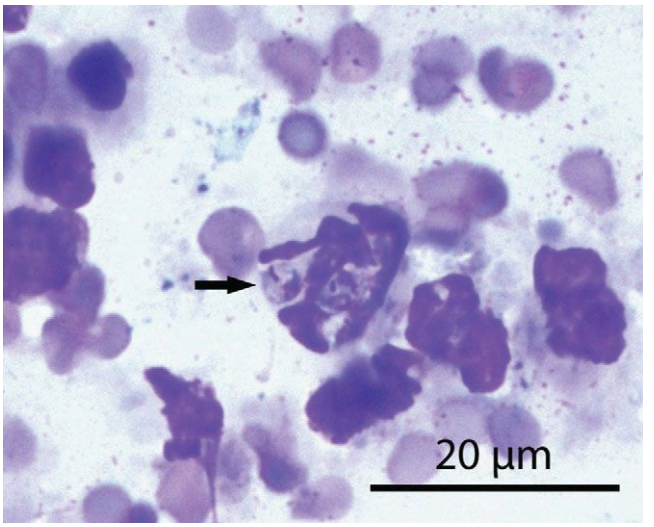


Fig. 2. Fine-needle aspirate of an ulcerated mass from a horse with cutaneous leishmaniasis exhibiting an intracellular amastigote (arrow). Photo courtesy of Dr. Mark Dunbar.

Culture and qPCR can be performed by the Centers for Disease Control and Prevention or by various university research labs. Isoenzyme analysis of cultured parasites has been the conventional approach for species identification. However, PCR and sequence analysis can identify the organism to the species level more rapidly and with greater sensitivity, depending on the targeted region.[18] It is worth noting in the context of equine leishmaniasis that PCR methods used to detect Old World leishmaniasis have been reported to fail to detect *L siamensis*.[1,5,6] Amplification of a 350–base pair internal transcriber spacer 1 (ITS1) fragment has been successful in identifying this organism.[1,5] Sequence analysis of the ITS1 amplification products classifies *L siamensis* as neither Old World nor New World.[5]

Serologic testing in humans and canines is the primary diagnostic test used for surveillance of visceral infections but is less reliable for cutaneous disease. Indirect fluorescent antibody (IFA) testing can be performed by the Centers for Disease Control. Unfortunately, it may cross-react with antibodies to *Trypanosoma cruzi*. One horse with *L braziliensis* was positive for anti-*Leishmania* antibodies on IFAT[11]; however, IFAT in one horse in Germany with confirmed *L infantum* cutaneous lesions on PCR failed to yield a positive result.[14] Cutaneous lesions in people may also result in no detectable level of serum antibodies; however, skin testing for delayed type hypersensitivity may be positive in human cutaneous disease. Delayed type hypersensitivity reaction skin testing has been performed in the horse[11] but is not standardized. Anti–*L infantum* antibodies have been

found in clinically normal horses in endemic areas through the use of a protein-A enzyme-linked immunosorbent assay (ELISA) and a specific lymphocyte proliferation assay to *L infantum*.[19] Other serologic tests used in people and canines with visceral lesions include a kinetic-based ELISA and a K39-antigen–based assay.[20] Quantitative PCR may be a more sensitive test, and can detect animals that are asymptomatic or have not yet seroconverted.[10]

5. Treatment

Different species of *Leishmania* have varying responses to treatment. Systemic treatment is often indicated in humans to reduce the risk of dissemination to the mucosa or viscera. Currently, the standard treatment for people is intravenous administration of the pentavalent antimony compounds sodium stibogluconate or meglumine antimonite. Unfortunately, these drugs have potentially severe side effects including arthralgia, myalgia, pancreatitis, and abnormal liver function tests, and results in treatment failure in 23.5% of cases.[21] Intralesional pentavalent antimony can be an effective alternative, resulting in higher drug concentrations at the site of infection while reducing the risk of systemic toxicity.[22] In some endemic areas, drug resistance for pentavalent antimony is seen.[23] In the United States, pentavalent antimony can only be obtained for military or investigational use from the Centers for Disease Control and Prevention, thus limiting clinical access.

Other drugs that have been used for human and canine leishmaniasis include allopurinol, miltefosine, amphotericin B in the lipid emulsion or liposomal form, ketoconazole,[18] paromomycin,[24] itraconazole, and cryotherapy. A randomized, double-blind, placebo-controlled trial of oral fluconazole was found to be safe and shortened time to resolution in people with cutaneous lesions.[25] However, a recent Cochrane database review of 38 clinical trials for cutaneous lesions through the use of different interventions found an absence of randomized, controlled, trial-based evidence for alternative treatments including surgery, oral itraconazole and fluconazole, rifampicin, metronidazole and cotrimoxazole, intravenous or topical amphotericin B, oral dapsone, photodynamic therapy, and laser and cryotherapy.[26]

Many of the reports of cutaneous leishmaniasis in horses describe spontaneous regression of lesions. Surgical resection,[5] pentavalent antimony,[11,17] and amphotericin and fluconazole[1] have all been used "successfully" in horses, but it is unclear how many would have resolved with no treatment.

6. Summary

Equine cutaneous leishmaniasis can no longer be considered just a foreign animal disease. With climate change and the spread of vector habitats, emerging diseases will continue to infiltrate the United States equine population. Leishmaniasis should be considered in the differential list for cutaneous lesions in horses; and cytology, histopathology, and/or molecular diagnostics should be performed on suspect lesions. Although this is not a fatal disease in horses, we can and should consider our equine patients as sentinels for this potentially fatal zoonotic disease.

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Emerging Outbreaks Associated With Equine Coronavirus in Adult Horses

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Equine coronavirus (ECoV) is associated with self-limiting clinical and hematological abnormalities in adult horses. Real-time polymerase chain reaction is a sensitive and fast diagnostic tool to document the presence of ECoV in feces from horses with lethargy, anorexia, fever, and changes in fecal character. Authors' addresses: Department of Medicine and Epidemiology, School of Veterinary Medicine, University of California, Davis, CA 95616 (Pusterla, Mapes, Wademan, White, Magdesian); Bracken Equine Clinic, 18675 Marbach Lane, San Antonio, TX 78266 (Ball, Sapp); Elkhorn Veterinary Clinic, 205 Oconnor Drive, Elkhorn, WI 53121 (Burns); Oak Hill Veterinary Services, 1755 Locust Street, Walnut Creek, CA 94596 (Ormond); SRH Veterinary Services, 295 High Street, Ipswich, MA 01938 (Butterworth); New England Equine Medical and Surgical Center, 15 Members Way, Dover, NH 03820 (Bartol); e-mail: npusterla@ucdavis.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Outbreaks associated with equine coronavirus (ECoV) in adult horses have rarely been reported in the literature. The purpose of this study was to describe clinical and laboratory results from 161 adult horses involved in outbreaks associated with ECoV.

2. Materials and Methods

The outbreaks occurred at four separate boarding facilities between November 2011 and April 2012 in the states of California, Texas, Wisconsin, and Massachusetts. After the molecular detection of ECoV in the feces from the initial index cases, the remaining herdmates were closely observed for the development of clinical signs. Fecal samples were collected from sick and healthy horses for the polymerase chain reaction (PCR) detection of ECoV.

3. Results and Discussion

Fifty-nine adult horses had development of clinical signs, with 12 to 16 sick horses per outbreak. The main clinical signs reported were anorexia, lethargy, and fever. Blood work was available from 10 horses with clinical disease, and common hematological abnormalities were leucopenia caused by neutropenia and/or lymphopenia. Feces were available for ECoV testing by real-time PCR from 44 and 96 sick and healthy horses, respectively. The

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overall agreement between clinical status and PCR detection of ECoV was 91%. The study results suggest that ECoV is associated with self-limiting clinical and hematological abnormalities in adult horses.

Acknowledgment

This study was supported by Boehringer Ingelheim Vetmedica.

Investigation of the Use of Pooled Fecal and Environmental Samples After an Enrichment Step for the Detection of *Salmonella* spp. by Real-Time Polymerase Chain Reaction

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Biosurveillance for *Salmonella* spp. in equine hospitals has become a recognized standard of care. The pooling strategy is able to reduce the overall costs of *Salmonella* spp. testing while maintaining the same detection accuracy as microbiological culture. Authors' addresses: Department of Medicine and Epidemiology (Pusterla, Mapes, Akana, Wademan, Magdesian) and Department of Pathology, Microbiology and Immunology (Byrne), School of Veterinary Medicine, University of California, Davis, CA 95616; Loomis Basin Equine Medical Center, 3901 Sierra College Blvd., Loomis, CA 95650 (Fielding); Hagyard Equine Medical Institute, 4250 Iron Works Pike, Lexington, KY 40511 (Slovis, Elam); e-mail: npusterla@ucdavis.edu. *Corresponding and presenting author.
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1. Introduction

Generally, microbiologic culture of feces, tissue, or body fluids is used to detect *Salmonella* spp. infection in horses. Reliability of isolation of the organism by culture is diminished by various factors, including method used to collect the sample, amount of sample submitted, temporal and seasonal variation in shedding of the organism, and method of bacteriologic culture. Clinical laboratories generally require at least 48 hours for presumptive detection of *Salmonella* spp. in feces with enrichment to detect small numbers of *Salmonella* spp. Several studies have evaluated the use of polymerase chain reaction (PCR) for the detection of *Salmonella* spp. in fecal and environmental samples. Although PCR has increased sensitivity and faster turnaround time, the costs associated with this testing platform are generally higher than microbiological culture. The purpose of this study was to evaluate the pooling of feces and environmental samples after a selective enrichment culture step for the detection of *Salmonella* spp. by real-time PCR.

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2. Materials and Methods

For the purpose of this study, 677 equine fecal and 686 environmental samples were collected. Each sample was inoculated into selenite or tetrathionate broth and incubated for 18 to 24 hours. After incubation, the enrichment broth samples were subcultured onto xylose-lysine-tergitol-4 or hektoen agar plates. Suspected *Salmonella* spp. colonies were subcultured and further identified with the use of biochemical assays. Concurrently to the microbiological analysis, 1 mL of the enrichment broth was processed for DNA purification. The samples were analyzed individually (1363 samples) and in pools of up to 10 samples (139 pools) with the use of a *Salmonella* spp. real-time PCR assay targeting the *invasion A* gene.

3. Results and Discussion

The pooling strategy was able to detect all fecal and environmental samples dually positive by PCR and culture. Three environmental sample pools tested PCR-positive; each of these pools contained two to five individual culture and PCR-positive samples. Two additional PCR-positive and culture-negative environmental samples tested negative by PCR when pooled together. Eleven fecal samples cultured positive for *Salmonella* spp. All these fecal samples were also PCR-positive at the individual and pooled levels. Eight additional PCR-positive and culture-negative fecal samples gave rise to five positive and three negative pools.

Acknowledgment

This study was supported by a grant from The Center for Equine Health, University of California, School of Veterinary Medicine at Davis and Zoetis.

Comparison of Immune Responses in Healthy Foals When a Multivalent Vaccine Protocol Was Initiated at 90 or 180 Days of Age

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Foals are capable of immune activation after a three-dose immunization series with a multivalent vaccine started at 90 days of age, despite the presence of maternal antibodies. Authors' addresses: Department of Clinical Sciences (Davis, Bryan) and Diagnostic Medicine Pathobiology (Wilkerson), College of Veterinary Medicine, Department of Statistics (Bello), Kansas State University, Manhattan, KS 66506; Pfizer (Zoetis) Animal Health, Global Headquarters, 100 Campus Drive, Florham Park, NJ 07932 (Hankins); e-mail: edavis@vet.k-state.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Induction of immunity to protect against infectious disease requires the implementation of an effective immunization program. In foals, most vaccine protocols are delayed until waning of maternal antibodies to avoid maternal antibody interference. It was the aim of this investigation to characterize immune activation among healthy foals in response to administration of a multivalent vaccine protocol and compare immune responses when foals were vaccinated at either 90 or 180 days of age.

2. Materials and Methods

Twelve healthy foals that received adequate colostrum were included in the investigation. Foals were blocked for age and randomly assigned to receive a three-dose vaccine protocol at 90 or 180 days of age. Immune activation was characterized by the measurement of CD4+ and CD8+ interferon-γ, interleukin-4, granzyme B, and major histocompatibility complex II expression after in vitro cellular stimulation with vaccine antigens that included Eastern equine encephalitis (EEE), Western equine encephalitis (WEE), West Nile virus (WNV), equine influenza virus (EIV), and equine herpesvirus (EHV)-1/4, and tetanus toxoid antigen. Total antigen-specific immunoglobulin G was assessed with the use of a flow cytometry–based microsphere bead assay. Time points of interest included 30 days after initial vaccination and at the end of the investigation, when vaccination was administered at approximately 11 months of age. Assessment of similarity was performed with bioequivalence testing; similarity was defined with a 95% level of significance.

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3. Results

Comparable results were obtained for several measures of cellular immunity. In particular, antigen-specific CD4+ and CD8+ cellular expression of the cytokines interferon-γ and interleukin-4 were evident at specified time points for several vaccine antigens.

4. Discussion

The data presented in this report demonstrate that young foals are capable of immune activation after a three-dose immunization series with a multivalent vaccine, despite the presence of maternal antibodies. Although immune activation does not automatically translate into protection, several of the immune indicators measured showed comparable expression in foals vaccinated at 3 months of age, relative to those in the control group of foals initially vaccinated at 6 months of age. In a high-risk situation in which immune activation may be required earlier than at the completion of a conventional vaccine series, our data support that foals can respond to immunization that is initiated at 3 months of age in a manner comparable to that in foals initiated at an older age, provided that the immunization protocols include three doses followed by a booster at 11 months of age.

Evaluation of Hematologic Screening Methods for Predicting Subsequent Onset of Clinically Apparent *Rhodococcus Equi* Pneumonia in Foals

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Hematologic screening parameters have poor sensitivity and poor specificity for predicting subsequent onset of clinically apparent *Rhodococcus equi* pneumonia. Practitioners at *R equi*–endemic breeding farms should be aware of these limitations of hematologic screening. Authors' addresses: Texas A&M University, College of Veterinary Medicine, College Station, TX 77843 (Chaffin, Cohen); 6666 Ranch, PO Box 130, Guthrie, TX 79236–0130 (Blodgett); PO Box 1523, Basalt, CO 81621 (Syndergaard); e-mail: kchaffin@cvm.tamu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Hematologic parameters, including white blood cell concentration (WBC) and fibrinogen concentration (FC), are commonly used at endemic breeding farms to screen for early detection of *Rhodococcus equi* pneumonia in foals. The objectives of this study were to estimate the sensitivity and specificity of three hematologic screening methods (sequential measurement of WBC, neutrophil concentration [NC], and FC) for predicting subsequent onset of clinically-apparent *R equi* pneumonia.

2. Materials and Methods

Foals (n = 270) were studied at an *R equi*–endemic farm. Foals were screened hematologically every 2 weeks from 3 to 19 weeks of age. Farm personnel were blinded to screening results. Foals were not treated with antimicrobials unless they demonstrated clinical signs of pneumonia. Tracheobronchial aspirates were obtained from all pneumonic foals.

3. Results

Seventeen percent of foals had development of clinically apparent *R equi* pneumonia. Cumulative sensitivities for WBC (using a cut-point of ≥13,000/μL), NC (cut-point of ≥10,000/μL), and FC (cut-point of ≥600 mg/dL) were 59%, 50%, and 59%, respectively, for predicting subsequent onset of clinically-apparent *R equi* pneumonia. Respective cumulative specificities were 37%, 55%, and 33%.

4. Discussion

Hematologic screening parameters demonstrated limited performance for predicting subsequent onset of clinically apparent *R equi* pneumonia in foals.

Acknowledgments

This project was supported by The American Quarter Horse Foundation, Link Equine Research Endowment at Texas A&M University, the 6666 Ranch, and the Michael and Paula Gaughan Fund for Equine Internal Medicine.

Research Abstract

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Evaluation of Ultrasonographic Screening Parameters for Predicting Subsequent Onset of Clinically Apparent *Rhodococcus Equi* Pneumonia in Foals

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Subclinical, sonographically visible lung consolidations are highly prevalent in foals. In most foals, such consolidations resolve without therapy or onset of clinically apparent pneumonia. Ultrasound screening parameters have good sensitivity but relatively weak specificity for predicting subsequent onset of clinically apparent *Rhodococcus equi* pneumonia. Authors' addresses: Texas A&M University, College of Veterinary Medicine, College Station, TX, 77843 (Chaffin, Cohen); 6666 Ranch, PO Box 130, Guthrie, TX, 79236–0130 (Blodgett); PO Box 1523, Basalt, CO 81621 (Syndergaard); e-mail: kchaffin@cvm.tamu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The objectives of this study were to estimate the sensitivity and specificity of three ultrasonographic screening parameters (sequential measurements of pulmonary consolidations including total maximal diameter [TMD], total cross-sectional area [TCSA], and total number of consolidations [TNC]) for predicting subsequent onset of clinically apparent *Rhodococcus equi* pneumonia.

2. Materials and Methods

Foals ($n = 270$) were studied at an *R equi*–endemic farm. Foals were screened sonographically every 2 weeks from 3 to 19 weeks of age. Farm personnel were blinded to screening results. Foals were not treated with antimicrobials unless they demonstrated clinical signs of pneumonia. Tracheobronchial aspirates were obtained from all pneumonic foals.

3. Results

Two hundred sixteen (80%) foals had development of sonographically visible pulmonary consolidations. Consolidations resolved without treatment or clinical illness in 79% of these foals; 17% of the 270 foals had development of clinically apparent *R equi* pneumonia. Cumulative sensitivities for TMD (cut-point of ≥200 mm), TCSA (cut-point of >100 mm^2), and TNC (cut-point of ≥2) were 89%, 91%, and 78%, respectively. Respective cumulative specificities were 62%, 54%, and 64%.

Research Abstract

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4. Discussion

Subclinical lung consolidations are highly prevalent in foals. In most foals with lung consolidations, the lesions resolve without clinically apparent illness or treatment. Ultrasonographic screening parameters have good sensitivity but relatively limited specificity for predicting which foals will progress to clinically-apparent *R equi* pneumonia.

Acknowledgments

This project was supported by The American Quarter Horse Foundation, Link Equine Research Endowment at Texas A&M University, the 6666 Ranch and the Michael and Paula Gaughan Fund for Equine Internal Medicine.

Effects of Astaxanthin and L-Carnitine Supplement on Muscle Damage and Incidence of Equine Tying-Up Syndrome

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Continuous administration of astaxanthin and L-carnitine supplement has preventive effects on the onset of tying-up syndrome and thus may contribute to maintaining performance in Thoroughbred horses. Authors' addresses: Hidaka Training and Research Center, Japan Racing Association, 535–1 Nshicha, Urakawa, Hokkaido 057–0171, Japan (Sato, Omura, Ishimaru, Korosue, Endo, Murase and Nambo); Life Science Division, Fuji Chemical Industry Co, Ltd, 55 Yokohoonji, Kamiichi, Nakaniikawa, Toyama 930–0397, Japan (Yamashita); e-mail: Fumio_Sato@jra.go.jp. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Tying-up syndrome is the most common muscle disorder in training horses. We focused on both the strong antioxidant effect of astaxanthin and the enhancement of muscle fatty-acid oxidation of L-carnitine. The effects of dietary administration of astaxanthin and L-carnitine supplement on serum markers of exercise-induced muscle damage and the onset of tying-up syndrome in training horses were investigated.

2. Materials and Methods

A supplement group ($n = 31$) received daily supplementation with astaxanthin (75 mg) and L-carnitine (3000 mg) for 8 weeks; a control group ($n = 32$) received no supplementation. Blood samples were collected after exercise training before supplementation and 3 days and 8 weeks after starting supplementation. The symptoms of tying-up syndrome in both groups were retrospectively evaluated.

3. Results

Within the control group, creatine kinase activity at 8 weeks was significantly increased compared with 3 days; no significant change was observed in the supplement group. After 8 weeks, creatine kinase activity in the supplement group was significantly lower than that in the control group; lactate dehydrogenase-5 also tended to be lower in the supplement group. The incidence of tying-up syndrome in the supplement group was significantly lower than that in the control group.

4. Discussion

Continuous dietary administration of astaxanthin and L-carnitine attenuates exercise-induced muscle damage and prevents the onset of tying-up syndrome in Thoroughbred horses.

Research Abstract

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Comparison of the Smartphone Electrocardiogram Device With a Reference Standard Base Apex Electrocardiogram in Horses

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Our data suggest that the smartphone-acquired electrocardiogram (SAE) accurately identifies cardiac rhythms and heart rate in horses. Authors' address: Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853; e-mail: msk16@cornell.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The smartphone-acquired electrocardiogram (SAE) device[a] incorporates electrodes into a smartphone[b] hand-held case, allowing wireless recordings of electrocardiograms. We hypothesized that the SAE would permit immediate and accurate heart rate and rhythm identification in horses with normal sinus rhythm and spontaneous arrhythmias when compared with a reference electrocardiogram (ECG).

2. Materials and Methods

A standardized base apex ECG and SAE recordings were acquired simultaneously from 20 horses (with spontaneous arrhythmias). Instantaneous heart rates were obtained from identical QRS complexes in which these were identified; 15-second average heart rates were obtained in which identical QRS complexes were not identified. Three observers independently evaluated the rhythm and the polarity of QRS depolarization for each recording. The results were compared within-observer and between observers.

3. Results

Instantaneous and average heart rates were identical in all cases in which exact matches could be made for comparison between the smart phone and reference ECG and were within 1 beat when average heart rates were calculated. Intra-observer agreement for rhythm assessment was very high, with no disagreement for equine ECGs. The polarity of depolarization revealed minimal disagreement between the SAE and reference ECG in horses. Inter-observer agreement for SAE ECGs was similar to that for reference ECGs, with all observers agreeing on the rhythm analysis, with minimal disagreement on polarity.

Research Abstract

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4. Conclusions

With this new hand-held device, ECGs can be observed immediately, and, when necessary, e-mailed to a consulting veterinarian or cardiologist for advice on diagnosis and treatment.

The authors thank AliveCor for providing the equipment (iPhone ECG case).

Footnotes

[a]iPhone ECG, AliveCor, San Francisco, CA 94108.
[b]4GS iPhone, Apple, Cupertino, CA 95014.

Lethal Avocado Toxicity in Three Horses in North America

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Avocado intoxication should be considered in horses presenting with the outlined signs. Owners should be advised of the guarded to grave prognosis associated with ingestion; accordingly, horses should not be allowed access to avocado trees. Authors' addresses: Alamo Pintado Equine Medical Center, 2501 Santa Barbara Avenue, Los Olivos, CA 93441 (Lorbiecki, Kwon, Hamer, Rubin, Priest, Lopez, Palmero); California Animal Health and Food Safety, PO Box 1770, Davis, CA 95617 (Poppenga); e-mail: jlorbieckidvm@gmail.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Avocado toxicity is reported in various species. Persin, a fungicidal toxin known to affect mammary and myocardial tissue, is often implicated. This report describes variable lethal cardiac, pulmonary, and neurologic manifestations in three North American horses after avocado exposure.

2. Materials and Methods

Three adult horses were presented to a referral center after 4 days of intermittent grazing in a Hass avocado (*Persea americana*) orchard. The horses presented at different intervals after exposure, each displaying unique signs. Clinical signs and hematological findings (available for all cases) and histological and toxicological findings (available for some cases) were reviewed and summarized.

3. Results

Case 1 had colic and cardiac arrhythmia. Case 2 had respiratory distress. Case 3 initially had mild facial edema and later respiratory and neurologic dysfunction. All horses presented with tachycardia, tachypnea, toxic mucus membranes, edema, elevated serum lactate, and variable polycythemia. Cases 1 and 2 presented with hypoproteinemia, hypocalcemia, and elevated creatine kinase levels. Cases 1 and 3 had elevated serum troponin I. Case 3 had pericardial effusion. All horses died or were euthanized despite supportive care.

4. Conclusions

Avocado intoxication resulted in various clinical manifestations but was uniformly fatal in this study. The variety and severity of signs may be attributable to consumed toxic dose of persin or of a yet undocumented compound.

Research Abstract

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Exploring and Meeting Client Expectations of the Veterinarian-Client Relationship Through the Use of Clinical Communication Skills

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1. Introduction

This report discusses the current research findings regarding veterinarian-client interactions and relationships and how they relate to equine veterinary practice. The application of the relationship-centered care model in equine medicine is described, and proven clinical communication skills that illustrate how to incorporate this model into equine veterinarian-client interactions are provided.[1–3]

2. Relevance

The relationship between an equine veterinarian and a given client can be complex, with elements of collegiality, friendship, and professionalism. The nature of the industry dictates that in many cases, equine veterinarians see their clients on a frequent basis; this necessitates a strong working relationship. The veterinarian must ensure that their relationship with the client facilitates appropriate provision of care for the horse; this requires trust, comfort, and confidence on both sides. Further complexities arise when the client-horse relationship is considered. This relationship is often multifaceted: horses can be investments, athletic partners, companions, or a combination of these. The veterinarian's understanding of the relationship between client and horse is important because it is often a driving factor in the client's decisions. Last, it is also important to consider the relationship between veterinarian and horse. Clients differ in their comfort levels with restraint, attitudes toward their horses, and knowledge of routine procedures; therefore, the manner in which a veterinarian handles and treats their horses can influence the veterinarian-client relationship and veterinary outcomes. The relationships described above form the foundation on which the veterinarian provides appropriate and effective care for the horse and are therefore essential components of veterinary practice.

In equine medicine, there is little research-based evidence regarding client expectations, veterinarian-client communication, and veterinarian-client relationships. The role of good communication in reducing malpractice risk in equine medicine has been discussed.[4,5] As well, in a survey of equine clients that evaluated 24 criteria of client expectations of the overall veterinary service experience, "communication with clients" was ranked fourth; "veterinary competency," "horse handling," and "doctor performance" were the top three.[6] This

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study also looked at areas in which client expectations and veterinarian perceptions differed; the area that had the largest disparity was client education abilities and opportunities.[5] These studies highlight the importance of veterinarian-client communication in equine practice; however, the expectations that clients hold of the veterinarian-client relationship or of veterinarian-client communication are unknown.

Research in companion animal medicine has investigated pet owner expectations of veterinarian-client interactions and veterinarian perceptions of clients' expectations.[7–9] One key area of expectation was veterinarian-client communication, specifically relating to client education and the provision of choice, as well as two-way communication between veterinarian and client.[7] Expectations surrounding the monetary aspects of veterinary care have also been described; these include open discussion of costs and their context and the consideration of the client's financial limitations.[8] Furthermore, it has been shown that on occasion, veterinarians misperceive client expectations.[7–9] When there is a disconnect between the veterinarian's perceptions and the client's expectations, optimal patient care is compromised and the veterinarian-client relationship is jeopardized. Therefore, it is important to recognize the gaps that exist between clients and veterinarians, such that the client's expectations can be managed effectively, and ultimately patient care is optimized.

Several components of companion animal veterinarian-client interactions have been described in the literature and have been shown to affect client and veterinary outcomes, including veterinarian satisfaction, client satisfaction, and adherence to recommendations.[10,11] Client adherence to recommendations has been positively correlated with measures of relationship-centered care and client satisfaction.[11] This study also demonstrated that clear recommendations that were fully explained were more likely to result in client adherence when compared with ambiguous recommendations.[11] Veterinarian satisfaction has also been positively correlated with veterinarian communication behaviors that encourage relationship development.[10] The value and benefits of good communication between veterinarian and client have also been demonstrated.[12,13] Students provided with training utilize more communication skills during clinical interactions and are rated higher by clients with respect to use of communication skills.[12] Specific strategies suggested for ensuring good communication include forming a relationship with the client, demonstrating empathy, educating the client, and enlisting the client as a partner in his or her horse's care.[5,14]

3. Relationship-Centered Care

Relationship-centered care is a model that outlines a framework for veterinarian-client relationships.[2] It involves the establishment of a partnership between client and veterinarian, such that the client's unique needs and expectations are balanced with the veterinarian's role as the medical expert. It incorporates the three primary relationships in veterinary medicine: veterinarian-client, client-horse, and veterinarian-horse.[2] The model of relationship-centered care in veterinary medicine is derived from patient-centered care in human medicine, which highlights the needs and concerns of the patient, such that they shape the care he or she will receive.[3,15] Partnership between doctor and patient, as well as the provision of care that emphasizes the value and uniqueness of each individual in a medical setting, are important principles of patient-centered care.[15] Given the nature and importance of the relationships in equine medicine, relationship-centered care is an appropriate model to use when seeking to form relationships with which a successful practice can be built and maintained.

Investigate the Client's Perspective

Investigating the client's perspective lays the foundation and sets the tone for the rest of the interaction.[3] If, from the beginning, the client is encouraged to participate, their opinions are validated, and they believe that they are understood, then the groundwork is set for the formation of a strong partnership, which will facilitate the rest of the appointment and relationship. Fully exploring the client's opinions and situation provides valuable information regarding what they have been experiencing, about the problem or history of the patient, and the expectations of the client.[3] This information will also be useful later in the interaction when trying to find a mutually acceptable plan, because the plan can be put in the context of the client's unique situation. In the event that a client is not given an opportunity to express himself or herself, problems can arise because the veterinarian does not have the full story, such as the client's past experience with the problem or the treatments that have already been tried. Investigating the client's perspective and history taking are not the same; however, they can be accomplished simultaneously. History-taking is more limited to information about the problem or disease process, whereas the client's thoughts and concerns are included when investigating his or her perspective. The communication skills below can facilitate efficient and thorough exploration of the client's perspective.[1–3]

Open-Ended Questions

The manner in which questions are asked will shape the quality and quantity of the response offered by the client.[1] There are two classes of questions—open-ended and closed-ended—which can be differentiated by the form the answer takes. Closed-ended questions can be answered with a simple yes or no, or another single word. Open-ended ques-

tions allow clients to fully express themselves and describe their experience, thus allowing the clinician to appreciate the whole story of the client and patient's problem.[1,2] At first glance, this may not seem desirable, because information that is unrelated or unnecessary is potentially provided. However, if only closed-ended or very focused questions are asked, valuable information from the client about what has been going on with the horse may be missed. Also, the time spent learning about the horse and client is important for relationship building and rapport. Examples of open-ended questions are "What has been going on with Lucy?" and "What have you noticed that has you concerned?"

Reflective Listening

Reflective listening is a way to demonstrate that the listener is paying attention, to facilitate the client sharing his or her thoughts, and to indicate comprehension.[1,2] A reflective statement is essentially paraphrasing what the client has said. This offers the client a chance to clarify or provide a correction if what was reflected was incorrect or to elaborate if there is more he or she wants to share.[1] It shows clients that the veterinarian is genuinely interested in what they have to say and ensures that he or she is catching all the pertinent information being shared. This skill is particularly useful when multitasking during an appointment, because the client may have concerns that the veterinarian is not listening if a physical exam or other procedure is being conducted when he or she is speaking. In this case, reflective listening demonstrates to the client that the veterinarian is still listening despite doing another task simultaneously. Reflective listening helps to form a partnership with the client and can help direct the appointment. Examples of how reflective statements often begin are "If I heard you correctly . . ." or "It sounds like . . .".

Pick Up and Investigate Cues

Clients often provide verbal and nonverbal cues throughout the interaction, and it is important to notice these cues and explore them with the client.[1] Research has shown that doctors in primary care only respond to 21% of patient cues, while ignoring the rest. The same research also showed that when the cues are not investigated, patients brought up the same concern repeatedly; this clearly indicated that whereas the patient had not overtly stated a concern or feeling, there was an issue that needed exploration.[1] Actively investigating a client's cues by asking an open-ended question—or using a reflective listening statement— provides an opportunity for the client to share his/her thoughts and feelings openly and for the veterinarian to demonstrate his or her interest in the client's experience.[1]

Common Ground

In many instances, the client and the veterinarian begin the appointment with different perspectives; one of the most important steps in a successful and satisfying encounter is bridging the gap between veterinarian and client and finding common ground.[3] This process has three primary steps for the veterinarian and the client: (1) developing a shared understanding of the problem, (2) deciding on common goals for treatment, and (3) determining the role of each of them going forward.[3]

The ability to find common ground is contingent on an effective investigation of the client's perspective having taken place. The predominant themes of this phase of the interaction are negotiation and sharing of information and feelings. Two-way communication is essential in this part of a client interaction; the veterinarian and client must listen actively to each other, respond appropriately, and express themselves in a way the other can understand.[3]

Clients often come into an interaction with ideas regarding the diagnosis[3] or what the treatment should be on the basis of their own experience, Internet research, or the advice of friends. This can be challenging for veterinarians to manage if the clinical picture they see does not match the client's ideas. The work done earlier to explore the client's perspective can allow the veterinarian to address the discrepancies directly and tactfully. Although the veterinarian may want to simply provide information about their interpretation of the clinical picture and carry on, it is important to address the client's ideas and frame the information that the veterinarian believes is important in a way that is meaningful to the client.[3]

Defining the goals of the treatment is also essential.[3] In some situations, clients have unrealistic ideas of what can be done, for example, making a lame horse sound or an aging horse as vital as they once were. Open discussions as well as utilizing skills to involve the client and share information about the horse's condition can be useful methods to bridge the client's desires and what can be accomplished.[3]

Finally, the roles of each party, going forward, must be established.[3] The information gained earlier in the appointment can help the veterinarian formulate a plan that will be appropriate for the client. It can often be tempting to make assumptions about what the client will or will not be willing to do; the use of communication skills that promote client involvement and promote shared decision-making are valuable to ensure that the plans made are what the client truly wants.

If common ground cannot be found in the key areas described, the likelihood that the client will be satisfied and the patient will receive the care it needs is decreased. As discussed above, open-ended questions and reflective listening are important skills that aid in gaining an understanding of the client's perspective.[1] Skills that promote good two-way communication, client involvement, and

negotiation of mutually acceptable ideas are provided below.

Checking in

This skill is used to solicit the client's perspective, most commonly after information has been imparted or during planning of diagnostics and treatments.[1] A check-in can be performed with respect to a number of different things, for example, level of knowledge on a given topic, understanding of an explanation, and whether there are any questions. Check-ins can take the form of closed-ended questions, for example, "Do you have any questions?"; nonverbal communication, for example, pausing after relaying information; or, most effectively, an open-ended question. The use of an open-ended question will help the inquiry sound sincere and allow the client to answer freely, for example, "How does that sound?" or "Could you tell me what you know about founder?"

It is important to actively seek feedback from the clients because it engages them in their horse's care and can improve adherence to recommendations and treatment outcomes.[3] Furthermore, it can help prevent disagreements because the client is more involved throughout the process.[15] Checking in also helps prevent making incorrect assumptions about the horse and/or client. Often, the veterinarian is familiar with how their clients handle certain situations or with the decisions their client would make. However, it is critical to avoid assuming that because they did 'y' last time, that they will do 'y' again; veterinarians are bound to provide all the options every time. By checking in and saying, "Would you like to hear the options?" or "What can I tell you so you have the information to make this decision?" the client can decide for himself or herself what he or she wants or needs to hear. This skill is versatile and integral in seeking to actively engage the client in the process of their horse's care.

Avoid Jargon

The use of medical terminology can be intimidating to clients, and it can create a barrier between veterinarian and client.[1,3] Often, clients may not want to admit they do not understand and are therefore unwilling to ask questions. In these situations, the veterinarian may not know that the client has not understood; this can lead to negative outcomes such as poor adherence to recommendations or switching veterinarians. The use of terminology that is easily understood facilitates client engagement, helps to ensure their understanding, and promotes their involvement.[1,3]

Shared Decision-Making

Shared decision-making requires the use of several skills, including open-ended questions, checking in, sharing thinking, and involving the client.[1] It is also an approach that the veterinarian must embrace. All clients are different and have different levels of desired involvement. It is important to engage clients in the process and determine through open inquiry how comfortable they are with decisions being made.

Shared Thinking

"Thinking out loud" is an effective way to share information with a client in a non-intimidating, nonconfrontational way.[1] It promotes detailed and thorough information-giving to the client. It also provides the client with information about the veterinarian's thought process and it helps clients understand why certain things are being said or done, for example, "I want to make sure that his runny nose is not because of something going on in his lungs. So I'm going to put a bag on his nose that will make him take deep breaths and make it easier for me to hear his lung sounds clearly." Another benefit of sharing thinking is that it can reduce confusion regarding lines of questioning or suggestions, because the client has been given context for the veterinarian's inquiries,[1] for example, "I think she might have a joint infection, which can be caused by her not getting enough colostrum when she was born. So I'm wondering if you could tell me about what happened after she was born." Further, it allows them to follow the reasoning behind the veterinarian's suggestions and can eliminate some of the uncertainty or suspicion regarding the veterinarian's recommendations.[1]

Involving the Client

Many of the skills discussed previously promote the client's involvement in their horse's care. For instance, the use of open-ended questions requires the client to think before answering and provide more than a simple yes or no. A question as simple as, "What suggestions do you have?" or "How were you hoping I could help?" can be very powerful and elicit helpful information that can point to a useful path forward. It is of particular importance to involve the client when discussing diagnostic or treatment options, because this will facilitate the client feeling empowered and respected.[1] It will also strengthen the partnership between veterinarian and client. Providing clients with choices and allowing them to determine what suits their needs necessitates their involvement.[1] This does not mean that the veterinarian does not make recommendations or suggestions, but, through negotiation and the provision of information and choice, the client is involved in the process of their horse's care.

4. Relationship-Building Skills

Relationship building often occurs naturally as an appointment progresses and over the duration of care of the horse; however, given the importance of a strong veterinarian-client relationship, careful attention must be paid to this process. A good relationship improves the likelihood the client will feel comfortable sharing his or her perspective, facili-

tates the process of finding common ground, and promotes adherence to recommendations[1,3]; it also reduces the likelihood of conflict. In equine medicine, the relationships formed between veterinarian and client can last for the horse's lifetime. Often veterinarians find these relationships the most satisfying aspect of practice, and they can be major contributors to client loyalty and practice success. Investigating the client's perspective and finding common ground are important components of relationship building.[1,3] However, there are additional skills that can be used at any point in the interaction to directly facilitate and support the relationship.

Empathy

Empathy has two distinct phases: first, the understanding and appreciation of another person's situation or feelings; second, providing support by communicating that understanding back to the person.[1,2,16] In some situations, it may seem unnecessary to comment on the client's experiences or concerns, but an overt expression of understanding can be very meaningful. Empathy statements demonstrate genuine interest in the client's experience, build trust, and contribute positively to the veterinarian-client relationship.[1,2] Examples of empathy statements are "I can imagine this would be difficult for you," "I can see that this hasn't been an easy thing to deal with," and "It sounds like you've been really worried about this."

Nonverbal Communication

Nonverbal communication is an important and often overlooked aspect of any clinical interview. Nonverbal cues are sent through posture, proximity, touch, body movements, facial expression, eye movement, speed of speech, and vocal tone.[1,2] Making eye contact, nodding appropriately, and having an open body posture all indicate to the client that one is paying attention and following along with what he or she is saying, in contrast to looking away, crossing one's arms, and stepping back, which suggest disinterest. Often, verbal communication conveys conscious purposeful messages, and nonverbal communication portrays feelings and emotions.[1] Therefore, attending to the nonverbal signs of the client can provide valuable information about the client's attitudes, thoughts, and feelings, which can be relayed back to the client through a reflective statement or used to guide the next step of the interaction.[2] Being aware of one's own nonverbal cues and those of the client can enhance the quality of the interaction, provide more information, and help to form a stronger relationship.[1,2]

Self-Reflection

A critical component of strengthening veterinarian-client relationships, as well as learning and improving use of communication skills, is self-reflection.[2,17] It is rare that an external evaluation is provided of how well one communicates with a client or whether one's relationship with the client is good. Instead, this information must be gained by introspection and reflection of the interaction that took place. Clinically, the outcome of a case can be used to retrospectively evaluate an antibiotic choice or a rehabilitation protocol. A similar process can be performed for interactions with clients by reviewing the clinical encounter; considering what happened, what was said, and then reflecting on how certain aspects could have been handled differently. Some helpful questions to consider are: Did I understand what the client was telling me? Did I understand what the client wanted from me? Did the client understand what I said? Did the client get what he/she needed? Is the horse going to get the care it needs? Is the client satisfied?

Take Home Messages

- There is strong evidence to support the use of relationship-centered care as a model of veterinarian-client relationships in equine medicine.
- Clinical communication skills can ensure effective veterinarian-client communication and assist in the building of strong veterinarian-client relationships.
- A good relationship between veterinarian and client can improve loyalty, patient outcomes, and job satisfaction.
- There is value in being critical of how one interacts with one's clients and the relationships one has formed with them. Through focusing on investigating the client's perspective, finding common ground and relationship-building strong veterinarian-client relationships can be formed.

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How to Go Green by Adopting Sustainable Business Strategies in Your Practice

Barbara G. Crabbe, DVM

Setting up a recycling program at your veterinary practice requires a minimal amount of effort and offers many advantages including a valuable marketing strategy, team building, and the knowledge that you are "doing the right thing" by taking steps to reduce your carbon footprint. Author's address: PO Box 526, Beavercreek, OR 97004-0526; e-mail: bcrabbedvm@aol.com. © 2013 AAEP.

1. Introduction

In 2009, our equine practice made the commitment to "go green" by adopting an extensive recycling program. Living and working in Oregon, where protecting the environment is a priority for many residents, our initial goal was simply to "do the right thing" by taking steps to reduce our carbon footprint and protect habitats for both wildlife and future generations. We could never have anticipated the many other rewards this commitment would bring.

In 2010, we were recognized with a BRAG (Business Recycling Awards Group) award by our local county. Our practice was written up in the local media, and our clients began to recognize and appreciate us for our efforts. Not only has our recycling program become one of our best marketing tools to date, it has had the additional benefit of contributing to a strong sense of teamwork among our employees. All of our doctors, technicians, and support staff have taken a sense of ownership in the program and enthusiastically help with sorting, delivering, and otherwise participating in our group recycling efforts.

2. What Can You Recycle?

It has been estimated that 100,000 marine mammals and one million seabirds die each year from eating plastic. These facts make recycling of the many plastic products that pass through a veterinary practice worthwhile. We have found ways to recycle almost every piece of plastic we see, including plastic syringe casings, needle caps, and shaving bags.

Paper makes up 37% of the waste stream in the United States and is one of the easiest and most common products recycled. In most states, paper can be mixed in a single bin and often picked up as part of a curbside recycling program. In addition to recycling, paper waste can be significantly reduced by maximizing the use of computerized records.

Other products that are fairly easy to recycle include wood, metal, Styrofoam packaging, electronics, and print cartridges. The list is practically limitless, and we were surprised to learn how easy it was to locate places to recycle these different items. For example, all old electronic items (working or not) can be dropped off at your local Goodwill. Within the first 6 months of initiating our recycling plan, we have reduced the waste at our practice to a half can weekly, whereas we typically fill two large recycling bins.

3. Setting Up a Recycling Program

The first step to setting up a successful program in your practice is to identify a recycling coordinator. This individual should be someone who is already

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enthusiastic about recycling and protecting the environment and should be well organized. The recycling coordinator can then begin by researching what products can be recycled in your area and where they are accepted. A very helpful resource for gathering this information can be found at www.earth911.com. This website offers a recycling center locator that covers states nationwide. When beginning a program, it is wise to start with a limited list of items to be collected, such as paper, plastics, glass, aluminum cans, electronics, and inkjet/toner cartridges. You can always add to the items recycled as the program develops.

The next step is to determine how items will be collected and how they will be transported to a recycling facility. We have found it best to set up separate bins or collection areas to minimize the amount of sorting necessary before transport. For example, we collect all of our hard plastic syringe casings and needle caps in small containers placed inside our ambulatory vehicles and at strategic locations in our clinic. These small containers are dumped into bags when full and transported to the recycling facility. Plastic shavings bags are collected into large bags kept in the barn area and joined with other miscellaneous plastics collected in bins in the offices and treatment areas that are placed next to paper bins. Electronics and print cartridges are collected in boxes placed by printers in our business office. By having multiple collection areas in convenient locations, employees are encouraged to recycle rather than dispose of all recyclable items.

Transport to recycling facilities occurs in a variety of different ways and may require some creative thinking. Many commercial waste haulers offer recycling services in addition to basic garbage service, so your first step should be to contact your garbage company to find out what recyclable products they will pick up. We have several clients who have joined us in our efforts and will occasionally stop by to pick up items for return. In turn, we will occasionally collect items from these clients. Sharing these duties not only simplifies transport, it has helped us foster relationships with these clients who share our concern for the environment. Finally, our doctors will occasionally transport items in the practice vehicles when drop-off locations are convenient to routes taken during farm calls, and staff members willingly transport items on their way to and from work.

4. Outside the Box

In addition to our in-clinic recycling program, we have expanded our efforts to include equine-oriented events in our area with the formation of "Team Green." We have arranged to set up recycling bins at horse shows and have coordinated a group of junior exhibitors to collect, sort, and return aluminum cans, glass, and plastic bottles. In Oregon, these items can be redeemed for cash, and we have started a scholarship fund with the proceeds earned that is earmarked for training scholarships for the Team Green members. This program has given us a great deal of visibility in the equestrian community and resulted in a significant amount of additional recycling.

In 2012, we went one step beyond our recycling mission to join our county's "Adopt-A-Road" program and have committed to clean up and maintain a county road clear of trash and debris. As we look to the future, we look forward to discovering even more sustainable business strategies we can adopt to help us "go green."

Helpful Resources

www.earth911.com
Natural Resources Defense Council, www.nrdc.org
www.practicegreenhealth.com
Goodwill Industries: www.goodwill.org

How to Create and Maintain a Legally Defensible Medical Record to Protect Your Clinic

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In today's litigious society, it is more important than ever for veterinarians to practice defensive medicine. Equine practitioners have several unique issues that make it particularly problematic for them to practice defensive medicine. These issues include but are not limited to practicing ambulatory medicine, which may necessitate using owners and trainers for animal restraint, as well as having a direct relationship with trainers and farm managers but often not with the owners themselves. Other issues include horses with multiple owners—each with their own insurance company and their own financial and emotional interests—and the high monetary value of some of our patients.

The primary step in practicing preventative medicine is to prevent misunderstandings with owners and agents. This is achieved largely through clear communication. The responsible party must understand all the procedures that are going to be performed on their horses—or the horses in their care—as well as the associated risks, costs, and prognosis. Many lawsuits arise when what the veterinarian thinks they said is different from what the owners think they heard. The best way to avoid this issue is to make sure the owners and agents understand what is said, giving the owner time to ask questions and having a description and estimate of a given procedure in written form so that a responsible party can read, understand, and sign the document once they are satisfied with the discussion.

Medical records are your first line of defense. Every time you write in a medical record, you are writing a legal document that could be read in court and could potentially affect, or possibly end, your career. One procedural mistake in a medical record could put the entire record in question as to its accuracy or validity. It must be noted that many complaints made to the State Boards of Veterinary Medicine are dismissed after the state board reads a complete and accurate medical record. A quick review of the proper guidelines for medical records are as follows:

1) Write everything in blue or black ink and not in pencil or red ink. All entries should have the date and time noted and should be signed (or initialed) as they are put in the record. The record should be legible and should not contain any abbreviations that are not universally known, understood, and accepted.

2) There should not be any blank lines. If you skip a line, a lawyer may claim that you left a blank area in the medical record and then went back later to fill it in and did not use up all the space left blank. Additionally, one should not write in margins or

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below and above the lined areas. Again, this may encourage a lawyer to claim that entries were added to the medical record at a later time.

3) Anything that is changed should have a single line drawn through it. The reader should be able to read what has been crossed out. There should be a date, time, and signature along with an explanation as to why the item was crossed out. An example of this would be with "2/19/2012 written in wrong chart" written next to it and then signed or initialed.

4) Do not go back and add anything to the record on a previous line. If you forgot something put it on the next available line and write "addendum" at the beginning of that line and state the time that the item was performed.

5) Everything that is discussed with the owner, trainer, insurance company, referring veterinarian, consulting veterinarian, and so on, should be written in the chart. Even conversations that occur in remote locations should be added into the chart as soon as possible.

6) Additionally, all treatments and observations throughout the day should be listed. Mundane observations such as fecal production and water consumption can become important if a hospitalized horse has development of colic. Even in the absence of disease, these observations are evidence of your continued monitoring and care of your hospitalized patients. Certainly, all procedures and all physical exam findings should be placed in the record. Normal findings are important to record because they may protect you in the future. For example, if you do not record a horse's normal temperature, you cannot prove that it did not have a fever or that you even checked its temperature.

7) If consent is needed you should have the owner or agent sign the consent form. If this is not possible and consent is given over the phone, you should consider having two people gain consent from the owner. Both people should record it in the medical record. A faxed, signed consent from the owner can be added to the record until an original can be obtained.

8) On field calls, your physical examination should be recorded. An example is that when you look at a foal with contracted tendons you should not just record "bandages applied." You should explain why you are doing any procedure. In this case, for example, for treatment of bilateral front fetlock contracture. Detailed information is important. In this case, for example, if tetracycline was given, then record the amount that was given and if it was diluted in a liter of saline and given through a catheter.

9) If multiple treatment options are given to an owner, be sure to record each option, with all pertinent information such as projected costs and prognosis. You should record which option the owner chooses and, if possible, why the owner chose that option. This is particularly true if the owner or trainer declines referral. Make sure that you record that referral to a specialty center was recommended or offered. If you consult with a specialty clinic about any case, record the communications with that clinician.

10) We recommend having the owner/agent sign the farm call invoices to acknowledge that the treatment was administered.

Confidentiality is paramount in any medical profession. A simple way to anger a client is to disclose private medical information concerning their animal(s) to a third party. As a referral hospital, we routinely have performance animals in our barn. When a client is in the barn, he or she may recognize the animal and ask questions regarding its condition. Disclosing information could greatly affect an animal's value, result in significant problems with an owner, and potentially cause a lawsuit. We cover any names on halters with white tape. Records are kept in an area where only employees have access. It is very important to emphasize to your lay and professional staff that they should not disclose any medical information about a client's horse to anyone—this includes even confirming that the animal is present at the hospital. In high-profile cases, the client is consulted and dictates the amount of information that we can release, if any. Additionally, all employees have a confidentiality clause in their contract, and lay staff members sign a confidentiality agreement.

The most important thing to remember with regard to communication is to be honest with owners. Mistakes will occur, but that is not equivalent to malpractice. Open communication and explanation are critical. An example of this comes from orthopedic surgery cases: on occasion, drill bits or screws can break, and these issues should not be hidden from owners. Communication must be initiated, including prognosis, treatment necessity, and options. We referred a yearling Thoroughbred horse back to its original surgeon to remove a transphyseal screw that he had placed. There were issues with the placement of the screw, and we believed that the surgeon who placed the screw would be better prepared to remove it. After surgery, the horse was returned to the farm, with no problems reported. Four months later, while taking survey radiographs for the yearling sales repository, it was discovered that the screw had broken off, and half remained in the leg. This information could have been better handled if we had known about it before the sale.

Another example seen frequently involves the intra-arterial injection. As long as you have used normal accepted procedures when giving an intravenous injection, recognized the problem when it occurred, and treated it appropriately, malpractice is not an issue. This is a known complication and accepted risk. I often hear owners being told that their horse had an allergic reaction to a particular drug—not to an intra-arterial injection. This is an unnecessary lie that makes things more difficult

later and results in bad feelings with the owner when they eventually learn the truth.

In our hospital, we take a photograph of every horse both when it arrives and when it is discharged from the hospital. Any lesions or cuts that may be present are photographed. These photographs are date- and time-stamped and are added to the medical record. Digital photography is increasingly accessible with the advent of camera phones. It is relatively inexpensive as well. Photographing lesions and problems allows for objective evaluation of the progress of a case without relying on subjective memories, and the images are easily shared with owners who may not be the animal's primary caretaker.

We weigh each horse on admission and discharge. This allows for more accurate drug dosing and protects the hospital from owner complaints about lost weight while the horse was hospitalized. We use a digital scale, although a weight tape and calculation can be used. Additionally, body score and condition is noted on the admission physical examination.

With business and case communications being increasingly conducted through text messaging, text messages are becoming part of the legal and/or case record. There are several ways to print text messages with the dates, sender, and recipient recorded in the printout. Such methods include specific software programs that transfer data from a phone, to forwarding messages to an e-mail, or even taking photographs of the text messages. Phone calls are summarized in the records and are often followed up with an e-mail stating "to reiterate our conversation of such date and time…" followed by a synopsis of the conversation, which is also printed and added to the medical record. This practice is also beneficial in that it gives the client the opportunity to better understand what was discussed during the conversation and for disputing the validity of the conversation.

On occasion, communications by phone, text, or fax are disputed by the client. When a document is faxed to a client, we record the date and time the item was faxed. We also keep a binder with the printed logs from our fax machine showing all incoming and outgoing faxes. This prevented, in one case, a judgment being vacated because the client claimed we never faxed a letter to them. We were able to prove, in a matter if moments, that we, in fact, did fax the client and that the fax was received. In addition, phone bills can be used to prove a phone call or text message was made or received, although the content of the message cannot be proven.

Communications through the United States Postal Service are also often disputed by our clientele in legal cases. When important documents are mailed by United States Post Office First-Class Service, you can ask for a certificate of first-class mailing. This is simply a receipt with the recipient's name and address and the date that it was mailed. This costs less than a dollar and is significantly less expensive than sending certified mail. It is legal proof of mailing, although obviously it does not prove content. In the rare instance that proof of content is required, we have our office staff mail the document and then write a sworn statement stating the content of the mailed document. This statement is then notarized.

In some instances, owners will make complaints to the state board or file for malpractice when they are compelled to pay a bill. Often, they threaten to do so first; therefore these cases are not a surprise. At this time, you must decide if the benefit of bill collection is worth the time defending yourself to a court or state board. We always pursue payment in these cases. Although this may not always be a good financial decision, it is our business policy because we believe that it is important to defend our reputation and support any associate veterinarians who are named in the complaint.

It is important to know all the individual state laws and rules governing the practice of veterinary medicine. There are variations in laws even in neighboring states. For example, in Massachusetts, an owner must give informed written and witnessed consent before tranquilization of any animal. Therefore, before you sedate a horse for a dental or other procedure, you must have the owner sign a consent form and have it witnessed. A signed invoice after a procedure or the fact that the owner gave verbal consent and held the horse for the tranquilizer injection is not sufficient. They can still claim they did not give prior consent. This is different from the laws in neighboring New York State.

In today's world, social media outlets such as Facebook and list servers are becoming more and more prevalent. Remember: Anything that you put on the Internet can always come back to haunt you. Nothing should be written that cannot be viewed by clients or defended in court, even on a personal Facebook page. A list server recently featured a thread regarding the necessity of wearing sterile gloves for castrations. Several veterinarians commented that this practice was not necessary and was an unnecessary expense. Individual veterinarians may agree or disagree with this statement, and it should be noted that a percentage of castrations will become infected despite precautions. Additionally, owners and/or trainers should be told both in verbal and written form of the possibility of complications from castration, including infection, with each procedure. It would be counterproductive to a defense if an owner brought you to court for an infected castration and could provide written documentation that you did not wear gloves because of the expense.

With owner ability to research on the Internet, the idea of regional standard of care is becoming less prevalent to almost nonexistent. It is difficult to support a claim that you had no knowledge or understanding of a treatment being performed on the other side of the country. Internet searches and

list servers make information readily available to all clinicians, no matter where they are located. Additionally, common surgery such as umbilical herniorraphy in a young foal is often safely performed in the field. If no hospital is readily available in the geographical area, complications are easier to defend because regional standards of care would dictate a field procedure. However, if a hospital is available and a complication occurs (even a commonly accepted complication), you may have to defend your decision not to offer referral for the surgery. If you offered and the owner declined and you can provide documentation, no defense is needed. A certain percentage of horses will have a complication such as an infection or dehiscence; therefore, this issue may arise. This does not mean that every surgery must be referred—just that documentation is necessary.

If you have any suspicion that a malpractice suit or complaint may be filed, it is imperative that you notify your malpractice carrier immediately. Failure to inform a malpractice carrier in a timely manner may void your coverage. Even more importantly, they may have suggestions on how to proceed. The malpractice carriers and their attorneys have much more experience and the legal knowledge to better handle the case. They may instruct you to cease communications with the client and let the attorney handle all ongoing issues. They will also provide you guidelines for the information you should start gathering to provide the most successful outcome.

How to Protect Your Practice From a Digital Disaster: Create an E-Mail Policy

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1. Introduction

Computers are here to stay. That seems pretty obvious, with our growing reliance on the various digital and electronic devices that are required to do business. Along with the computers, we have also become Internet-dependent. Because the Internet is firmly planted as an integral part of our business process, digital communication has also become a major part of practice.

Currently, the primary business form of digital communication is still e-mail. This may change, but this use of the computer offers employees an efficient way to exchange information. The problem is that computers and the efficient transfer of data can get the unsuspecting business owner in trouble.

Employees can send out vital secrets, private patient information, or harassing messages to clients or to the public. They can also receive or download a whole host of problems in the form of malware, pornography, or illegal software.

Do your employees understand the use and potential abuse of the business computer? Can you search your business computers for potentially dangerous or libel e-mails without creating legal problems? Do you have an e-mail policy in place?

2. Discussion

An e-mail policy is critical in today's business environment of increased exchange of digital information. A clear and informative policy will not only offer you some protection but it will give your employees a chance to understand the limits and cautions required when they use business computers to transmit e-mails.

There are five basic policies to consider for your e-mail protocol. These include, but are not limited to, computer ownership, e-mail ownership, e-mail retention, e-mail privacy, and proper e-mail content (netiquette). There are other components that can be added for a more detailed and complete policy. These might include protocols for cell phone use, spam, downloads, networking, texting, and online security; however, if you have no policy in place, at least start with the basic five.

The first component of your e-mail policy is to make ownership and use of your computer system very clear. The point here is to make sure your staff is well aware that all components of your digital system are company property, including the software and the data. These electronics are for the primary use of doing business. If your company allows personal use of the computer system. then you should also establish clear guidelines for nonbusiness use.

Following the chain of ownership, the next policy establishes who actually owns the e-mails. The basic premise is that each and every e-mail generated from within the business e-mail system is the prop-

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erty of the business. If the e-mail system is based on a server within the business, most employees can understand that ownership. When the business e-mail system is based on a remote server such as Yahoo, Gmail, or AOL, some employees have difficulty understanding that the e-mails are the property of the business. This does not include the private e-mail accounts of your employees who reside on these remote servers, but it does include the individual accounts that are within your business e-mail system such as Outlook.

Clarifying the ownership status of the e-mails lends itself to the next component of your policy that addresses the expectation of e-mail privacy. Protection of your business demands the ability to access relevant and critical information, even if it is contained within an e-mail. This means that your employees should not expect their e-mails to be private. Of course, this does not mean that you are going to monitor their business e-mail accounts every day, but they should be aware that inspection is an option to the business.

Being able to review the e-mails of any employee in the business leads into the next policy that establishes the right of the business to purge the e-mail system. We all know how the numbers of e-mails have blossomed over the years. The sheer number can tax the memory volume of most storage devices. You can chose to buy more and more memory, or you can chose a more effective method and purge all e-mails on a regular basis. The time frame that seems to be the most effective for veterinary practices is 90 days.

This means that any relevant information that remains in the e-mail system will be deleted. Consequently, there should be an e-mail retention policy that offers clear guidelines for what information should be saved. E-mails that contain information related to medical records should be moved to the proper patient record. If the e-mail contains relevant business information, then the e-mail should be printed or digitally stored in the proper business folder.

However, be cautious. If there is a pending legal matter that encompasses digital communication, then the purging process must be suspended.

The final component to incorporate into your policy is a guideline for e-mail content. When business e-mails are sent, they represent the practice. This includes the practice culture and attitude. Having guidelines for how the e-mail is written is critical to maintaining your professional image.

Imagine that one of your clients is considering a sophisticated and expensive procedure. Then they get an e-mail from your office that is full of grammatical or spelling errors. This has the same image as performing a beautiful arthroscopic procedure on a knee only to send the horse home with a leg bandage that is sloppy and dirty. Established basic e-mail content protocols might prevent your best client from receiving a message that is all capital letters, the digital equivalent to screaming. Offering your employees e-mail content guidelines will help maintain professionalism.

3. Summary

In today's digital business environment, it is crucial to have clear and concise guidelines that inform your employees and protect your business. Once the e-mail protocols are established, make sure to incorporate them into your practice. The best way to ensure awareness is to insert the completed policy right into the employee handbook. A stand-alone policy document is also possible, but, in either situation, the contents should be reviewed with the staff and their exposure to the policy confirmed with a signature.

How to Create Engaging PowerPoint Presentations

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1. Introduction

There is little question that PowerPoint has revolutionized our ability to deliver presentations to all types of audiences in all sorts of venues, from the huge rooms and screens at American Association of Equine Practitioners to local Veterinary Medical Associations (VMA) groups at your neighborhood restaurant to client presentations in your practice area. Fortunately, PowerPoint is one of the most straightforward of the Microsoft products to use. With very little training, nearly anyone can create a slide deck in a short amount of time. The challenge lies in how to create a memorable PowerPoint presentation that effectively communicates your message in an interesting and engaging manner.

It is with some humility that I present this topic, because my presentations have admittedly put plenty of students and veterinarians to sleep over the years. At the same time, I am regularly complimented on my slides. The purpose of this presentation is to share my philosophy, techniques, and experiences to create PowerPoint slide decks that engage the audience. I freely admit that I am obsessed with PowerPoint and enjoy being creative with its many features, but one does not have to be similarly obsessed to improve one's ability to create more engaging presentations.

2. General Philosophy

Slides are important. We are visual people. Attractive and well-composed slides can make a tremendous difference in the effectiveness of any presentation. When I first began my academic career, I did not believe that I was a great presenter, so I knew that I had to make great slides. My goal was to make slides that people not only wanted to look at but wanted to stare at. I knew that there had to be motion and something attractive or interesting to see. Because attention spans are short, each slide should not stay on the screen for long. Although my techniques have evolved over the years, my philosophy was, and continues to be, that veterinarians are no different from toddlers transfixed on *Sesame Street,* in which the actors capture kids' attention with short, attractive, and seemingly simple skits. After 15 years of presenting, I have also learned the value of showing one's personality and telling at least one story. The latter will depend on the type of presentation and meeting, but, for the most part, everyone enjoys the mental break that a story provides.

3. Know Your Audience

This is one of the most important considerations for any speaker. For the most part, your target audi-

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ence should not be the smartest person in the room or the person with the most expertise in your area of focus or study. The most effective presenters are able to reach everyone in the room regardless of their previous level of knowledge or experience. Unfortunately, many speakers have become so focused on their specialty that they can no longer recall their baseline knowledge on the topic. Effective speakers ensure that they bring everyone up to speed, without being condescending, before launching into the complexities of their study or presentation. Doing so will engage everyone in the room and increase the reach of your message. In general, most speakers, especially those early in their career, overestimate the general knowledge level with the idea that "everyone knows that." One slide is often all it takes to bring everyone up to the same level. In so doing, you may even teach the other experts in the room a new way to present this seemingly basic information. There is little doubt that we all learn from each other during presentations, albeit at different levels.

4. Know Your Venue

The size of the venue and location of the screen(s) should affect the composition of your slides. In a small room in which attendees are sitting close to the screen, more information can be included on each slide. If the room is large and the screen relatively small, a presentation designed for a small room will be much less effective. This is especially true for meeting rooms that are long and narrow, in which people enter from the back of the room. This presents the most challenging situation for a speaker. There is often only a single, relatively small screen in the front of the room. In such cases, content is often very challenging to see by the majority of people in the room. In general, the larger the room, the more real estate your images and videos should occupy on each slide. Although this will result in a larger number of slides per presentation, this alone will not increase its duration.

In very large rooms, such as those used for the Annual American Association of Equine Practitioners Convention, multiple screens are often used. The presenter may not be able to see any of the screens, because they are often located in front of the presenter. This can be very disconcerting. For this reason, it is a great idea to explore the stage during a break to familiarize yourself with the layout of the room, stage, and screens. In such cases, a laser pointer may be ineffective to direct the viewer to a specific structure or lesion. Even if the presenter can use a laser pointer, it can only be directed to one screen at a time. Animated arrows can be used instead to direct the eye toward a lesion. Arrows will show up on all screens and will remain in place long enough for all viewers to be directed to the lesion or region of interest. It should also be mentioned that effective use of the laser pointer is a skill in itself. It is quite challenging to hold a laser still on a particular lesion, especially when nervous and/or over-caffeinated.

5. Know Your Projector

Many veterinarians have witnessed presentations with images that were so dark that they were unreadable on the screen, especially of imaging studies. Invariably, the presenter will often apologize profusely for the darkness (or brightness) of their images and mention how much better they project on their laptop. This clearly does not help the meeting attendee and can quickly become annoying if the presenter fixates on this problem throughout their presentation. I have been guilty of this and have learned to prevent this situation by testing the projector before my scheduled presentation time. If the images are too dark, the projector brightness can be easily adjusted by selecting the menu button on the projector and searching for the image or picture menu to find the brightness and contrast settings. With the exception of very large venues, the projector is almost always sitting on a table that is very accessible to you as the presenter. I routinely adjust brightness settings on the projector, even if the AV person discourages me from doing so. It only takes a few minutes and makes a huge difference in the viewer's experience. You have probably spent countless hours putting your presentation together. It only takes a few minutes to make these adjustments and it is time very well spent.

6. Number of Slides

Many presenters become fixated on a specific number of slides for a given presentation length. Whereas one slide per minute of presentation is sometimes used as a general guideline for many presenters, this is not set in stone and should not be relied on. I have given 20-minute presentations with 60 to 70 slides and 1-hour presentations with 40 slides. Much of this depends on the design of the room, which affects the amount of content on each slide and therefore the number of slides in a presentation. As mentioned above, more slides and less content per slide in large rooms will enable better viewing from far away.

Speaking time per slide is dependent on the amount of content discussed per slide and how much is actually stated. Although intuitive, many presenters crowd a slide with so much content in an effort to "reduce" slide numbers and to meet a predefined number of slides. In effect, this accomplishes little to reduce speaking time. By spreading the same content onto two or more slides, the information will become more visible and pleasing to the eye, without affecting the time to deliver the content.

7. Slide Design and Composition

There is nothing worse than sitting through an entire day watching presentations with a blue background and yellow or white text, yet this "guideline"

continues to be perpetuated as a standard for veterinary presentations. There are countless slide designs and color schemes available on PowerPoint, Microsoft Online, and other online sources. Additionally, color schemes can be easily modified within PowerPoint to suit individual tastes. It is recommended to test how some brightly colored schemes will project on the screen, because they may appear dramatically different than on your laptop screen. Presenters should also keep in mind that green and red, colors commonly used for arrows and other indicators, cannot be differentiated by those who are colorblind (8% of men). Given the current demographics of equine practitioners, this can be a problem for a percentage of attendees.

Text

Text should serve to guide both the presenter and the audience through each slide and should summarize the main points of the slide. Neither entire sentences (unless quotes) nor entire paragraphs should be included on slides. Too much text is overwhelming to the audience and prompts them to read and not listen to your message. Additionally, text should only be included on a slide if it will be discussed. Presenters should not assume that attendees will read text that is not presented. Font size should also be considered. In larger rooms, a larger font will improve visibility for attendees, especially in long, narrow rooms in which attendees tend to congregate in the back of the room near the door. In contrast, a very large font can be obnoxious and give the impression that you are "yelling" at the audience, similar to using all caps in e-mails.

Images

Images improve the viewing experience. My goal is generally to have at least one image per slide, ideally to support the concept being delivered on the slide but also for aesthetic purposes. Many presenters effectively use horse images of all types for this purpose. If these images are "borrowed" from the web or from others' presentations, the source should be revealed to represent the person who acquired that image. Copyrighted images should not be used without the owner's permission.

Images can be easily cropped within PowerPoint with the use of the crop tool under the Picture Tools menu. Photoshop is not necessary for this purpose. This is especially useful to crop identifying information on imaging studies to protect patient anonymity. This is important for all horses, but especially when a horse is recognizable at the local, national, or international level. Another advantage of using PowerPoint to crop images is that you can later uncrop the image on the slide to reveal the patient information. Image brightness, contrast, and multiple other settings can also be adjusted within PowerPoint. Increasing or decreasing the brightness can also be effective to alter how the images are projected on the screen. This is especially useful when a projector is out of reach and cannot be adjusted. For an even more polished look, the background of images can be removed to show only the horse or limb itself. This can easily be performed within the Mac version of PowerPoint but unfortunately cannot be performed with the Windows PowerPoint version. Non-Mac users can use Photoshop to remove the background, although this is much more cumbersome and time-consuming. Images should be saved as .png files to preserve the background transparency (Fig. 1).



Fig. 1. Before and after images show the effect of removing the background from the region of interest. This can be performed within PowerPoint if the Apple version is used but not if the Microsoft version is used. Although time-consuming, Photoshop can be used to create background transparency, as was performed in this image. The right image was saved as a.png file to preserve image transparency after insertion onto the slide. A drop-shadow effect was also added from the Picture Format drop-down menu within PowerPoint for added dimension.

General Composition

The composition of a slide should create a balanced and pleasing look. It is not necessary to select the exact slide format from the layout menu, because images and videos can be inserted into any slide layout, and text boxes can be readily altered in size. Text should not extend to the edges of slides, especially at the bottom, where it may be cut off when projected. Images should be large enough to be visible from the back of the room and generally should not be smaller than one fourth of the slide. A suggested rule of thumb is no more than two images per slide, because more than two images will often crowd the slide. If images are placed side by side, their size can be adjusted with the use of Picture Tools with PowerPoint to make them equal in size, when desired. Although images can be moved

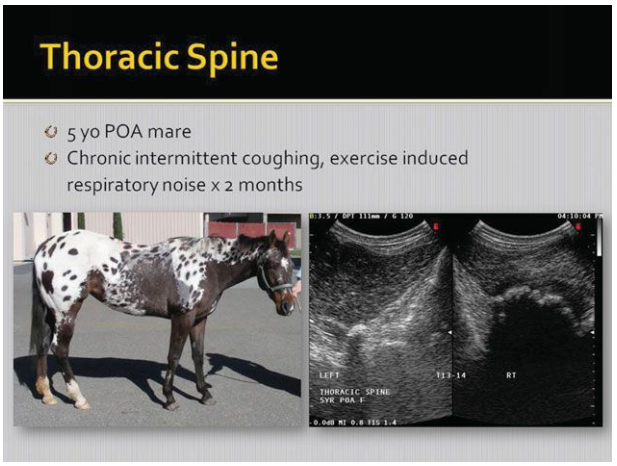


Fig. 2. Images placed toward the bottom of the slide will be challenging for viewers in the back of small rooms to see. This is especially true when using a floor mounted screen in a room with a flat floor.

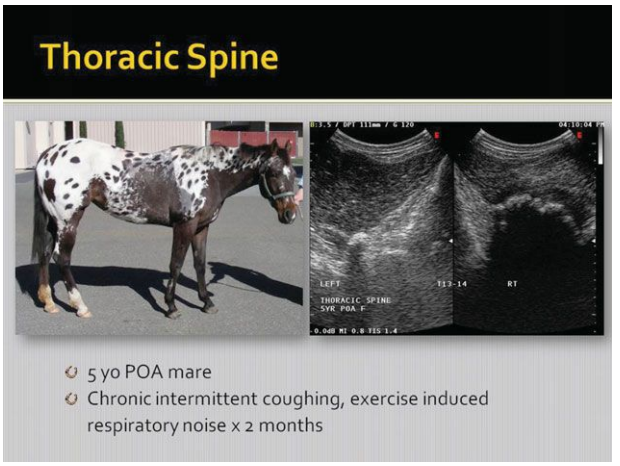


Fig. 3. This is the same slide as shown in Fig. 2, but the images have been moved toward the top of the slide to enable better visualization by all attendees, regardless of their location in a room.

anywhere on the slide, "nudging" with the up, down, right, or left arrows will allow images to be positioned exactly as desired.

When considering the position of text and images, many presenters tend to place text toward the top of the slide with images below the text and toward the bottom of the slide (Fig. 2). If the images are the primary focus of the slide and you are presenting in a small and flat room, people in the back of the room will find it difficult to see the images over others' heads. This is especially true if the projector screen cannot be raised very high. In such cases, it is recommended to place the images toward the top of the slide and the text near the bottom (Fig. 3).

8. Revisit Your Message

After all slides are made, it is recommended to step away from your individual slides and look at your presentation from a more global perspective. This is the time to revisit the message(s) that you hope everyone will take away from your presentation as they leave the room. Ask yourself if the presentation has effectively illustrated those messages. Especially with scientific presentations, it is easy to get lost in the details of the study or focus too much time on side tangents that your main conclusion(s) and take home messages do not come across in the presentation.

9. Effective Use of Animation

There are countless ways to animate slide content. I use animation extensively to lead myself and the viewer through each slide. It helps to keep me on track and prevents viewers from "reading ahead" and not focusing on the topic at hand. Several animation techniques will be demonstrated during the presentation. It is possible for animations to be overdone; some may believe that to be true for my slides. Over the years, however, I have reduced the variety of animation types used for text and images. My preference is "wipe from left" for text and "dissolve in" for images.

Arrows are useful to direct attention to lesions or anatomic structures on images, especially when animated with the use of "wipe" up, down, right, or left, depending on the desired direction. Arrows are very useful when a laser pointer is either challenging or impossible to use, as discussed above. This is especially true for novice presenters who may be quite nervous and shaky. It is often shocking to discover how much the laser shakes, which only makes the shaking worse. Presenters tend to counter this by making big sweeping movements on the slide that do not necessarily direct the viewer to any one location. Laser pointer stability can be improved by bracing the elbow or arm against the body to reduce shaking. Animation for videos are discussed below.

10. Videos

The ability to insert videos into slides has transformed presentations to allow seamless transitions from static images to movies, but nearly every presenter has a horror story about their videos not playing in a presentation. This should not deter their use, because the most common reason is a basic lack of understanding of PowerPoint's functionality with videos. Most importantly, one must understand that movies are not completely imported into a slide. For example, if you have inserted a movie named "Spot LF.mpeg" into the slide, you will see the first frame of the movie displayed on the slide itself. When you click on the movie to make it play, you are essentially asking PowerPoint to function as a movie player (similar to Windows Media Player, QuickTime, etc). PowerPoint will then

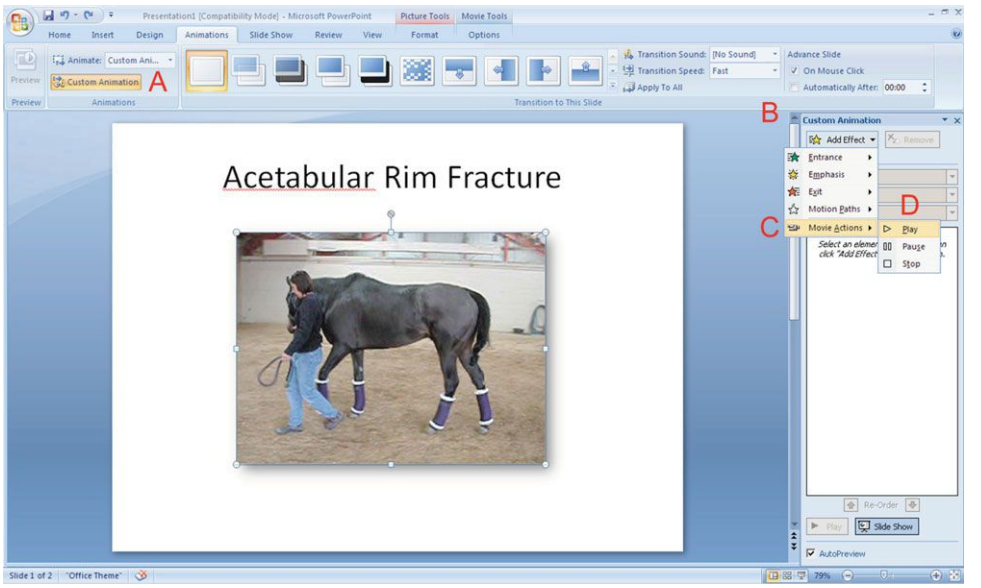


Fig. 4. Custom animation control of videos: From the "Animation" menu, click on "Custom Animation" (A) to bring up the Custom Animation menu seen to the right of the computer screen (B). Next, click on the movie itself to select it and to enable the "add effect" button in the Custom Animation menu at right. From the "add effect" drop-down menu, hover over "Movie Actions" to show its options (C) and select play (D). This will add the movie to the slide's animation scheme and allow the movie to be played without clicking directly on the movie.

"search" for that file name in the folder of the presentation and then play it on the slide. It is therefore critical that the movie be located in the same folder as the presentation, so that PowerPoint can find the file and play it. This is the most common reason why a movie will not play in a presentation. It is also important not to alter the movie file name after it has been inserted into the slide. For example, if we decided to change the name of the movie "Spot LF.mpeg" to "Rosie LF.mpeg," PowerPoint will continue to look for "Spot LF.mpeg" because that was the file initially inserted. Because PowerPoint can no longer find Spot LF.mpeg, the movie will not play. This can be fixed by deleting and then reinserting the movie with its new file name or by changing the file name back to its original name.

Video playing can be made even more seamless through the use of Custom Animation features for movies. I use this feature on nearly every video imported into presentations so that I can play a movie from anywhere in the room with a handheld clicker. It saves the steps of walking back to the podium, using the mouse to "wake up" the cursor, then moving the cursor to the movie, and finally clicking directly on the movie. This process is shown in Fig. 4. From the "Animation" menu, click on "Custom Animation" to bring up the Custom Animation menu on the right side of the computer screen. Next, click on the movie itself to select it and enable the "Add Effect" button in the Custom Animation menu. From the "Add Effect" drop-down menu, hover over "Movie Actions" to show its options and then select "play." This will add the movie to the slide's animation scheme, as shown in Fig. 5. By clicking on the drop-down menu associated with this file in the custom animation drop-down menu, several options become available to further modify how the movie is played. Although this may seem like several steps, it is quite rapid to learn and use and results in a seamless way to play movies within presentations.

Finally, it is important to realize that the PC version of PowerPoint does not allow insertion of QuickTime movies onto slides. QuickTime movies must be converted to another file type, such as mpeg and avi, and then added to the slide. Multiple video editing programs are available for this purpose. If the Mac version of PowerPoint is used, QuickTime movies can be imported and played within PowerPoint.

11. Slide Delivery

Some but not all seasoned presenters are able to project a single image on a slide and deliver a message that engages the audience without any prompting or notes whatsoever. For most presenters, that is not the case. Some speakers practice extensively until they have memorized their entire presentation. This may work well for first-time or novice presenters, but this is not time-efficient in the long run. Others will type out their entire presentation and read it directly from a script. When performed well, a presentation delivered in this fashion can come off as extremely well-polished, with attendees not realizing that they are being read to. When

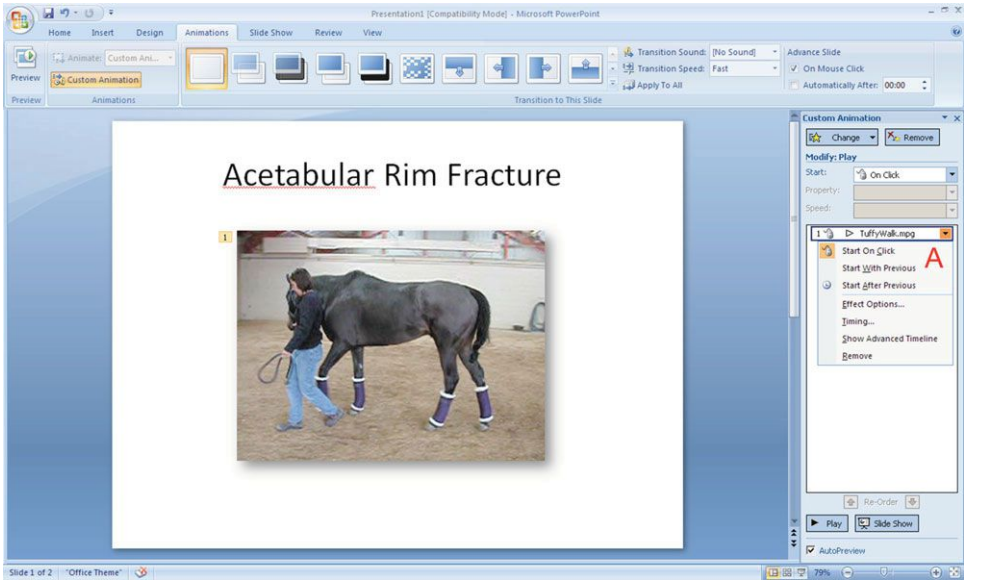


Fig. 5. Once the movie is added to the animation scheme of the slide (A), as shown in Fig. 4, several options can be accessed through the drop down menu, including when to start (on click, after previous, with previous), repeat, and many more.

performed poorly, it is obvious that the speaker is reading the entire presentation. Although this may be frowned on, it may be the only way to get some speakers through a one-time presentation. It should not be the method of choice on a regular basis.

Ideally, slides should be designed with bullet points that will trigger each speaking point or finding to be discussed. It is acceptable to read some slides, as long as this is not the strategy for presenting each and every slide. Whenever possible, a microphone should be used, unless presenting in a small room. Novice speakers are often reluctant to use a microphone, but most speakers do not have a strong enough voice to adequately project throughout an entire presentation. It is also important to use voice inflections and to pause to take a breath between each slide. Nervous presenters should resist the temptation to click to the next slide before they have finished speaking about the previous slide.

12. Dealing With the Sleeper

In a very large room, it is unlikely to notice a sleeping attendee, especially if bright lights are shining in your eyes. If you are in a smaller room, there is no doubt that eventually you will find a person sleeping during a presentation. It is important not to take this personally or as evidence that you are boring. Some people fall asleep as soon as the lights are dimmed. If this becomes too distracting, it helps to simply not look at them. It may also be helpful to make the audience "blurry" or to avoid looking directly at any one person. Alternatively, identify those who are actively engaged and make eye contact with them.

13. Non–PowerPoint Options

Keynote is Apple's presentation software program. Although Keynote offers some interesting effects, it may not be supported at large conferences in which presenters are required to upload a PowerPoint presentation. Additionally, Keynote presentations cannot be opened in PowerPoint unless they are first converted to PowerPoint, in which case, features unique to Keynote will no longer be present. For these reasons, Keynote has not gained widespread acceptance despite claims of superior utility by many Apple users. Another PowerPoint alternative is Prezi.com, an online resource to make presentations. Although this may be of interest for some, movements through each "slide" can be very distracting and somewhat disconcerting. Because the presentations are stored online, Internet access is required to deliver the presentation.

Review of Current Understanding of Pituitary Pars Intermedia Dysfunction

Dianne McFarlane, DVM, PhD, Diplomate ACVIM

Equine pituitary pars intermedia dysfunction (PPID) is a commonly diagnosed disease in the aged equine population. Recognition of PPID has increased considerably over the past two decades, in part because of an increase in client awareness of the condition as well as an increase in the number of aged horses receiving veterinary equine PPID care. This review focuses on the most current understanding of the development and progression of equine PPID. Author's address: Oklahoma State University, Department of Physiological Sciences, Center of Veterinary Health Sciences, Stillwater, OK 74078; e-mail: diannem@okstate.edu. © 2013 AAEP.

1. Introduction

Anatomy and Function of the Equine Pituitary

The equine pituitary gland lies ventral to the optic chiasm, separated from the brain by a fold of dura mater known as the diaphragma sellae. It is suspended from the hypothalamus by the infundibular stalk. The equine pituitary gland has four lobes; the pars distalis, pars intermedia, pars tuberalis (collectively known as the adenohypophysis), and pars nervosa (neurohypophysis).

Melanotropes of the pars intermedia produce a hormone precursor protein, pro-opiomelancortin (POMC), which undergoes extensive tissue-specific cleavage to yield adrenocorticotropic hormone (ACTH), melanocyte-stimulating hormones (MSH), β-endorphin, corticotrophin-like intermediate lope peptide (CLIP), lipotropins, and several other small peptides. In the pars intermedia of the healthy animal, the primary hormones produced are α-MSH, β-endorphin, and CLIP. Nearly all plasma ACTH originates from the pars distalis, and minimal ACTH is produced in the pars intermedia of the healthy horse.[1]

Equine pars intermedia activity has been shown to be inhibited by dopamine and stimulated by thyrotopin-releasing hormone.[2,3] The pars intermedia receives direct innervation from the dopaminergic neurons of the periventricular nucleus of the hypothalamus. These axons project through the infundibular stalk along the periphery of the pars nervosa, then travel into the pars intermedia, where they terminate on the endocrine cells of the pars intermedia, the melanotropes. Dopamine is released at the pars intermedia from the nerve terminals of the hypothalamic periventricular neurons that synapse directly on the melanotropes.[4] In the presence of dopamine secretion of pars intermedia, POMC-derived peptide hormones are decreased. If dopamine is removed either by surgically cutting the hypothalamic pituitary connection or by genetically deleting the dopamine receptor, melanotropes will proliferate, hypertrophy, and increase production of POMC-derived peptides.[5–7]

NOTES

The products of POMC are diverse and highly pleiotrophic in function. α-MSH has a role in metabolism, obesity, stress, and inflammation. Because of these critical functions, this hormone is currently the focus of significant research attention, with more than 1250 PubMed citations in the past 5 years alone. α-MSH induces an anorexic response and feeling of satiety[8] and has broad anti-inflammatory effects that include decreased production of a wide array of cytokines and other molecules, factors that contribute to inflammation.[9,10] α-MSH also impairs neutrophil function, including oxidative burst, chemotaxis, and adhesion.[11,12] CLIP is the cleavage product generated from the c-terminal portion of ACTH. Little is known about the function of CLIP; however, both CLIP and its cleavage product, β-cell tropin, have been shown to stimulate the release of insulin from rodent beta cells.[13] β-Endorphin is a potent endogenous opioid μ-receptor agonist that functions in analgesia and reduction of pain-associated inflammation.

Similar to several other species such as hamsters and sheep, activity of the pars intermedia in horses has a robust seasonal rhythm, with increased output occurring as day length shortens.[14–19] As a result, the plasma concentration of the pars intermedia hormones, including α-MSH, are greater in the autumn (August through October) than in the winter or spring.[18,19] It has been suggested that this adaptation helps to prepare the animal for the metabolic and nutritional pressures of the approaching winter.

2. Pituitary Pars Intermedia Dysfunction

Equine PPID is associated with increased size and activity of the pars intermedia. At necropsy, horses with PPID have enlarged pituitary glands caused by hyperplasia, hypertrophy, and a single large or multiple small adenomas. Enlargement of the pars intermedia is often accompanied by compression of adjacent structures. PPID was previously characterized as a benign neoplasia of the equine pituitary gland; however, clinical, pharmacological, biochemical, and histological data all indicate that PPID is a neurodegenerative disease with loss of inhibitory dopaminergic input to the pars intermedia. Typical of any neurodegenerative disease, age is the primary risk factor for PPID, and progression of clinical signs occur slowly, making early diagnosis problematic.

In horses with PPID, there is a marked reduction of dopamine in pars intermedia tissue[20] as well as a profound loss of dopaminergic periventricular nerve terminals and cell bodies.[21] Further evidence that PPID results from loss of dopamine is the improvement in clinical signs and plasma hormone concentration that is observed when horses with PPID are treated with a dopamine agonist such as pergolide.[22–24] Although the precise cause of neurodegeneration in PPID is unknown, several studies have provided evidence that oxidative damage occurs to the dopaminergic neurons, although at this time it is unknown whether oxidative stress is a cause or consequence of PPID.[25–27]

Neuronal accumulation and aggregation of misfolded proteins is a mechanism that contributes to the pathogenesis of most neurodegenerative diseases, including Parkinson's disease, a dopaminergic neurodegenerative disease of aged people. In Parkinson disease, the protein that accumulates in the dopaminergic neurons is α-synuclein. α-Synuclein is natively unfolded; however, under certain cellular conditions, α-synuclein can aggregate in dopaminergic nerve terminals, disrupting cellular function and triggering cell death.[28] Conditions that promote accumulation of α-synuclein include excessive concentration caused by increased production or decreased clearance, oxidation or nitration, and synuclein gene mutations.[29,30] α-Synuclein protein and gene expression was found to be increased in the pars intermedia of horses with PPID.[21] In addition to being more abundant, pars intermedia α-synuclein appears to be excessively nitrated in horses with PPID, a modification that promotes aggregation.[21] It is unknown if failure of protein clearance also contributes to α-synuclein accumulation in horses with PPID. Misfolded proteins are removed primarily through autophagy, the process by which damaged proteins or organelles are recycled by the lysosome.[31] Assessment of autophagy in the periventricular neurons of horses with PPID is ongoing.

3. Clinical Signs of PPID and Diagnostic Testing

Clinical signs of PPID probably are the result of overexpression of the pars intermedia hormones. Late in the disease, it is also possible that loss of hormones from the adjacent, compressed lobes of the pituitary may also contribute to the clinical syndrome. Weight loss caused by muscle atrophy, behavioral changes, secondary infections, and changes in haircoat are some of the most common signs of PPID. Laminitis occurs with PPID but less frequently than was originally suggested. It is likely that laminitis occurs only in horses with PPID and concurrent insulin dysregulation. Further work is ongoing to identify the mechanism of development of endocrinopathic lamintis.

Testing for PPID involves measurement of endogenous hormone concentrations, including ACTH or α-MSH or dynamic testing. Dynamic testing, which measures the response of the pars intermedia to stimulation or inhibition, may be a more discriminating approach to disease diagnosis. Because of the increase in PI activity in the fall, false-positive diagnostic test results for PPID are common when testing is performed during the autumn if reference intervals are not adjusted for season.[18–20] In addition, clinical signs of PPID are often more pronounced in the autumn, most notably an increased incidence of laminitis.[32] Because pasture composition also changes significantly with season,

studies are needed to determine the role of hormone increase in seasonal development of laminitis.[33] Diagnostic testing for PPID will be discussed in more detail in another session.

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Current Understanding of Equine Metabolic Syndrome

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Based on current evidence, elevated triglyceride concentrations and disturbances in insulin dynamics (fasting hyperinsulinemia and/or exaggerated insulin response to oral or intravenous glucose administration) are the most consistent features of the equine metabolic syndrome (EMS) phenotype. Whereas obesity and/or regional adiposity have been widely regarded as a primary feature of EMS, our recent findings suggest that obesity per se may not be a primary driver of the syndrome. Instead, obesity may be a marker of an underlying metabolic dysfunction that, depending on other environmental factors (eg, diet, level of physical activity), drives adipose tissue accretion and development of obesity. Thus, the presence or absence of obesity should not be used as a diagnostic criterion. Furthermore, although dietary restriction for correction of obesity remains an important management goal, the underlying metabolic dysfunction in affected animals may persist after weight loss. It is evident that further research is needed to better elucidate the EMS phenotype as well as its underlying pathophysiology. It is possible—even likely—that the syndrome that we now term EMS actually represents more than one condition. Thus, as new information becomes available, a change in the definition of EMS, even the name itself, may be warranted. Authors' addresses: Department of Large Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, 736 Wilson Road, East Lansing, MI 48824 (Geor); Department of Veterinary Population Medicine, College of Veterinary Medicine, University of Minnesota, 1365 Gortner Avenue, St Paul, MN, 55108 (McCue, Schultz); e-mail: geor@cvm.msu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Equine metabolic syndrome (EMS) refers to a cluster of clinical abnormalities associated with an increased risk of laminitis.[1,2] Although reports describing a phenotype that we now recognize as EMS were published as far back as the 1970s,[3] the term EMS was not introduced to the equine medicine vernacular until the early part of the last decade.[1] A number of different names have been used to describe this condition, including obesity-associated laminitis, peripheral Cushing's syndrome, and prelaminitic metabolic syndrome. The terms endocrinopathic laminitis and pasture-associated laminitis add to the confusion over nomenclature. Endocrinopathic laminitis refers to laminitis arising from hormonal rather than inflammatory conditions; by this definition, EMS-associated laminitis (and the laminitis that occurs in association with

NOTES

Table 1. Summary of Findings Related to Obesity, Regional Adiposity, and Endocrine/Metabolic Variables in Published Studies of the Equine Metabolic Syndrome Phenotype

	Treiber et al, 2006[5]*	Frank et al, 2006[8]	Bailey et al, 2008[6]	Carter et al, 2009[7]*
Breed(s)	Welsh/Dartmoor ponies	Six breeds	Mixed-breed ponies	Welsh/Dartmoor ponies
Sample size	160	12	80	74
Obesity (BCS) in EMS	Yes	Yes	No	Yes
Regional adiposity in EMS	Yes	Yes	No	Yes
Hyperinsulinemia in EMS	Yes (21.6 vs 10.7 mU/L)	Yes (50.5 vs 9.1 mU/L)	Yes† (69.5 vs 21.5 mU/L)	Yes (20.5 vs 8.8 mU/L)
Insulin resistance in EMS	Yes (RISQI)	Yes (CGIT)	Yes† (RISQI)	Yes (RISQI)
Fasting glucose	Not different	Higher in EMS (83.9 vs 66.9 mg/dL)	Not different	Not different
Triglycerides	Higher in EMS (97.2 vs 52.3 mg/dL)	Not different	Higher in EMS† (0.55 vs 0.38 mmol/L [49.0 vs 34.0 mg/dL])	Higher in EMS (53.0 vs 39.0 mg/dL)
NEFAs	Not different	Higher in EMS (366.5 vs 197.1 μmol/L	Not evaluated	Not evaluated

BCS indicates body condition score; RISQI, reciprocal of the square root of the serum insulin concentration; CGIT, combined glucose-insulin tolerance test; NEFAs, nonesterified fatty acids.

*Data obtained from the same population of Welsh and Dartmoor ponies.

†Serum insulin and triglyceride concentrations and RISQI differed between ponies with and without a history of laminitis in summer but not in winter.

pituitary pars intermedia dysfunction) can be regarded as endocrinopathic in origin. Similarly, pasture-associated laminitis has sometimes been used synonymously with EMS. This report describes current understanding of the clinical features of EMS as well as underlying pathophysiology. Other papers in this *In-Depth* series describe methods for diagnosis of EMS and aspects of nutritional and medical management.

2. Defining the EMS Phenotype

Johnson[1] recognized that primary features of a laminitis-prone phenotype (ie, obesity, insulin resistance) were analogous to those described for the metabolic syndrome (MetS) in humans, which is a constellation of abnormalities, including obesity, dyslipidemia, glucose intolerance, and hypertension associated with increased risk of cardiovascular disease and perhaps also diabetes mellitus.[4] The suggestion that the underlying pathophysiology of EMS is similar to that of MetS and the increased clinical recognition of "EMS" has spurred a number of observational and experimental studies in the past decade. Data emerging from these studies have provided some insight regarding features of the phenotype and its pathophysiology.

In a published consensus statement from the American College of Veterinary Internal Medicine (ACVIM),[2] the EMS phenotype was defined by the following criteria.

> (1) Generalized obesity and/or increased adiposity in specific locations (regional adiposity): An increase in the amount of fat surrounding the nuchal ligament ("cresty neck") is a common example of regional adiposity in affected animals but abnormal fat deposits also may be evident close to the tail head, behind the shoulder, or in the prepuce of the mammary gland region.
>
> (2) Insulin resistance (IR) characterized by hyperinsulinemia and/or abnormal glycemic and insulinemic responses to oral or intravenous (IV) glucose or insulin challenges.
>
> (3) A predisposition toward laminitis that develops in the absence of other recognized causes, such as grain overload, retained placenta, colitis, colic, or pleuropneumonia.

However, descriptions of the metabolic phenotype of laminitis-prone horses and ponies have varied among published studies (Table 1),[5–8] and the features that define EMS are a subject of ongoing debate in the equine veterinary community. What has become increasingly clear is that the relationships between the primary phenotypic features and key diagnostic measurements in EMS are complex—its phenotypic manifestation is highly influenced by environment; variation in measurements between individuals caused by breed, age, sex, and other factors make it difficult to establish clear diagnostic criteria; and not all components of the syndrome (eg, obesity) may be present in individuals with underlying metabolic derangements. In the following sections, we discuss these key criteria, the current scientific data that support/refute these criteria, and what is known regarding the pathophysiology of EMS.

3. IR and Hyperinsulinemia

Although our understanding of the etiology and pathophysiology of EMS is far from complete, it seems likely that IR and/or hyperinsulinemia play an important role. From a biological viewpoint, IR is usually defined in terms of the reduced ability of a given concentration of insulin to lower blood glucose levels. There has been much discussion about IR in the context of EMS; however, very few studies have reported quantitative data on insulin sensitivity and other aspects of glucose and insulin dynamics in affected animals.[8–10] Furthermore, the clinical definition and recognition of IR in equine medicine are not straightforward because "gold standard" methods for assessment of insulin sensitivity cannot be readily applied in clinical practice while simpler methods are either highly labile (eg, measurement of fasting insulin and glucose concentrations) or have not been thoroughly evaluated as valid surrogate indicators of insulin sensitivity (eg, fasting insulin; combined glucose-insulin tolerance test). The recognition of significant breed differences in insulin sensitivity, fasting insulin concentrations, and insulin responses during an oral sugar test (OST) further complicates development of a universal clinical definition of IR in horses.

The majority of reports have used "fasting" or "resting" measures of insulin and glucose and/or indices derived from these measurements (eg, the reciprocal of the square root of insulin concentration) as surrogate indicators of IR. Hyperinsulinemia has been shown to be a feature in the EMS phenotype in ponies[5–7] and Morgan horses,[8] although there is wide variation in values among studies that may in part be explained by differences in sampling conditions (eg, feed withholding versus pasture grazing before collection of blood samples). The current consensus view is that a fasting insulin concentration >20 mIU/L indicates IR. In a sample of 300 healthy, nonlaminitic horses in southwest Virginia, the prevalence of basal hyperinsulinemia (defined as insulin concentration >20 mIU/L) was 18%,[11] whereas another study in Australia reported a 28% prevalence of hyperinsulinemia (same cutoff value) in randomly selected ponies.[12] In the pony study, age, body condition score (BCS), supplementary feeding, and a history of laminitis were identified as risk factors for hyperinsulinemia.[12]

Fasting hyperinsulinemia is typically (but not always) accompanied by normoglycemia, which suggests compensated IR, that is, an increase in pancreatic insulin secretion occurs in response to reduced tissue insulin sensitivity, resulting in maintenance of glucose homeostasis. Two studies in ponies that used minimal model analysis of a frequently sampled glucose tolerance test have provided evidence of compensated IR, indicated by lower insulin sensitivity, higher acute insulin response to glucose administration (a measure of insulin secretion), and no difference in glucose tolerance when comparing laminitis-prone to nonlaminitic ponies.[9,10] In the early stages of type 2 diabetes of humans, hyperinsulinemia is also considered to be a compensatory response to IR. However, there also are some data suggesting that hyperinsulinemia per se can induce IR.[13] Thus, it may be questioned whether hyperinsulinemia in EMS is the "cart or the horse."

Mechanisms other than a compensatory increase in pancreatic secretion to counter tissue insensitivity may contribute to the fasting hyperinsulinemia and exaggerated insulinemic response to glucose administration observed in EMS animals. The plasma or serum concentration of insulin is primarily determined by its rate of secretion and clearance, with approximately 80% of endogenous insulin removed by the liver.[14] In humans and some animal species, there is evidence of decreased insulin clearance in insulin-resistant states (eg, diabetes mellitus, obesity, nonalcoholic fatty liver disease).[15,16] This reduction in insulin clearance is thought to be a mechanism to preserve β-cell function and also to maintain peripheral insulin levels in the face of IR.[16] One recent study in horses has shown that reduced insulin clearance contributes to higher blood insulin concentrations in obesity,[17] and it is also possible that decreased insulin clearance is a factor in the hyperinsulinemia observed in EMS animals.

Other hormones may contribute to an "upregulation" of insulin secretion in EMS, for example, the incretin hormones glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) which are secreted by the gut in response to nutrient cues. GLP-1 and GIP are labeled incretin hormones because they potentiate glucose-mediated insulin secretion and account for the higher insulin secretory response that is elicited by oral when compared with IV administration of glucose.[18] The insulinotropic action of GLP-1 was augmented in high-fat–fed obese and insulin-resistant mice compared with normal mice.[19] Studies in humans have yielded conflicting information regarding the effects of obesity and type 2 diabetes on incretin hormone secretion and the incretin effect.[20,21] One view, however, is that these conditions result in impairment in secretion and/or action of incretin hormones, and current studies are focused on the potential benefits of incretin-based therapy in type 2 diabetes.[20] At present, there is no published information on incretin hormone secretion or action in EMS.

There may be breed and/or genetic variation in insulin sensitivity and dynamics that affects susceptibility to EMS. Several review articles have raised the possibility of breed predisposition to EMS, with Welsh and Dartmoor ponies, Morgans, Tennessee Walking Horses (TWH), Saddlebreds, Arabian, and Paso Fino breeds thought to be more susceptible.[1,2,22] Affected horses and ponies appear to have high metabolic efficiency, meaning

that they require fewer calories for maintenance of body weight when compared with unaffected animals (ie, they are "easy keepers"). Ponies in general have lower insulin sensitivity[23] and a higher prevalence of hyperinsulinemia when compared with horses.[11,12] Given the observational[5–8] and experimental evidence[24,25] linking hyperinsulinemia to laminitis, it is possible that these inherent differences in insulin sensitivity and dynamics contribute to the higher clinical expression of EMS in ponies. Ponies also tend to consume more feed than horses when provided ad libitum access, which may contribute to exacerbation of hyperinsulinemia and risk of laminitis especially in animals maintained at pasture.

In human MetS, IR and/or hyperinsulinemia are thought to contribute to other components of the syndrome including dyslipidemia and vascular endothelial dysfunction that can lead to persistent hypertension.[26] Increased de novo hepatic lipogenesis is a feature of IR and hyperinsulinemia in humans and in animal models.[27] Activation of the mammalian target of rapamycin complex-1 and subsequent induction of sterol regulatory element-binding protein-1c, a master regulator of hepatic lipogenesis, is one possible mechanism for hyperlipidemia and increased hepatic fat accumulation in insulin-resistant states.[28] Increased plasma triglyceride concentrations were a feature of the EMS phenotype in a closed herd of Welsh ponies[5] and in outbred ponies with a history of recurrent laminitis,[6] although in the latter study the relative hypertriglyceridemia was evident during summer but not winter. An increase in nonesterified fatty acid (NEFA) delivery to the liver may be an alternative or additional factor underlying increased lipid concentrations.[29] In a small, mixed-breed group of obese, insulin-resistant horses, increased serum NEFA concentrations (but not triglycerides) were detected,[8] whereas serum NEFAs were not useful in the differentiation of an EMS phenotype in ponies,[5,7] and increased serum NEFA has not been a feature of the EMS phenotype in our recent study of >600 horses (below). In one report, increased very-low-density lipoprotein triglyceride concentrations were observed in horses with EMS.[8]

Insulin contributes to the regulation of vascular tone through effects on the synthesis and release of nitric oxide (NO; a vasodilator) and endothelin-1 (ET-1; a vasoconstrictor).[30] In other species, NO synthesis is impaired in IR, whereas hyperinsulinemia itself stimulates increased ET-1 production; the resultant imbalance between NO and ET-1 favors vasoconstriction and is thought to contribute to hypertension in insulin-resistant states such as MetS and type 2 diabetes mellitus.[31] Bailey et al[6] detected arterial hypertension in mixed-breed, recurrent laminitic ponies during summer but not in winter, whereas in our own studies, mean blood pressure did not differ between groups of EMS versus non-EMS ponies when measured in early spring, summer, and fall.[32] Further research is therefore needed to determine whether or not vascular endothelial dysfunction is a feature of EMS.

4. Obesity

Similar to the situation in human populations, the prevalence of obesity in horse and pony populations appears to be on the rise, and there is a growing concern regarding the adverse health effects of an expanded fat mass.[33] One definition of obesity in horses is a BCS ≥7/9 (Henneke scale), with recent studies showing that body fat represents ~20% to 25% of total mass in the animals with a BCS at or above this cutoff.[34,35] The prevalence of obesity in horse and pony populations, defined as a BCS ≥7/9, has varied between ~20% and ~50% in recent studies,[36,37] whereas obesity has been proposed as a risk factor for pasture-associated laminitis in ponies.[5,7] Certainly, obesity and/or the presence of one or more enlarged subcutaneous fat deposits (regional adiposity) have been regarded as defining characteristics of EMS.[2,22] Anecdotally, horses and ponies diagnosed with EMS may be obese and/or have regional fat accumulation in the nuchal ligament region (cresty neck), behind the shoulder (unilateral or bilateral), around the tail-head, and in the preputial or mammary gland regions. Occasionally, the presence of abnormal swelling around the prepuce or mammary glands caused by adipose tissue deposits and associated edema is the presenting complaint.[22] In published studies, high BCS and measures of apparent neck crest fat accumulation ("cresty neck score,"; neck circumference–to–height ratio, NCHR) have been associated with the EMS phenotype in horses and ponies[5,7,8]; however, obesity has not been a consistent finding across studies.[6] Additionally, definitions of obesity have varied among studies (eg, BCS >6/9[5] versus BCS >7/9[8]; Henneke scale).

Information from a recent study of >600 horses and ponies with (PL) and without (NL) a history of laminitis (previous 12 months) has provided further insight into the EMS phenotype, including the association with obesity.[38] The data collected included morphometric measurements and indices (eg, BCS, NCHR, girth-to-height ratio), fasting glucose, insulin, triglyceride and leptin concentrations, and glucose and insulin concentrations 75 min after an oral sugar challenge (OST; 15 ml corn syrup per 100 kg BW). The predominant breeds sampled were Morgan horses, TWH, Arabians, and Welsh ponies. Across breeds, fasting insulin concentration, insulin 75 minutes after corn syrup administration, and fasting serum triglycerides were the variables most consistently elevated in animals with a history of laminitis. This observation confirms previous results in ponies[5,6] but, in contrast to another report,[8] demonstrates that elevated serum triglycerides might also be a key feature of the EMS phenotype in horses. Unlike some previous reports, morphometric measures (NCHR, etc) and BCS did not discriminate between the PL and NL groups. Prior weight

loss in PL animals may have confounded this assessment, although it is noteworthy that within the PL cohort metabolic variables (insulin, triglycerides, etc) did not differ between obese (BCS ≥7) and nonobese (BCS <7) animals, suggesting that the metabolic derangement persists even after weight loss. Within the NL cohort, however, serum triglycerides, fasting insulin, and post-OST insulin were higher in obese when compared with nonobese animals. One interpretation of these findings is that obesity per se is not a requisite feature of the laminitis-prone (ie, EMS) phenotype, but components of the phenotype such as IR may be exacerbated when affected animals become obese.[38] This interpretation is consistent with anecdotal clinical observations of the laminitis-prone phenotype—not all affected animals express obesity or regional adiposity, whereas, conversely, not all obese animals appear to be at increased risk of laminitis. Simply put, obesity in and of itself does not equate with EMS.

In humans, too, it is now recognized that the presence of obesity-related metabolic disturbances, including IR, hyperlipidemia, inflammation, and hypertension, varies widely among obese individuals.[39,40] In fact, in some studies, up to 30% to 35% of obese adult humans have a metabolically healthy phenotype, giving rise to the term "metabolically healthy obese."[39] On the other hand, up to 40% of adults of normal weight and body mass index have metabolic perturbations typically associated with obesity and MetS, such as dyslipidemia, hyperinsulinemia, and nonalcoholic fatty liver disease.[39,40] Although the mechanisms underlying these different phenotypes are not fully understood, it is currently believed that differences in visceral fat accumulation and the response of adipocytes to increased lipid accumulation are important factors.[39] Adipose tissue produces a large array of proteins, including pro-inflammatory cytokines and other hormone-like proteins termed adipokines that exert local (paracrine) and systemic (endocrine) effects.[41] Studies in animal models have indicated that obesity results in a progressive dysregulation of adipose tissue function, including marked pro-inflammatory signaling that leads to the development of a systemic inflammatory response, which, in turn, results in the development of IR and other metabolic abnormalities.[41,42] However, not all excess fat carries equal risk with visceral adiposity most strongly associated with systemic inflammation and IR, perhaps because of higher cytokine and adipokine production as well as release of NEFAs into the portal circulation that contribute to the development of hepatic IR.[42]

Our preliminary observations that obesity per se is not a defining feature of EMS mirrors findings in humans—IR and hypertriglyceridemia can occur in nonobese horses, whereas, conversely, the metabolic profile of some obese horses does not differ from that in lean individuals. One hypothesis is that obesity is not a primary cause of EMS but instead it may be a marker of an underlying metabolic dysfunction, which, depending on other environmental factors (eg, diet, level of physical activity), drives adipose tissue accretion and development of obesity. If so, there are two very important implications for the identification and management of EMS. First, the presence or absence of obesity cannot be used as a diagnostic criterion. Second, whereas dietary restriction and weight loss may result in some improvement in insulin sensitivity and so forth in affected animals, the underlying metabolic dysfunction is likely to persist. Thus, a diagnosis of EMS may be justified in some horses and ponies with recurrent laminitis problems that are not obese (or show regional adiposity) because they have other components of the phenotype, for example, hyperinsulinemia or high serum triglycerides. Then again, a diagnosis of EMS is not appropriate in horses or ponies that are obese (BCS ≥7) but without evidence of laminitis or disturbances in insulin dynamics and lipid metabolism.

Further studies are needed to examine the impact of obesity on the metabolic health of horses and its role in EMS. Cross-sectional studies have shown an inverse relationship between BCS and insulin sensitivity[43] as well as positive relationships between apparent adiposity, resting insulin concentrations, and blood markers of inflammation (eg, serum amyloid A, tumor necrosis factor (TNF)-α, mRNA encoding for interleukin (IL)-1β, and TNF-α.[43,44] On the other hand, adipose tissue (nuchal crest fat) and systemic markers of inflammation were unchanged in Arabian geldings after a 20% weight gain (increase in mean BCS [Henneke scale] from 6/9 to 8/9).[a] Additionally, insulin sensitivity was unchanged in Thoroughbred geldings after an ~15% increase in body weight,[45] whereas another recent study of horses and ponies reported no change in insulin sensitivity after an ~20% in increase in fat mass.[46] These observations reinforce the idea that factors other than BCS and adipose tissue mass contribute to variance in insulin sensitivity.

Research is also needed to examine the role of the adipokines leptin and adiponectin in EMS. Leptin, the product of the *ob* gene, provides information to the brain regarding the availability of body fat stores, promoting satiety and a reduction in food intake when energy balance is positive or fat stores are plentiful.[47] Circulating leptin concentrations are generally in direct proportion to body adipose mass.[41,47] In contrast, serum adiponectin concentrations generally correlate inversely with fat mass, although in humans, hyperinsulinemia is associated with hypoadiponectinemia independent of fat mass.[41] It has been proposed that high leptin and low adiponectin concentrations are useful markers of IR and EMS.[22] Certainly, high leptin concentrations have been observed in insulin-resistant horses and ponies,[7,8] including nonobese animals. In our own studies, however, serum leptin was not useful for the identification of laminitis-

prone animals, although, consistent with previous studies, leptin concentrations were strongly associated with BCS. Similarly, an inverse relationship between blood adiponectin (total or high-molecular-weight forms) concentrations and BCS has been reported in horses.[48,49]

5. Laminitis

A current or recent episode of laminitis is the most common reason for a horse or pony to be evaluated for EMS.[2,22] The severity of EMS-associated laminitis is variable, ranging from subclinical (ie, the presence of divergent growth rings on the hoof wall but not overt lameness) to mild lameness that is only detected when horses move across a hard or uneven surface, to more severe lameness and other classical signs of laminitis. Laminitis tends to be recurrent in affected animals.[22] The results of informal surveys of practitioners support the impression that the majority of laminitis cases in practice are secondary to EMS,[50] and clinical observations suggest that EMS horses or ponies often develop laminitis when grazing at pasture (pasture-associated laminitis), especially under conditions that favor increased accumulation of water-soluble carbohydrates (WSC) in pasture forages, for example, spring and early summer, or after substantial rainfall in the summer or fall.[5,51] It has been suggested that EMS-associated laminitis begins as a seasonal problem, with episodes coincident with changes in pasture WSC content, but then becomes a year-round problem as the disease progresses.[52,53] In our own studies, incident laminitis in horses or ponies with suspected EMS was associated with pasture grazing in ~55% of cases.[38] EMS-associated laminitis, therefore, also appears to occur in circumstances not associated with exposure to pasture or other changes in diet composition. Another hypothesis is that animals with an EMS phenotype are more susceptible to sepsis-associated laminitis (ie, colitis, colic, Gram-negative sepsis), although data in support of this notion are lacking. There is an obvious need for data from well-designed epidemiological studies, such as the ongoing American Association of Equine Practitioners Foundation–supported case-control study of pasture- and endocrinopathy-associated laminitis, to determine the prevalence of EMS-associated laminitis and to provide evidence-based information on risk factors such as season, access to pasture, metabolic phenotype, and concurrent disease.

The discovery that prolonged hyperinsulinemia can induce laminitis in healthy horses and ponies may have important implications for understanding of EMS- and pasture-associated laminitis, providing a potential explanation for episodes of disease after consumption of feeds and forages that elicit pronounced insulinemic responses. Studies of grazing horses have shown a positive relationship between pasture NSC content and circulating insulin concentrations,[54] and marked exacerbation of hyperinsulinemia has been observed in ponies with an EMS phenotype when they are grazing spring pasture (NSC ~15–18% DM); moreover, the hyperinsulinemia coincided with episodes of laminitis in the EMS ponies.[5,32] Whether or not hyperinsulinemia plays an essential or exclusive role in the development of EMS-associated laminitis remains to be determined. Clinical observations have indicated that not all animals with profound and persistent hyperinsulinemia develop clinical laminitis, whereas, conversely, some animals with an EMS phenotype are not hyperinsulinemic during episodes of laminitis. Circulating insulin concentrations can vary markedly in EMS animals; therefore detection of an association between hyperinsulinemia and incident laminitis is challenging. Nevertheless, it also must be considered that other mechanisms may contribute to the development of laminitis in EMS.

The association between grazing NSC-rich pasture and incident laminitis in EMS animals raises the possibility of hindgut carbohydrate overload as a triggering mechanism. If a chronic inflammatory state is a component of EMS, then affected animals may be more susceptible to inflammatory injury of the lamellar tissues in the face of carbohydrate overload, which, in the experimental starch and oligofructose models,[53] elicits marked systemic and laminar inflammatory responses. In a recent study of insulin-resistant ponies, however, the feeding of a high NSC diet for 7 days that mimicked exposure to spring pasture had no effect on laminar pro-inflammatory signaling but did result in exacerbation of hyperinsulinemia.[55]

Vascular endothelial dysfunction caused by IR and/or hyperinsulinemia also may affect susceptibility to laminitis in EMS.[53] It is noteworthy that in healthy horses and ponies, an infusion of insulin that induces marked hyperinsulinemia (~1000 mU/L) causes an increase in hoof wall temperature that suggests digital vasodilation.[25] On the other hand, prolonged exposure of equine vascular tissue to high concentrations of insulin in vitro resulted in deterioration of vasodilatory responses.[56] More research is therefore needed to elucidate the interrelationships between IR, hyperinsulinemia, and digital hemodynamics in the context of laminitis susceptibility.

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Diagnosis and Medical Management of Endocrine Disorders in Aged Horses

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Aged horses are at risk for pituitary pars intermedia dysfunction (PPID), and insulin dysregulation (hyperinsulinemia and insulin resistance) is an underlying problem in some animals. Hyperinsulinemia and insulin resistance are exacerbated by PPID, and this increases the risk of laminitis. It is therefore recommended that all aged horses be screened for PPID and hyperinsulinemia and the magnitude of these problems be assessed. Endocrine disorders can be successfully managed in aged horses through appropriate husbandry and medical treatment. Author's address: Department of Clinical Sciences, Tufts Cummings School of Veterinary Medicine, North Grafton, MA 01536 and School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington, United Kingdom; e-mail: Nicholas.Frank@tufts.edu. © 2013 AAEP.

1. Introduction

Pituitary pars intermedia dysfunction (PPID) is the most important endocrine disorder of aged horses, with a prevalence rate of 21% recently reported for horses more than 15 years of age.[1] Hyperinsulinemia and insulin resistance (IR), which can be collectively referred to as insulin dysregulation, are also concerns in aged horses and can occur concurrently with PPID. Hyperinsulinemia and IR are components of equine metabolic syndrome (EMS) and can remain an underlying problem for the life of the horse or pony. It is likely that EMS has a genetic basis, with the phenotype expressed in the younger animal if obesity is allowed to develop and/or later in life when PPID exacerbates the underlying condition. Equids with PPID can therefore either have lower insulin concentrations and normal insulin sensitivity or hyperinsulinemia and IR, and this can only be determined through diagnostic testing. Testing is highly recommended because hyperinsulinemia and laminitis are associated in equids, with evidence provided by epidemiological studies[2,3] and experimental models.[4,5] In this presentation, diagnostic testing for PPID, hyperinsulinemia, and IR are reviewed as well as medical treatments for these endocrine disorders.

2. Diagnostic Testing for PPID

When selecting a diagnostic test for PPID, the first question to ask is whether early or advanced disease is suspected. If the horse has advanced PPID, the disorder can be diagnosed on the basis of clinical signs alone. Hirsutism had 71% sensitivity and 95% specificity as a diagnostic test for PPID in horses with postmortem evidence of pituitary disease.[6] Although hirsutism is considered pathognomonic for PPID in aged horses, care should be taken to consider the differential diagnoses of chronic systemic disease and malnutrition. Advanced PPID prolongs the anagen phase of hair growth and increases hair length, and the long haircoat of affected horses is better referred to as hypertrichosis rather

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than hirsutism.[7] Although advanced PPID can be diagnosed on the basis of clinical examination alone, it is still advisable to measure adrenocorticotropic hormone (ACTH), glucose, and insulin concentrations before making treatment and diet recommendations.

Early PPID is more challenging to diagnose, and there are two tiers of testing. A plasma ACTH concentration can be measured as a screening test for early PPID.[8] This is the simplest approach to diagnosing PPID, but clinicians are cautioned that some horses with early disease have normal results. To perform this test, a blood sample is drawn and submitted for measurement of ACTH. Plasma ACTH concentrations increase in the late summer and autumn[9,10]; therefore season-specific reference ranges have been established. It is also important to consider the assay used by the laboratory to measure ACTH and the familiarity of laboratory personnel with equine samples.

Blood is collected by means of jugular venipuncture into plastic tubes containing ethylenediaminetetraacetic acid. Tubes should be placed in a cooler with ice packs or refrigerated and centrifuged the same morning or afternoon of collection.

Interpretation: From November to July, a plasma ACTH concentration >35 pg/mL by chemiluminescent assay confirms the diagnosis of PPID. During the August to October period, an ACTH concentration >100 pg/mL strongly indicates that the horse has PPID, whereas a concentration of 50 to 100 pg/mL is a weak indication of disease. If clinical signs of PPID are present, this equivocal result should be interpreted as positive, and treatment is recommended. However, treatment should not be initiated in horses with ACTH concentrations within this range if there is no historical or clinical evidence of PPID. These horses should be monitored and rechecked in 3 to 6 months.

Because hormone concentrations naturally increase in the late summer and autumn, this time of year should be selected so that the physiological alterations serve as a natural stimulation test. Plasma ACTH concentrations increase in healthy horses during this period, and even higher peaks are detected in those with PPID. It should be noted that this is a new recommendation and reflects a shift in our approach to diagnosing PPID.

The thyrotropin-releasing hormone (TRH) stimulation test [11,12] is the second tier test for detecting early PPID. This test was developed by Dr Jill Beech at the University of Pennsylvania and is thought to be the most sensitive diagnostic test for PPID available at present. Although it is referred to as a second-tier test, it is also appropriate to perform this test as a first-tier approach. This is likely to become the recommended approach in the future because protirelin (synthetic TRH) is made available to practitioners by pharmacies. Testing is not recommended during the August-to-October period at present because season-specific cutoff values have not been established.

Fasting is not required for this test. A baseline (time = 0) blood sample is collected, and 1.0 mg (total dose) TRH is then administered IV as a bolus. A second blood sample is then collected 30 minutes later. Blood samples are handled as described above, and plasma is submitted for the measurement of ACTH concentrations.

Interpretation

- Baseline ACTH concentrations are interpreted as described above.
- Results are negative if ACTH concentration is <35 pg/mL (chemiluminescent assay) at 30 minutes.

There is a strong indication of PPID if the ACTH concentration is >75 pg/mL at 30 minutes when testing is performed from November through July. There is a weak indication of PPID if the ACTH concentration is between 35 and 75 pg/mL at 30 minutes when testing is performed from November through July.

- If accompanied by clinical signs, treatment is recommended.
- If there is no historical or clinical evidence of PPID, testing is repeated after 3 to 6 months.

Some clinicians prefer to collect the second blood sample at 10 minutes and interpret the results (November through July) as follows: Negative if <85 pg/mL, weak indication if 85 to 100 pg/mL, and strong indication if >100 pg/mL. Research to establish reference intervals is ongoing.

3. Diagnostic Testing for Hyperinsulinemia

Testing for hyperinsulinemia is recommended for all aged horses with clinical signs of endocrine disorders or laminitis. The author also recommends screening horses for hyperinsulinemia as part of routine health checks in the same way that humans are screened for hypercholesterolemia. High-risk populations for hyperinsulinemia include pony, Morgan horse, Paso Fino, and Arabian breed groups, as well as any horse with generalized obesity or obvious regional adiposity. Aged horses are placed in the high-risk category if PPID is developing or there is a history of EMS.

Fasting insulin concentrations were previously recommended as the diagnostic test for hyperinsulinemia in horses,[13] but this recommendation has changed recently. When fasting insulin concentrations are measured, a cutoff value of 20 μU/mL is recommended for the general population if the radioimmunoassay is used. Breed-specific reference ranges are also being developed. The glucose concentration should be measured in the same sample to detect hyperglycemia, and diabetes mellitus has

been associated with PPID in aged horses.[14] Fasting conditions are achieved by leaving only one flake of hay with the horse after 10 PM and drawing blood the next morning, or feeding as normal in the morning and then collecting the blood sample after 4 PM.

An oral sugar test (OST) has been introduced to better assess postprandial hyperinsulinemia in equids,[15] and this test detects insulin dysregulation in horses with normal fasting insulin concentrations. The test is performed by fasting the horse as described above and then administering corn syrup[a] at a dosage of 0.15 mL/kg PO (75 mL for a 500-kg horse). Syrup is given by mouth with the use of 60-mL catheter-tip syringes, and blood samples are collected for glucose and insulin measurements 60 and 90 minutes later. The owner can administer the corn syrup before the veterinarian arrives.

Interpretation

- Results are normal if the insulin concentration is <45 μU/mL (radioimmunoassay) at 60 and 90 minutes.
- Results indicate hyperinsulinemia if the insulin concentration is >60 μU/mL at 60 or 90 minutes.
- Results are considered equivocal if the insulin concentration is 45 to 60 μU/mL at 60 or 90 minutes. Testing is repeated at a later time, or the combined glucose-insulin test should be considered.
- Results indicate an excessive glucose response if the glucose concentration is >125 mg/dL at 60 or 90 minutes.

Owners sometimes express concern about administering corn syrup to horses with suspected hyperinsulinemia or laminitis. Problems have not been encountered in the author's experience, but a two-step approach can be adopted if necessary. The first step is to measure the fasting insulin concentration. If the concentration is >20 μU/mL, then this result provides evidence of insulin dysregulation and further diagnostic testing is optional. It is still helpful to perform an OST in a horse with modest hyperinsulinemia to assess insulin responses to oral sugars and to make diet recommendations, but this is not essential. If the fasting insulin concentration is <20 μU/mL, then the OST is recommended to complete the evaluation, and the owner can be reassured that the test is safe to perform in their horse.

Resting triglyceride and leptin concentrations can provide additional information in the management of endocrine disorders in aged horses. Hypertriglyceridemia has been identified as a predictor of laminitis risk in ponies, with cutoff values of 57 and 94 mg/dL established from two studies of the same population.[2,3] A lower cutoff value of 27 mg/dL has been proposed for horses by McCue et al[16] after reviewing data from an EMS genetics study, and breed-specific reference ranges may be forthcoming. Leptin concentrations can be measured for diagnostic purposes if blood samples are sent to the Cornell Animal Health Diagnostic Laboratory (Ithaca, New York). When horses of approximately the same body condition score were compared, horses with high leptin concentrations (>12 ng/mL) had significantly lower insulin sensitivity than those with normal leptin concentrations (<2 ng/mL).[14] Higher-than-normal leptin concentrations (>4 ng/mL) indicate that adipose tissues are abnormal and secreting excessive amounts of leptin.

Other markers of metabolic dysregulation may be incorporated into diagnostic panels in the future. Wooldridge et al[17] recently validated an ELISA for high-molecular-weight adiponectin and reported concentrations of 3.6 ± 3.9 μg/mL (mean ± SD) and 8.0 ± 4.6 μg/mL for obese and lean horses, respectively. Serum amyloid A [18] and ferritin[19] concentrations are also associated with obesity and insulin resistance and may provide additional evidence of metabolic dysregulation.

4. Medical Management of PPID

Pergolide mesylate[b] is administered to horses with PPID to restore dopaminergic inhibition of melanotrophs. This ergot alkaloid drug binds to D2 receptors and inhibits proopiomelanocortin hormone synthesis.[20] Pergolide is prescribed at a starting dose of 0.002 mg/kg (1 mg for a 500-kg horse), and the dosage range extends to 0.01 mg/kg (5 mg for a 500-kg horse). Approximately 30% of horses exhibit anorexia when treatment is initiated, and this problem can be avoided by starting with half of the dose for the first 2 days.

Cyproheptadine can be administered in combination with pergolide at a dosage of 0.25 mg/kg PO q 12 hours.[8] When these drugs were compared as single treatments, pergolide was more effective. Seventeen of 20 horses (85%) with PPID improved after pergolide treatment, compared with only two of seven horses treated with cyproheptadine.[20] Cyproheptadine antagonizes serotonin, which is thought to be a stimulatory neurotransmitter for pars intermedia melanotrophs, and mild sedation is an occasional side effect of treatment. The author considers cyproheptadine therapy as an additional treatment when the horse reaches a pergolide dosage of 0.006 mg/kg (3 mg/d for a 500-kg horse).

The goals of medically managing PPID in an aged horse depend on disease severity. In early PPID, the pergolide dosage should be adjusted until plasma ACTH concentrations return to reference range, with adjustments after 30 days. However, this goal cannot be attained in some advanced cases. Clinical signs are important to monitor in both situations, and the primary goal when treating advanced PPID is to achieve a clinical response. This response can sometimes be seen when pergolide is administered at a lower dosage over a long period of time, so even owners with financial limitations

should be encouraged to provide treatment. Ideally, the dosage of pergolide should be increased in advanced PPID cases until ACTH concentrations exhibit a downward shift, which indicates a response, even if concentrations remain above reference range. Treatment of PPID is particularly important when glucocorticoid-mediated IR exacerbates underlying hyperinsulinemia. Insulin concentrations decrease and insulin sensitivity improves with better control of PPID, and this may lower the risk of laminitis.

5. Dietary Management of PPID

Fasting insulin concentrations and OST results are normal in some horses with PPID and abnormal in others. This is consistent with the theory that PPID exacerbates hyperinsulinemia, and most horses improve with pergolide treatment. However, care must still be taken to provide the appropriate diet if hyperinsulinemia persists. A diet composed of hay, low-sugar/low-starch pellets, and vegetable oil (one-half to 1 cup twice daily) should be selected, and pasture access should be limited. In contrast, horses with PPID that do not have problems with postprandial hyperinsulinemia can be provided with senior feeds and free access to pasture.

6. Medical Management of Insulin Dysregulation

Before a management plan is formulated, it must first be determined whether known exacerbating factors for hyperinsulinemia and IR, including obesity, high-sugar feeds, and PPID, are present.

Relationships between obesity and insulin sensitivity in equids are not as straightforward as they might seem. Body fat mass is negatively correlated with insulin sensitivity in equids,[21] and yet some obese horses have profound hyperinsulinemia whereas others have normal insulin values. Genetics are likely to play an important role in determining the magnitude of insulin dysregulation associated with obesity. If it is accepted that insulin dysregulation is genetically determined, then obesity is better viewed as an exacerbating factor; this is supported by the finding that hyperinsulinemia and IR are detected in nonobese horses. In the author's experience, insulin values improve with weight loss in most obese horses with hyperinsulinemia. Whether obesity is a cause or an exacerbating factor for hyperinsulinemia, there are other health risks associated with this problem, and horses should be maintained in appropriate body condition. Equine obesity is a major concern in the United States, with a study of horses in Virginia revealing prevalence rates of 32% for the overconditioned state and 19% for obesity.[22] Although owners are sometimes concerned about older horses losing condition as they age, obesity should not be promoted through overfeeding. Aged horses that are obese should be placed on a weight reduction diet. A concept of weight loss resistance has been proposed to describe the marked individual variability in responses to dietary interventions,[23] and this is a familiar situation for veterinarians managing obesity in equids.

Pituitary pars intermedia dysfunction is also an exacerbating factor for hyperinsulinemia and IR and should be controlled with pergolide treatment. Some affected horses have a history of obesity but have lost body fat mass and only retain regional adiposity within the neck and tailhead regions. Others are still obese when PPID first begins, and we must rely on subtle signs of pituitary dysfunction, including delayed shedding of the winter haircoat.

There are two indications for pharmacological intervention in the management of insulin dysregulation in aged horses. The first is the acceleration of weight loss in obese animals and the second is management of postprandial hyperinsulinemia after exacerbating factors have been managed.

7. Levothyroxine Sodium

Levothyroxine accelerates weight loss in horses that are placed on a controlled diet, and this is accompanied by increases in insulin sensitivity.[24–26] Pretreatment with levothyroxine for 14 days also prevented healthy horses from developing IR after endotoxin infusion.[27] Levothyroxine is administered at an approximate dosage of 0.1 mg/kg, which is rounded to 48 mg/d for horses weighing 450 to 525 kg. A mild state of hyperthyroidism is induced and sustained for 3 to 6 months until body fat mass decreases. When levothyroxine is discontinued, the dosage should be lowered to 0.05 mg/kg for 1 week and then 0.025 mg/kg for a second week.

8. Metformin Hydrochloride

Because the oral bioavailability of the available metformin formulation is low (7.1 $\pm$ 1.5% in fasted horses),[28] it has been questioned whether the established effects of this drug on insulin sensitivity in humans occur in horses. Additional information was recently provided to resolve this question when Durham et al[29] demonstrated that metformin (30 mg/kg) given orally 30 minutes before an oral glucose tolerance test significantly lowered glucose and insulin concentrations. These findings suggest that metformin can limit postprandial hyperinsulinemia even if its effects on insulin sensitivity are weak. The current recommendation is therefore to administer metformin at a dosage of 30 mg/kg, given 30 to 60 minutes before feeding. Metformin is available as 1-gram tablets and is well tolerated with the exception of oral irritation in some horses.

9. Ethical Considerations

The author consults on study design for Boehringer Ingelheim Vetmedica, Inc.

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[a]Karo Light; ACH Food Companies, Inc, Cordova, TN 38016.
[b]Prascend; Boehringer Ingelheim Vetmedica, Inc, St Joseph, MO 64506.

Dietary Management of Endocrine Disorders in the Older Horse

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Control of ration quantity and composition is important in the management of horses with pituitary pars intermedia dysfunction (PPID) or equine metabolic syndrome (EMS) that exhibit obesity and/or hyperinsulinemia. In obesity, restriction of daily dry matter intake to 1.25% to 1.5% of current body weight will facilitate weight loss and improvement in insulin sensitivity in most horses, although the rate of response will vary between animals and more severe restriction (dry matter intake 1.0% body weight) may be needed. Access to pasture grazing should be limited during management of obesity (eg, use of grazing muzzles). Insulin resistance and hyperinsulinemia may persist after weight loss, dictating the need for long-term restriction of dietary nonstructural carbohydrates (<10–12% dry matter), including restricted pasture grazing. Author's address: Department of Large Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, 736 Wilson Road, East Lansing, MI 48824; e-mail: geor@cvm.msu.edu. © 2013 AAEP.

1. Introduction

The proportion of the horse population that can be regarded as "older" or "aged" appears to have increased in recent years. One retrospective study reported that in 1989 only approximately 2% of the equine referral cases at a university veterinary hospital were over 20 years of age, but this had increased to 12.5% by 1999[1] and approximately 20% by 2003.[2] In a survey of the horse population in the United States performed in 2005, 7.6% of the total population was reported to be over 20 years old.[3] A more recent study in the United Kingdom reported that 30% of horses were aged 15 years or older,[4,5] with 11% between 20 and 30 years and 2% over 30 years of age.[5] An increased willingness of owners to maintain older horses together with improved health care and nutrition may account for this apparent increase in prevalence of old horses.

The two most common endocrine disorders of horses and ponies are pituitary pars intermedia dysfunction (PPID; *aka* equine Cushing's disease) and equine metabolic syndrome (EMS).[6,7] PPID is more prevalent in aged horses; in general, affected animals are >15 years of age when clinical signs are first recognized, and the risk of PPID increases with age.[6] In EMS, the age of onset is less well defined, although a recent consensus statement indicated that the syndrome is often initially detected in animals between 5 and 15 years of age.[7] A primary clinical concern with both PPID and EMS is laminitis, hence the term *endocrinopathic laminitis*. The true prevalence of laminitis in horses with PPID is unknown, although some authors suggested that it may be as high as 50% to 80%,[8,9] whereas by definition, EMS animals are predisposed to laminitis, especially when kept at pasture.[7] The mechanisms underlying endocrinopathic laminitis have

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not been fully elucidated, but insulin resistance (IR) and hyperinsulinemia are thought to be contributing factors. IR and/or evidence of abnormal insulin dynamics is a defining feature of EMS. As well, hyperinsulinemia can be detected in PPID, and, in one study, a serum insulin concentration of >188 mU/L was a poor prognostic indicator for 1- to 2-year survival in affected horses.[10] Obesity and/or regional adiposity are additional factors that may directly or indirectly affect the risk of endocrinopathic laminitis. First, increased adiposity may underlie or exacerbate IR; second, dysregulated adipose tissue in obesity may release proinflammatory cytokines and adipokines that contribute to lamellar injury; and third, increased weight-bearing with obesity may increase strain on the digital dermal-epidermal interface. A major goal in the nutritional management of EMS and PPID, therefore, is to minimize the risk of laminitis through dietary strategies that correct obesity, enhance insulin sensitivity, and avoid exacerbations of hyperinsulinemia. Other age-related problems with nutritional implications, such as poor dentition and reduced digestive efficiency, must also be considered in the development of a feeding program for the older horse with PPID or EMS.

This report briefly reviews some of the nutritional implications of aging and then focuses on dietary management of obesity, IR, and hyperinsulinemia in the context of PPID and EMS. Note that some horses with PPID are not insulin-resistant or hyperinsulinemic; these animals can be fed according to nutritional recommendations for older horses.

2. Selected Aspects of Aging in Horses

Digestive Function

Early studies reported decreased digestive capacity in aged (>20 years of age) horses[11] with reduced apparent digestion of fiber, phosphorus, and protein. However, a subsequent study did not support these findings,[12] and it has been suggested that chronic parasitic scarring of the large intestine contributed to the apparent reduction in digestive capacity observed in earlier studies.[11] The reported decrease in fiber digestion also might have been caused in part by dental abnormalities, because dentition was not assessed in early studies of older horses. However, common dental abnormalities such as small points or hooks (<3 mm in size) do not appear to affect nutrient digestibility. On the other hand, extremely poor dentition can impair feed intake and contribute to weight loss. Severe dental problems in the older horse (eg, wave mouth, loss of several molar teeth) can make it necessary to provide a "no long-stem fiber" diet or a commercial "senior feed." Commercial senior feeds can be blended with other fiber sources (eg, alfalfa cubes, hay pellets, and soaked sugar beet pulp) to ensure adequate fiber intake.

Overall, current evidence suggests that advanced age (>20 years) alone does not significantly affect digestive efficiency in horses. Many healthy old horses that are provided adequate internal parasite control and dental care can maintain good body condition and nutritional status when they are fed rations that provide calories and nutrients in quantities that meet or exceed the recommended amounts.[13]

Body Condition

Weight loss or difficulty in maintaining adequate body condition is a common complaint in old horses. In one study, 17% of older horses were reported to lose weight within the 12-month period before examination.[14] Weight loss may arise for many reasons, such as dental abnormalities, renal and hepatic disease, or PPID. Cold climatic conditions also may contribute to weight loss; it has been suggested that old horses are less tolerant to temperature extremes. Ralston et al[15] reported that horses over 20 years of age required higher feed intake during winter months to maintain body condition when compared with younger horses kept in the same environment. Temperatures below the lower critical temperature (LCT) will increase caloric needs regardless of age.[13,16] Although the LCT may be affected by several factors (eg, acclimatization), in northern temperate climates the LCT is estimated to be −1ºC to −5ºC for horses kept outdoors. As a general guideline, caloric intake should be increased by 2.5% for each degree Celsius below the LCT. Most importantly, body condition should be monitored carefully during prolonged bouts of cold weather and the amount of feed should be adjusted according to condition loss or gain.

Pro-Inflammatory State and IR

Recent studies have reported that aged horses, like humans, show evidence of a pro-inflammatory state (ie, "inflamm-aging") that may contribute to the development of age-associated IR and other morbidities (eg, osteoarthritis).[17–19] In one study, old horses (>20 years) had higher levels of inflammatory interleukins (blood mRNA for interleukin [IL]-1, IL-15, IL-18, and tumor necrosis factor [TNFα]) and increased frequency of interferon gamma (IFNγ)- and TNFα-producing cells in the circulation when compared with horses <10 years of age.[18] Additionally, obesity appeared to exacerbate the age-related increase in inflammatory cytokine production by blood mononuclear cells.[19] Reduction of body weight and body condition in overweight old horses significantly reduced the percentage of IFNγ- and TNFα-positive lymphocytes and monocytes as well as circulating concentrations of TNFα.[19]

In humans, it is well documented that advancing age is associated with a decline in insulin action, with a 40% to 60% reduction in insulin sensitivity reported in healthy older adults (>55–60 years)

compared with younger adults.[20,21] Malinowski et al[22] observed a markedly higher insulinemic response to oral glucose administration in old Standardbred mares (>25 years of age) compared with middle-aged (~15 years) and young (~7 years) Standardbred mares, suggesting that aging in horses also results in a decline in insulin action. The insulinemic response to dietary starch and sugars is therefore likely to be more pronounced in older horses and may justify the feeding of a diet with restricted starch and sugar, regardless of EMS or PPID status.

3. Management of Obesity

As discussed elsewhere in these proceedings, not all horses with EMS are obese; however, dietary restriction, in combination with increased physical activity, is indicated for overweight or obese horses (body condition score [BCS] ≥7/9) because the obesity is a likely contributor to IR and possibly other metabolic abnormalities. Obesity and/or regional adiposity are sometimes evident in PPID although, more commonly, weight loss is reported by owners and clinical examination reveals moderate-to-thin BCS with poor muscle mass.[6]

"Eating less" and "exercising more" are key strategies to achieve a more ideal body weight (BW) and condition.[23] An evaluation of the current feeding program and housing is the starting point for any weight loss program—what feed is currently being provided (including supplementary feed, hay, pasture quality, and time allowed for grazing) and in what quantities? Proximate analysis of feeds and forage will enable estimation of true digestible energy (DE) and nutrient intake, but simply weighing the ration and using "book" or manufacturer-supplied nutrient values for each component will provide reasonable estimates of feed, energy, and nutrient intake. Maintenance energy requirements for horses typically range between 30 and 35 kcal DE per kg BW per day, for example, 15.0 to 17.5 Mcal DE/d for a 500-kg horse[13]; the low end of this range has been used to estimate maintenance needs of "easy keeper" horses or ponies with a tendency to be overweight or obese.

It is important to set realistic goals for weight loss. Consistent with anecdotal clinical observations, recent studies have demonstrated considerable between-animal variation in the rate of weight loss when obese (BCS ≥7/9) horses and ponies were subjected to dietary restriction. Specifically, weekly weight losses of 0.16% to 0.55% of BW (relative to BW at the end of the first week of restriction) were observed when animals were provided daily dry matter intake (DMI) at 1.25% of BW for 16 weeks.[24] Thus, a target weight loss of up to 0.4% to 0.5% per week is reasonable, but it is important for owners to be aware of the between-animal variation in response to identical dietary restriction. The following guidelines are recommended for the development of a weight loss program.

- The diet should be based on forage or a forage substitute. In most situations, grain and manufactured, calorie-dense feeds should be eliminated from the diet (eg, commercial sweet feeds, feeds containing added fats). Excessive feeding of other "treats" such as carrots and apples also should be curtailed.
- Early-maturity hay with a high leaf-to-stem ratio should be replaced by later-maturity hay that typically has lower energy content (no more than 2 Mcal per kg DM). When mature grass hays are fed, intake of vitamin E, copper, zinc, and other minerals may not meet requirements, and provision of a vitamin-mineral supplement is therefore recommended. Many feed companies market low calorie "ration balancer" feeds for this purpose. In addition to vitamins and minerals, these products contain sources of high-quality protein and are usually designed to be fed in small quantities (eg, 0.5 kg/d, fed as is or mixed with hay chop). Alternatively, forage-based, low-calorie feeds that contain added vitamins and minerals are now available commercially. This type of feed offers convenience and may be used as a substitute for hay or fed as a component of the ration along with hay.
- The amount of forage and feed provided will depend on the severity of obesity as well as the previous level of feeding, but in general weight loss is induced when energy intake is restricted to 70% to 75% of requirements at current body weight. On the basis of recently published work, a restriction of DMI to approximately 1.25% of BW daily results in clinically safe and effective rates of weight loss as well as improvements in indices of IR.[24,25] The author recommends initially providing no more than 1.5% of current BW in feed and forage (DM basis, noting that most forages and feeds are approximately 85–90% DM), that is, 3.7 kg for a 250-kg pony and 7.5 kg for a 500-kg horse. If there has been minimal weight loss after 6 to 8 weeks, daily DMI should be decreased to 1.25% BW for a further 6- to 8-week period. Some animals may require more severe restriction to 1.0% BW as daily DMI. The feeding of lower amounts of feed (<1.0% BW as DMI) is not recommended.
- The substitution of straw for up to 50% of the hay ration may be considered. This is one way to lower the energy density diet and maintain a reasonable level of DMI. On average, the energy content of straw is lower than that of grass hay. The straw should be clean and contain minimal cereal head; thoroughly shaking the straw before feeding will remove any loose cereal.
- The ration should be divided into three or four feedings per day. Strategies to prolong feed

intake time should also be considered, such as the use of hay nets with multiple small holes.

- Access to pasture grazing should be restricted. It is preferable to maintain turnout, either in a large dry lot or at pasture with the horse wearing a "grazing" muzzle that restricts but does not eliminate grazing. In a recent study of ponies provided 3 hours of access to autumn pasture, the application of a grazing muzzle reduced DMI by approximately 80% when compared with no muzzle.[26] Turnout into a large dry lot is another way to restrict grazing while still encouraging exercise. It should be noted that simply restricting the time allowed for grazing may not be an effective strategy for weight loss. Ponies have been observed to consume up to 1% of BW as DM within 3 hours of pasture turnout.[26]
- Make all dietary changes gradually, and avoid prolonged periods of feed withholding. Abrupt starvation in obese ponies, donkeys, and miniature horses (especially pregnant animals) carries the risk of hyperlipemia and is not recommended. In addition, severe dietary restriction (daily DMI <1.0% BW) may increase the risk of gastric ulceration and stereotypic behaviors.
- Develop, and continually update, an appropriate weight maintenance program once the target weight and body condition have been achieved. This will include monthly assessment of BW and BCS to ensure that the feeding program is appropriate to the current level of physical activity and other environmental influences on energy requirements (eg, ambient conditions).

The BCS system is useful for estimation of subcutaneous fat mass in equids but may not be a sensitive indicator of weight loss, at least during the early phase. In two recent studies, changes in BCS were poorly correlated with changes in BW during weight loss programs.[24,27] On the other hand, significant decreases in heart and belly (at the level of the umbilicus) girth measurements as well as the depth of the retroperitoneal (ventro-abdominal) fat depot was detected during weight loss. It is therefore recommended that in addition to BCS, heart and belly girth (and, if feasible, ultrasound fat depth) be recorded. These measures should be recorded at monthly intervals during the weight loss program and every 6 to 8 weeks during weight maintenance.

4. Countering IR/Hyperinsulinemia

In obese animals, weight loss can be accompanied by improvement in insulin sensitivity.[24,25] However, IR and exaggerated insulin responses to consumption of feeds rich in nonstructural carbohydrates (NSC; starches, sugars, and/or fructans) can persist after weight loss. Furthermore, some animals at initial diagnosis of EMS or PPID are not obese but do have evidence of IR and abnormal insulin dynamics. Marked postprandial increases in circulating insulin are a concern because of the potential for high insulin concentrations to cause laminitis.[28,29] Restriction in dietary NSC is therefore important in the long-term management of EMS and PPID animals with IR. Key approaches are listed below.

(1) No grain or sweet feeds (ie, feedstuffs rich in starch and/or sugars). The starch content of oats, barley, and corn are approximately 45% to 50%, 60% to 65%, and 65% to 70% of DM, respectively. Sweet feeds contain grains plus molasses, and the NSC content of some of these feeds can exceed 40% to 45% DM. Provision of these feeds to insulin-resistant equids is likely to exacerbate hyperinsulinemia.

(2) Restricted or no access to pasture. At certain times of the year, pasture forage NSC content may approach 30% DM or more. In susceptible animals, ingestion of this NSC-rich forage will increase risk for development of laminitis (even with moderate NSC content, if there is a plentiful supply of grass, overall NSC intake over a relatively short period of time can be quite high).

(3) A diet based on grass hay (or hay substitute) with low (<10–12% DM) NSC content.

(4) Feeding for maintenance of BW and BCS.

Weight gain will typically exacerbate IR, so it is important to avoid overfeeding. Regular evaluation of BW and/or BCS is the best way to assess the adequacy or otherwise of energy provision.

The core diet for IR animals should be based on grass hay. Mature hay is preferred because of lower digestible energy and NSC content when compared with less mature hay. Ideally, the results of proximate nutrient analysis, including direct measurement of starch and sugars (ie, NSC), should be reviewed before selection of the hay. An NSC content of less than 10% to 12% DM is currently recommended. In the absence of data on hay NSC content, a common recommendation is to soak the hay in water for 30 to 60 minutes before feeding to leach water-soluble carbohydrates (WSC; sugars and fructans). However, recent work has suggested that under typical management conditions, this practice is variable in outcome and may not result in substantial change in the WSC content of some hays.[30] Soaking hay should therefore be used as an adjunct to choosing low-NSC–containing hay. Although sufficient scientific evidence is not currently available to determine whether the 10% to 12% DM upper limit of NSC content is optimal, clinical experience suggests that this advice is reasonable. Additionally, in a small study of healthy Quarter horses versus horses affected by polysaccharide storage myopathy, hay with an NSC content of <10.8% did not affect glycemic and insulinemic responses, whereas a moderate increase in serum insulin concentration was observed after consumption of hay with NSC >16%.[31]

A ration of mostly hay may not meet energy requirements, particularly for PPID animals in which

weight gain is desired or for insulin-resistant horses that are in work. In these situations, the goal is to provide additional calories without exacerbating hyperinsulinemia/insulinemic responses to feeding. One approach is to add nonmolassed sugar beet pulp to the ration, for example, 0.5 to 1.0 kg/d. Beet pulp is rich in highly digestible fibers, provides more DE when compared with most hay types, and does not elicit a marked glycemic or insulinemic response unless molasses is added at the time of processing. Beet pulp shreds should be soaked in a volume of water 3- to 4-fold higher than that of the beet pulp before feeding. The energy density of the ration also can be increased by feeding vegetable oil mixed with sugar beet pulp shreds or with hay cubes that have been softened in water. Corn and soy oils are commonly used in horse diets but must be fresh, nonrancid, and introduced gradually to the ration. One standard cup (approximately 225 mL or 210 g) of vegetable oil provides 1.7 Mcal of digestible energy. Depending on energy requirements, one-half to 1 cup of oil can be fed once or twice daily (up to a maximum of ~1 mL oil/kg BW). Smaller amounts (eg, one-fourth cup once daily) should initially be fed, with a gradual increase over a 7- to 10-day period. Stabilized rice bran (~20% fat) is another option for increasing the energy density of the diet, provided that the calcium:phosphorus ratio of the final ration is considered. A number of commercial feeds are available that contain relatively low starch and sugar content (<20–25% NSC, DM basis) and can be used to add calories to the diet of the lean EMS or PPID horse.

Pasture Access

Pasture access is another consideration in the management of the insulin-resistant, hyperinsulinemic horse. In general, most affected horses can be allowed access to pasture after resolution of the most recent bout of laminitis, but some restriction of grass intake (ie, application of a grazing muzzle) is usually recommended, especially during periods when pasture forage NSC is likely to be high—during spring and early summer; after summer or fall rains that cause the grass to turn green; and when pastures have been subjected to drought or frost stress, all conditions that favor fructan accumulation.

Dietary Supplements

A number of supplements are marketed with claims for improved insulin sensitivity or reduced risk of laminitis, but evidence of efficacy is scant.[32] Many products contain magnesium, chromium, and/or cinnamon. Chromium is thought to potentiate insulin action through activation of insulin receptor kinase and/or inhibition of insulin receptor tyrosine phosphatase.[33] However, the feeding of a supplement containing 5 mg/d chromium, 8.8 g/d magnesium, and other unspecified nutraceuticals for 16 weeks did not alter morphometric measurements, resting serum glucose and insulin concentrations, or insulin sensitivity in obese horses with a history of laminitis.[34] Daily supplementation with 45 g short-chain fructo-oligosaccharide (scFOS) for 6 weeks resulted in a modest improvement in insulin sensitivity and a decrease in resting serum insulin concentrations of obese Arabian horses fed a 50:50 grass hay and sweet feed diet.[35] In EMS horses undergoing dietary restriction (1.25% of body weight as DMI); however, loss of body weight and improvement in insulin sensitivity were unaffected by the addition of scFOS to the diet.[24] More work is needed to evaluate the effects of various nutraceutical supplements marketed for the management of EMS or PPID. Nonetheless, owners should be encouraged to focus on caloric restriction, forage NSC, and so forth rather than the feeding of supplements with unproven efficacy in the management of IR.

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Immunosenescence in Horses

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The process of aging is associated with changes in both adaptive and innate immune responses in people, and similar changes appear to occur in aged horses. Excessive inflammation and impaired response to pathogen challenge may predispose the geriatric horse to many diseases of old age. Few age-specific recommendations are currently available for vaccination of the older horse. In addition, the effect of old age on risk of infectious disease is poorly documented. More work is needed to better understand the interactions of age, diet, vaccination, and disease risk. Author's address: Oklahoma State University, Department of Physiological Sciences, Center of Veterinary Health Sciences, Stillwater, OK 74078; e-mail: diannem@okstate.edu. © 2013 AAEP.

1. Introduction

Immunosenescence is the term that describes the changes that occur in the immune system with advanced age. Changes reported to occur in aged people include alterations in the composition of lymphocyte populations as well as blunted or dysregulated immune responses.[1] Immunosenescence is an important risk factor for morbidity and mortality of the aged and contributes to development of the infectious, neoplastic, and inflammatory diseases that afflict the elderly population. Current available evidence suggests that horses undergo similar age-related changes in immune function as do people.

2. Considerations of Aging Studies in Immunology

Scientific evaluation of age-induced changes in immune function is not straightforward. First, it is important to separate age-associated functional changes from disease-induced changes. In an aged population, this can be difficult because of the high prevalence of co-morbidities that contribute to chronic, low-grade inflammation. Ideally, a strict recruitment protocol should be used. In human immunological research, a screening procedure known as the SENIEUR protocol has been developed,[2] which combines a history, clinical examination, diagnostic blood work, and urine analyses to identify healthy aged individuals for study inclusion. No such standardized protocols have been adopted in veterinary gerontology. In studies of aged horses, it is important to consider hormonal status during the study period because several of the pituitary and adrenal hormones are strong modifiers of immune function. The profound effect of season on the output of pars intermedia hormones and the high prevalence of pituitary pars intermedia dysfunction (PPID) in the aged equine population[3,4] can markedly confound a study investigating age-associated immune function in horses. Other important considerations when assessing immune function in aged horses include diet, endoparasite load, medications, nutritional supplements, exercise, season, transport, travel, housing, and environmental conditions of the study participants.

A second consideration when investigating age-related changes of immune function is how to define "aged." Age-induced changes may be missed if the

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study group is too young. Less intuitively, a study population that is too old may cause age-related immune defects to be missed because the selected population may have survived to extreme old age as the result of exceptional immune function.[5] It is likely that biomarkers of aging differ with the progression of animals from old to very old age. Certainly, risk of specific diseases, frailty, or other impairments vary along the spectrum of aging. Finally, it is also important that the control population be a suitable age. For example, adult animals are needed in the control population if the goal of the study is to compare the immune function changes that develop in old age, whereas the control group would include younger animals if the goal was to assess the whole spectrum of age-associated immune changes.

3. Age-Associated Changes in Immune Function

Cell Populations

Age-associated alterations in lymphocyte subset populations have been documented in several species including people, dogs, and rodents.[6–9] A robust decrease in naive T-cells, thought to be the result of thymic involution, is a consistent finding in aged people and rodents.[10–13] In addition, it has been suggested the loss of naive T-cells may be the result of lifelong infection by viruses, especially herpes viruses such as cytomegalovirus or other chronic antigenic stimulants.[14,15] Because of a lack of available antibodies that differentiate equine memory cells from naive cells, changes in naive T-cell populations have not been directly examined in aging in the horse.

Other findings in human leukocyte populations include alterations in total lymphocytes, CD4, CD8, and B-cells. The CD4:CD8 ratio, an indicator of whether lymphocyte populations favor an inflammatory or immunosuppressive bias, has been reported to be altered in human geriatric populations.[16–18] Several studies have investigated lymphocyte populations in aged horses and ponies.[19–22] Total lymphocyte populations decrease in aged horses.[19–21] Other observations include a decrease in the total number of CD4, CD8, and B-cells[21] and an increase in the percentage of CD4 lymphocytes in aged horses.[22] In ponies, a decrease in total monocyte and eosinophil counts has been reported.[20] The CD4:CD8 ratio was found to be increased in aged horses,[21] suggesting that a proinflammatory state occurs in old horses as in people.

Cytokine and Acute-Phase Protein Profiles in the Aged Horse

Serum cytokine profiles in the aged human typically are those of a pro-inflammatory phenotype.[23,24] Aged horses show similar cytokine profiles with increased gene expression of tumor necrosis factor (TNF)-α, interleukin (IL)-6, IL-1β, IL-8, interferon (IFN)-γ, IL-15, and IL-18[22,25] and increased proinflammatory: anti-inflammatory cytokine ratios including IL6:IL10 and TNF-α:IL10 ratios.[25] Serum cytokine concentration of TNF-α was increased in aged horses in one study[22] but not another.[25] Several confounding factors might affect serum TNF-α concentration including concurrent illness, inflammation, or season of sample collection. The role of other serum cytokines in aging of horses has not been extensively examined because of the limited availability of valid assays. The effect of age on serum acute phase protein concentrations is not yet known, although assays validated for use in horse are now available for the measurement of serum amyloid A and C-reactive protein.

PBMC Cytokine Response to Stimulation

Several studies have evaluated the impact of aging on the responsiveness of equine peripheral blood mononuclear cells (PBMC) after immune stimulation. Inflammatory response after stimulation of whole blood or PBMC from healthy aged people results in a greater release of proinflammatory cytokines than that observed in adults.[26,27]Similar studies have revealed an increase in TNF-α and IFN-γ production from PBMC after stimulation in aged horses.[22,25]

Lymphocyte Function

One of the most consistent findings in immunosenescence is a decrease in the ability of lymphocytes to proliferate. Studies in rodents and people have shown impaired lymphocyte proliferation may involve a decrease in serum IL-2 concentration or IL-2 receptor expression.[11,28,29] However, a decrease in lymphocyte proliferation has also been reported independent of these two factors, suggesting defects in intracellular signaling may also occur with aging.[30] Horses also have an age-associated decrease in lymphocyte proliferation[20,22,31] that is not responsive to IL-2 supplementation and not associated with a change in lymphocyte IL-2 receptor expression.[20] This suggests that, similar to what has been reported in people, the age-associated defect in lymphocyte division in horses may be caused by alterations in intracellular signaling.

Neutrophil Function

Although immunosenescence affects primarily adaptive immunity, changes in innate immunity occur as well. Innate immunity includes the response of neutrophils and macrophages as a first-line defense against pathogens. Age-associated changes in neutrophil function that have been reported in people and rodents include decreases in phagocytosis, oxidative burst, chemokinesis, and chemotaxis. Neutrophil numbers appear to be maintained.[32–35] In healthy aged horses, neutrophil adhesion, oxidative burst, and phagocytosis were all found to be unchanged, whereas chemotaxis was increased, compared with that observed in healthy adult horses.[36] In contrast, preliminary data suggest that horses

with PPID have impaired neutrophil function, perhaps contributing to the increased frequency of bacterial diseases such as abscesses and sinusitis. Because of the difficulty making an early diagnosis of PPID, it may be necessary to consider all aged horses at higher risk for neutrophil impairment unless proven otherwise.

Vaccine Responsiveness

One of the major health concerns regarding immunosenescence in people is a failure of the geriatric population to develop adequate titers after vaccination, particularly after influenza virus immunization.[37] As a result, morbidity and mortality caused by influenza infection is greatest in the very old. The response of aged horses to influenza vaccines has also been reported to be less robust than that of young or adult horses in several studies.[19,37] Muirhead et al[38] reported a decrease in immunoglobulin subtypes immunoglobulin (Ig)Gα and IgGβ in aged horses, although single radial hemolysis titers suggested both adults and aged horses had an adequate response to be protective. With the use of a different vaccine, Horohov et al[20] reported a 10-fold decrease in resultant titers in aged ponies, compared with adults. Response to naive antigenic challenge was also examined in horses >20 years of age. Contrary to what has been reported in primates,[39–41] the magnitude of a primary antibody response did not decline with age.[38] When a rabies vaccine was administered to rabies vaccine–naive aged horses, the antibody titer after both the initial and second immunization did not differ from that of adult horses. However, in this study, 80% of both the control and aged population had low serum selenium concentrations, which could result in a suboptimal vaccine response in both groups. Further studies are needed to clarify the ability of the aged horse to respond to naive and amnestic vaccine challenge and to assist in the development of ideal vaccination protocol for geriatric horses.

Although the scientific literature is sparse, aged horses appear to be at greater risk of development of neurologic disease after West Nile infection compared with human adults.[42] Although not necessarily the result of immune impairment, in an experimental model of neuropathogenic equine herpes virus (EHV-1) infection aged horses appear more susceptible to develop neurologic signs.[43] Enhanced risk of infectious disease may be minimized by a complete and appropriately administered vaccination schedule.

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Maintaining the Health of Aged Horses: Practical Tips for the Equine Practitioner

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Aging in the horse has been a focus of interest and research in the past two decades. Although endocrine dysfunction in the form of pituitary pars intermedia dysfunction (PPID) is the most specific age related disease, all body systems are affected by aging. This report will highlight how these other body systems are most affected in an attempt to develop a diagnostic and therapeutic plan for maintaining our older horses. Author's address: Tufts Cummings School of Veterinary Medicine, 200 Westboro Road, North Grafton, MA 01536; e-mail: MaryRose.Paradis@tufts.edu. © 2013 AAEP.

1. Introduction

What is considered old in the horse? Many have asked questions about aging in the horse, and our definition of "old" is changing. In the past 20 years, we appear to be seeing more horses over the age of 20 years in our hospital. A small study indicated that from 1989 to 1999 we saw an almost six-fold increase in horses presenting with problems who were 20 years of age or older.[1] Numbers rose from 2.2% of the hospital population to 12.5%. In a 2007 survey of equine veterinarians in New England, all practitioners responded "yes" when asked if they had horses 20 years of age or older. Thirty percent of responding veterinarians stated that between 21% and 40% of their caseload was composed of horses 20 years of age or older (unpublished data).

So, are horses living longer than before? It is hard to separate all the different factors—from economic to increased longevity. In the 1998 National Animal Health Monitoring System (NAHMS) study, 7.5% of resident equids were 20 years of age or older.[2] In the New England study in 2007, the proportion of horses over the age of 20 years was double the national average. However, this does not necessarily mean that horses are living longer from 1998 to 2007, because the NAHMS survey only looks at horses at farms in which there are more than five horses. Many retired horses live on small home farms and may be underestimated by the NAHMS study.

There is some controversy in research concerning the age at which a horse is considered geriatric. In early studies and more recent studies in Australia and Great Britain, researchers would consider horses old if they were 15 years or older.[3,4] When owners of older horses were asked at what age did they notice aging changes in their horse, the answer was around 23 years of age. Age was considered a negative factor in the purchase of a horse if they were 16.5 years old or older.[5]

Are there specific breeds that live longer than others? There does not appear to be any breed of horse that has a "longevity gene," with the exception of the pony. In multiple studies, ponies have a higher percentage of animals in the more-than 30

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years of age category especially when compared with the percentage of ponies in the younger population.[1,5,6] Anecdotally, mules are also stated to have long lives. Why the difference? Some say that ponies originally come from areas of the world that require a hardier constitution. Others state that perhaps the smaller body size plays a role as seen in other species. Wilmink et al[7,8] found a difference in second-intention wound-healing between horses and ponies. Ponies healed faster than horses from a standardized wound, so perhaps there are physiologic differences that contribute to longer life span.

2. Are the Changes We See Caused by Old Age or Disease?

It is difficult to clearly denote whether some of the signs we see in the aging animal are caused by normal aging changes or by disease processes. For instance, older animals are known to have decreased spontaneous activity. In the older horse, we may wonder if the decrease in spontaneous activity is not secondary to old age but a result of degenerative orthopedic disease. The general signs of aging in the horse include loss of "top line" muscle mass—sway back, graying of haircoat, and musculoskeletal stiffness. However, one could also argue that decreased muscle mass is secondary to decreased activity or pituitary pars intermedia dysfunction (PPID) and that stiffness is secondary to arthritis.

Older horses have many of the same medical issues as young horses, but some conditions appear to increase with age. The specific disease most commonly associated with old age in the horse is PPID, commonly known as equine Cushing's disease. This was covered in the previous talks in this session. Although changes can be seen in all body systems of the aging horse, the gastrointestinal system, the musculoskeletal system, and the respiratory system are the most commonly presented for veterinary care in the older horse. When owners were asked about the use of medications for chronic problems, 25% of old horses versus 6% of young horses were on regular medications. The medications fell into three categories of treatments: pain relief, recurrent airway inflammation, and PPID.[1] The rest of this discussion will look at these body systems plus a few other problems that are most commonly encountered in the horse more than 20 years of age.

3. Gastrointestinal Problems

Colic was the most common reason for geriatric animals to be evaluated at a referral center in the study performed by Brosnahan et al.[1] Williams[6] looked at the body systems represented in horses more than 15 years of age at postmortem examination and found that lesions in the gastrointestinal system were the most common reason for death. The lesions varied, but included intestinal volvulus, displacements and ruptures.

Brosnahan et al[1] showed that 45% of horses 20 years of age or older presenting to a hospital for colic had large-colon problems, whereas 40% of the cases were small-intestine lesions. Forty-four percent of small-intestine problems were caused by lipomas.[1] Strangulating lipomas of the small intestine were the second most common disease problem of geriatric horses. This finding is supported by results of studies that indicate a significant increase in the incidence of strangulating lipomas with increasing age of horses.[9] Large-colon impaction was the fifth most common specific disease diagnosed in the horses studied by Brosnahan et al. Gastric lesions, including ulceration and neoplasia, were found in 18% of horses presented with abdominal pain.[1]

Dental disease probably plays a large role in the incidence of large-colon impaction and in esophageal choke in the older horse. Older horses are significantly more likely to have a history of dental disease than younger horses. In the old horse study from Britain, 95% of the horses more than 15 years of age had dental abnormalities.[4] This was often overlooked by owners who reported that only 10% of their old horses had dental disease, whereas an additional 15% of owners reported that their horses exhibited "quidding" of hay. Owners reported that dental care was provided less than once yearly in 11% of older horses. Dental care was provided by doctors of veterinary medicine 49% of the time in older horses, with the remaining care being provided by dental technicians.[1]

As the horse ages, the teeth continue to wear down. Even the horse that has had good dental care all its life can develop a "smooth" mouth. Smooth mouth occurs from normal wear of the enamel ridges of the teeth over time. This process may be hastened by chronic ingestion of sand or overly aggressive floating practices. As the tooth continues to wear down, it may appear as two teeth—this is because the tooth is worn to the level of the roots.

Other dental conditions that are commonly encountered in the older horse include wave mouth, step mouth, hooks, shear mouth, and equine odontoclastic tooth resorption and hypercementosis (EOTRH).[10,11] Wave mouth is the result of uneven wearing of the cheek teeth. Because molars are always permanent teeth, the first molar or fourth cheek tooth is the oldest tooth in the mouth. In wave mouth, the upper PM4 and M1 are often worn to the gum line, whereas the lower opposing teeth are longer, creating an arcade that wears and grinds food abnormally. A step mouth is similar but is usually caused by the absence of one tooth and the overgrowth of the opposing tooth. Hooks develop on the upper first cheek tooth and the lower sixth cheek tooth in response to malocclusion of the dental arcade and decreased wearing surfaces. Shear mouth is a condition in which the lingual points of the lower teeth come in contact with the hard palate. Laceration of the gums or palate may occur in these

problems. EOTRH is a painful disorder of incisor and canine teeth in some aged horses. The etiology is unclear but thought to be related to periodontal inflammation. Extraction of affected teeth appears to be the treatment of choice.[11]

Radical correction of severe dental disease may not be effective in improving the animal's ability to eat forage. Shortening the height of abnormally long and sharp teeth should be considered to improve the comfort of the animal. Changes to dietary management may be necessary.

4. Body Condition and Nutrition in the Older Horse

When one thinks of older people and animals, thinness and frailty come to mind. In one study, 68% of the horses more 20 years of age were rated as having good to moderate body condition.[1] This was comparable to that of the younger animals. Twenty-eight percent of the older individuals were rated as fat or very fat, and only 4% had a poor or very poor score.[5] It was concluded that older horses did not have difficulty maintaining weight and that obesity might be more of a concern than thinness.[5] When asked what the diet of the older horse consisted of, 89% said that they fed hay, with 7% feeding a hay substitute and 4% not receiving hay in any form. Fifty-one percent of the older subjects were on some type of senior feed. More than half of them received a supplement: 66% received a general vitamin/mineral supplement and 47% received a joint care product.[5]

What do we actually know about nutritional needs of the older horse, and how do they differ from the younger animal? Essentially, when dealing with healthy older horses, their nutritional requirements may not differ from that of the younger adult. The problems arise in that many of our older individuals are not normal. Dr Ray Geor has provided us with guidelines for feeding the older horse with endocrine abnormalities in the previous talk. Obesity and insulin resistance can put the older horse at high risk for laminitis. This should be diagnosed, and these horses should be fed according to his recommendations.

There have been recommendations that the older horse needs a different diet because of decreased ability to digest food. Feeds may come in the extruded form to increase the surface area of the feed pellets exposed to digestion. Prebiotic and probiotic compounds may be added with the idea of increasing the geriatric horse's digestive flora. Because the older person and the older horse tend toward decreased muscle mass, many of the senior feeds will have a higher protein content than their performance horse feeds. Certain amino acids, such as lysine and biotin, may be added to the senior feed to help in "muscle maintenance," haircoat, and hoof care. The senior feeds are often advertised as complete feeds for the horse that has trouble ingesting long-stem roughage, and fat is added to increase the caloric content.

Often, feeding of the geriatric horse comes down to feeding whatever they will eat. Horses with poor dentition will have problems with hay and whole grains. Alternative forages include grass, hays with minimal stem, and soaked hay cubes or haylage. The use of current senior diet formulations can be helpful in keeping weight on the geriatric animal because of the increased digestibility of these products. One must be careful when switching to a senior feed because some of these feeds are not calorie-dense and cannot be fed on a volume basis. By continuing to feed the same volume, one may actually be decreasing the total caloric intake for the animal, which may result in unexpected weight loss.

5. Weight Loss in the Older Animal

The diagnostic workup for weight loss in the older horse is the same as for horses of any age. The amount of weight that is gained or lost is dependent on the calories that are taken in versus the calories that are used. The decreased intake in the older animal may be caused by lack of good-quality feed, poor appetite secondary to a debilitating disease, poor dentition, maldigestion, and malabsorption. Reasons that an older horse may have an increased utilization of calories may relate to environmental cold, increased level of exercise (less likely), and increased catabolism secondary to a debilitating disease. Recurrent airway obstruction (RAO) is a good example of a debilitating disease in the older horse. Not only does the increased respiratory rate and effort increase the caloric need of these animals, the affected animals cannot take time away from breathing to actually eat enough to meet this need. Neoplasia should be on a list of differential diagnoses for weight loss in the older horse. Although it is not as common as it is in other species, it is still seen. Abdominal neoplasia may include lymphosarcoma, squamous cell carcinoma, adenocarcinoma, leiomyosarcoma, melanoma, and mesothelioma.[12]

A good nutritional history is important, and it should include grain and forage analysis. A thorough physical examination and a complete blood cell count and chemistry profile should provide a minimum data base. From this point, a per rectum palpation and an abdominal ultrasound will be important to check for abdominal masses or intestinal abnormalities. Abdominocentesis is helpful in ruling out peritonitis but less helpful in diagnosing neoplasia because tumor cells are rarely shed by the tumors. Gastroscopy is useful in determining the presence of gastric ulcerations and squamous cell carcinoma of the stomach. A rectal biopsy may be useful in diagnosis of inflammatory bowel disease. If a diagnosis is not found after this workup, an exploratory laparotomy may be the best option.

6. Musculoskeletal Disorders in the Older Horse

Musculoskeletal problems were the second most common presenting complaint in the Brosnahan

hospital study. Of the horses presenting for lameness, 37.5% had laminitis (mostly in conjunction with PPID), and 55% had lameness classified as degenerative disease. Older horses were significantly more likely to be given long-term medications than were younger horses. The most prevalent medication given to older horses fell in the nonsteroidal anti-inflammatory drug (NSAID) and chondroprotective categories. One can assume that this is an indication of chronic musculoskeletal pain.[1]

It is hypothesized that earlier injury to joints, muscles, tendons, and ligaments set up the geriatric horse for progressive degenerative changes in the musculoskeletal system. Loss of shock absorption is caused by chronic inflammatory changes in the synovial fluid, cartilage defects, and sclerosis of subchondral bone. Osteophytes are a hallmark of osteoarthritis. Cartilage research in old horses demonstrated that glycosaminoglycan (GAG) levels remain constant throughout life, but there are age-related decreases in proteoglycan size though the loss of GAG chains.[13] Older horses also have greater pentosidine crosslinks, which may predispose older horses to osteochondral disease caused by stiffer and more brittle cartilage.[14] A progressive degeneration of the suspensory ligaments in the hind limb is seen in some geriatric horses (particularly older broodmares). Clinically, these horses have straight hock conformation and progressive sinking of the fetlocks.

Management of osteoarthritis in the older horses involves judicious use of NSAID drugs and increasing mobility. Increasing the mobility of the animal plays a role in decreasing pain. One should avoid stall rest in these animals. Unlimited turnout is preferred. If the older horse is still in work, modifications to the training program may need to be implemented. Older horses will probably take longer to become physically fit. Older horses fatigue quicker than younger horses. Continuous low-level work will be better for the older horse than a "weekend warrior" session. The addition of nutraceuticals such as glucosamine-chondroitin sulfate has been shown to improve lameness grade, flexion test grade, and stride length in horses with osteoarthritis. Acupuncture, chiropractic treatment, and massage therapy may be helpful in the older horse that has multiple problems.

7. Respiratory Problems in the Older Horse

Respiratory tract disease was the third most common body system affected among the geriatric horses in the Brosnahan study. Although RAO or heaves is not restricted to old horses, age has also been determined to be a risk factor for RAO. It was the most common specific respiratory tract disease diagnosed in our study population. In the survey study, 19% of the older horses that were receiving medication were receiving drugs that were compatible with the treatment of heaves.[5] RAO is an inflammatory airway disease similar to asthma in humans. Inflammation of the airway plus bronchoconstriction leads to severe obstruction.

Clinical signs of RAO include increased respiratory rate and effort, cough, the development of hypertrophied muscle along the ventral rib cage, exercise intolerance, and increase crackles and wheezes on auscultation of the lung fields.

Treatment of RAO is aimed at decreasing inflammation and bronchoconstriction. Environmental reduction of allergens is a main objective in the treatment of this disease. Providing 24-hour turnout, eliminating hay from the diet, improving ventilation, and decreasing dust in the environment are important elements of the treatment. For a more rapid reduction in lung inflammation, oral or inhaled steroids are the primary anti-inflammatory drug of choice. Dexamethasone and prednisolone are the system drugs of choice, and beclomethasone is the most commonly inhaled steroid.

Bronchodilation can be achieved with several different drugs, including the following: albuterol (inhaled), clenbuterol (oral), and aminophyline (oral). Inhaled albuterol is probably the most effective.

8. Ophthalmology in the Older Horse

Although not commonly reported by owners, 94% of horses more than 15 years of age had at least one abnormality identified by external ocular and ophthalmologic examination by a veterinary investigator. Degeneration of the vitreous was the most common lesion seen in the aging horse.[15,16] In aging humans, the vitreous liquefies and creates "floaters." This may be seen in horses as a consequence of recurrent uveitis. Senile retinopathy is the second most common ophthalmologic problem seen in geriatric horses. The findings consist of irregular linear hyperpigmentation in the non-tapetal fundus and depletion of pigment in the adjacent areas.[15] Ireland at al[15] reported that the median age for senile retinopathy was 24 years. In the same study, 58% of the geriatric horses examined had evidence of cataracts. The median age for affected animals was 23 years; for bilateral cataracts, the median age was 25 years.[15]

9. Neoplasia

Squamous cell carcinoma and melanomas are the two tumor types that increase in incidence with age. Squamous cell carcinoma is the most prevalent. It is most commonly seen on the eye and equine penis and prepuce.[17] Masses may be single or multiple. In one study, affected animals had a mean age of 16.5 years of age.[17] The lighter, non-pigmented skin is more susceptible to development of squamous cell tumors. It spreads by metastasis to local lymph nodes. Preputial lesions metastasize to the corpus cavernosum penis and inguinal lymph nodes.

Approximately 80% of older gray horses have evidence of external melanomas. These rarely metas-

tasize, but in a study of central nervous system tumors, melanomas were the second most prevalent and appear to be age-related.[18]

10. Extending or Ending Life

The long duration of life of horses enhances the bond with their owners, despite their loss of performance and reproductive capacity by sometimes one to two decades. Older horses can lead healthy lives into their 30s, particularly if care is taken to address problems as soon as they are recognized. The development of an annual or biannual geriatric assessment is important in helping to identify treatable problems. This assessment should include a thorough physical examination with special attention paid to the body condition of the animal, dental wear, appetite, presence of long hay fibers or whole grain in the manure, haircoat abnormalities, musculoskeletal stiffness or lameness, ophthalmic abnormalities, and the development of cardiac murmurs. Blood work should include a complete blood cell count, chemistry profile, adrenocorticotropic hormone levels, and perhaps insulin levels. Fecal examination for parasites should also be included. Therapeutics will often be directed at improving the quality of life by reducing pain, improving nutrition, and relieving the stress of chronic respiratory or endocrine problems.

The veterinarian is often asked when it is best to euthanize the older horse. The NAHMS study stated that "old age" was the most frequent cause of death in horses more than 30 years of age. Ireland et al[19] reported that lameness, colic, chronic illness, acute illness, laminitis, and fracture were the top six reasons for euthanasia. Factors that influenced owners to euthanize their older horses were a hopeless prognosis, veterinary advice, and poor quality of life.

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Review of Management of Anestrus and Transitional Mares

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Although mares are regarded as seasonally polyestrous animals, great individual variation exists regarding the onset of fall transition and duration of ensuing winter anestrus, as well as the onset of spring transition. Although most mares transition to a period of reproductive quiescence, approximately 10% to 20% of mares continue to exhibit estrous cycles throughout the year.[1] The onset of winter anestrus and subsequent spring transition are primarily dependent on photoperiod or day length.[1] Day length varies by latitude, and mares are more likely to enter winter anestrus in more extreme latitudes and more likely to cycle yearround in the lower latitudes nearer the equator. Changes in photoperiod are detected by the retina and processed by the pineal gland. During periods of darkness, the pineal gland releases melatonin.[1] Melatonin appears to play a pivotal role in seasonality of reproduction; however, the mechanism by which melatonin exerts its action in the mare is unclear. In the autumn, as day length decreases, the duration of melatonin secretion increases. During the spring, as day length increases, the duration of melatonin secretion decreases, and a concurrent increase in gonadotropin-releasing hormone (GnRH) release is observed. In response, the anterior pituitary increases secretion of gonadotropins, and follicular activity begins. It appears that luteinizing hormone (LH) plays a more critical role than follicle-stimulating hormone (FSH) in initiating the onset of cyclicity. During anestrus, when GnRH is low, LH is at basal levels, whereas FSH does not change dramatically throughout the year.[2] Although other factors, both systemic and local at the ovarian level, appear to be involved in the onset of anestrus and the resurgence of cyclicity, many questions remain unanswered, (as reviewed in Donadeu and Watson[3]).

Whether or not a mare transitions to winter anestrus and the duration of her anestrus may be influenced by factors such as nutrition, age, breed, and temperature. Studies have shown that mares in good body condition are more likely to cycle year around, and mares in good body condition that do enter into anestrus begin cycling on average 1 month earlier than mares in poorer body condition.[4,5] Similarly, an increasing plane of nutrition or grazing on green grass is associated with an earlier return to cyclicity.[6,7] Older mares tend to begin cycling slightly later than young mares,[8,9] and po-

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nies tend to be more seasonal than horses.[10,11] Ambient temperature is considered a modulating factor during the spring transition. A warm spring seems to initiate earlier cyclicity.[12,13] Conversely, breeders will often report that a sudden cold snap during spring transition will cause mares to revert back to an anestrous-like state. This is especially true in conditions of extreme cold that create a demand for energy to maintain the core body temperature. Temperature probably modulates the effectiveness of dopamine antagonists such as sulpiride, used in an attempt to initiate cyclicity.[14] It is often difficult to separate out confounding variables when examining the effect of one variable on the onset of cyclicity. For example, in the spring, as the day lengthens, temperatures are usually on the rise and horses tend to be turned out to graze on spring pastures rather than be kept indoors. All three factors probably are conducive to mares emerging from winter anestrus into a transitional state.

For many breeds of horses, an industry imposed birthdate of January 1 has created an incentive to produce early season foals. This is advantageous in breeds in which horses compete in athletic events at a relatively young age because an earlier foal is of course more mature than another foal born later in the season that year. To produce maximally competitive foals, breeders strive to deliver them as close to January 1 as possible; however, this becomes difficult because the mare has an 11-month gestation period, and an early delivery necessitates an early conception. Therefore, it is important that mares begin regular, fertile estrous cycles as early in the year as possible. However, mares are not typically reproductively active at the time of desired breeding; therefore, many resources have been invested in the effort to induce earlier cyclicity. There are two targets at which to aim when attempting to induce earlier cyclicity: (1) shorten the anestrous period and (2) shorten the transition period. Artificial light to supplement the photoperiod is commonly used by breeders to induce earlier cyclicity in anestrous mares. Providing artificial light to simulate increased day length earlier in the year is a relatively easy, inexpensive, and effective method to initiate an earlier return to cyclicity; however, mares still go through a period of transition.[15] Photoperiod adjustment is usually begun around December 1 in the Northern hemisphere. A common protocol is to provide a period of light for 15 to 16 hours per day (followed by 8 to 9 hours of darkness). This is best accomplished by turning the lights on at the end of the day for a few hours. Alternatively, a short (1 to 2 hours) period of light can be provided during the night (usually 8 or 9 hours after onset of darkness) to interrupt the duration of the melatonin influence (as reviewed by McCue et al[16]).

As mares come out of anestrus and enter the transitional period, multiple waves of follicular growth and regression will be seen if the mares are observed closely through ultrasonography. Sharp et al[15] reported that pony mares in Florida had, on average, 3.7 follicular waves before the first ovulation of the year. The early follicles are not steroidogenically competent and do not produce sufficient quantities of estrogen to alter concentrations of estrogen in the serum. They can, however, produce low levels of estrogen that can result in estrous behavior. During this transitional period, estrous behavior exhibited by a mare can be very erratic. Transition typically lasts for weeks, and mares may exhibit prolonged periods of estrus, interspersed with variable periods of passivity or rejection of the stallion. This presents a frustrating and sometimes confusing situation for horse owners who desire to breed their mare early in the season. When a mare is in obvious estrus, it would seem advisable to breed her; however, if she is still in transition, that display of estrus is probably not accompanied by a fertile cycle. Therefore, breeding a mare in transition is a waste of time and resources. Furthermore, breeding during transition can actually decrease the chances of getting a problem mare in foal. For example, the repeated deposition of semen into the uterus of a mare with impaired fertility and the resulting inflammation from each breeding can exacerbate an already tenuous situation.

As the period of transition progresses and the developing follicles increase in size, they eventually become steroidogenically competent and produce detectable levels of estrogen approximately 1 week before the first ovulation.[17] The estrogen stimulates production of LH from the pituitary, and the eventual LH surge will result in maturation and ovulation of a dominant follicle.

Researchers have investigated many ways to shorten transition and advance the onset of cyclicity in the effort to produce early season foals. Some techniques have included follicle aspiration, use of ovulatory inducing agents such as human chorionic gonadotropin (hCG) or recombinant LH, and various schemes of administering GnRH or GnRH analogues in the form of pumps or slow-release formulations. Ultrasound-guided aspiration of the dominant follicle during transition resulted in a shortening of transition in studies performed in our lab. Mares that underwent follicular aspiration had a rise in progesterone 10 days after a 35-mm follicle was observed, compared with 35 days in control mares.[18]

Attempts to induce ovulation during transition by pharmacological means have not been routinely successful. Studies on the use of either exogenous GnRH or GnRH analogues to induce cyclicity have had conflicting results. Most researchers have observed an increase in both FSH and LH in response to exogenous GnRH treatment in anovulatory mares; however, the effects on follicle dynamics and ovulation vary. Deep anestrous mares were administered varying dosages (0, 50, or 100 ng/kg/h) of

GnRH through osmotic minipumps.[19] Ovulation did not occur in control mares; however, in both groups receiving 50 or 100 ng GnRH, ovulation in three of 10 and seven of 10 treatment mares occurred 20 and 19 days after treatment, respectively. A dose-dependent increase in LH was also observed.[19] Similarly, Allen et al[20] increased GnRH in anestrous pony mares and light horse mares by injecting either one or two slow-release polymer implants impregnated with either 0.9 or 1.8 mg of GnRH agonist (ICI 118 630). The implants delivered either 30 or 60 μg GnRH analogue, respectively, for 28 days. Thirteen pony mares (76%) and 120 horse mares (88%) ovulated within 3 to 18 days after treatment. Seventy of the 100 horse mares bred during that time conceived.[20] Conversely, Mumford et al[21] did not observe an increase in follicle development (number or size) after fitting anestrous mares with subcutaneous implants containing varying dosages of the GnRH analogue goserelin and observed only a 35% increase in ovulation rate. The increased ovulation rate depended on dosage as well as ovarian status before treatment.

The success of treatment with GnRH or its analogues appears to depend on how deep into anestrous a mare is. The use of the GnRH agonist deslorelin in late-transitional mares advanced the date of first ovulation by approximately 2 weeks, but repeated injections were needed.[22] Any treatment is less likely to be effective if a mare is in deep anestrus. As spring progresses and the more advanced a mare is into transition, the more likely a treatment is to be successful. Thorson et al[23] found that if mares received a continuous infusion of GnRH (100 μg/h for 28 days) beginning in February, they would return to acyclicity when treatment was stopped. However, if treatment was begun in March, mares would continue to cycle. Recently, Newcombe et al[24] were successful in initiating earlier cyclicity with deslorelin in mares pre-treated with intravaginal progesterone (CIDR). Ninety-six percent (143/149) of the mares treated with deslorelin pumps ovulated earlier, but that rate of ovulation depended on the size of the largest follicle at time of treatment.[24] The use of GnRH and its analogues appears to have limited advantages in inducing earlier cyclicity, and, of those methods that have been successful, repeated injections were needed and timing was crucial. Frequent, repeated administration of GnRH analogues has not yet proven practical and/or inexpensive for breeders.

In another approach to hasten the onset of the ovulatory season, Schauer et al[25] supplemented growing follicles with equine LH (eLH) (from pituitary extracts) during early transition to stimulate development of steroidogenically active dominant follicles with the ability to respond to an ovulatory stimulus. Mares received eLH, q 12 h IV, until a follicle reached 32 mm, at which time they received hCG, 3000 U IV. Although eLH treatment stimulated follicle growth, it failed to produce steroidogenically active follicles responsive to hCG. Furthermore, eLH did not hasten the onset of the ovulatory season because the treated mares that did ovulate subsequently returned to an anovulatory transition state.[25]

Progesterone has also been used in various approaches, including slow-release formulations, oral progestogens, and intravaginal devices.[26] Staempfli et al[27] administered a single dose of a slow-release formulation of progesterone to early- and late-transitional mares. Treatment had no effect on the early-transition mares but resulted in ovulation in 10 to 24 days in 10 of 12 mares treated in late transition, versus only three of 12 control mares during the same time period. Many other studies, dating back to the 1970s, have found similar results. Mares in anestrus or early transition do not respond to progesterone therapy, whereas ovulation may be advanced in mares in late transition (as reviewed by McCue et al[28]). When a mare has reached the stage of transition in which follicles of 30 mm or larger are detected, she can usually be successfully "normalized" into regular estrous cycles by daily feeding of the oral progestogen, altrenogest[a] or an injection of a long-acting formulation of progestin.[b14]

One of the most promising approaches to inducing earlier cyclicity in mares has been to manipulate endogenous prolactin. Prolactin administered to anestrous mares has been shown to stimulate ovarian activity and to induce ovulation in seasonally anovulatory mares.[29,30] Another way of manipulating prolactin is through the use of dopamine antagonists such as domperidone or sulpiride. Dopamine antagonists counteract the effects of hypothalamic dopamine on the adenohypophysis and enhance prolactin secretion in the winter, when plasma concentrations are normally very low.[30,31] It is assumed that the dopaminergic antagonists cause their effect by stimulating prolactin secretion. Early studies examining the effects of dopamine antagonists in anestrous mares found that sulpiride, either 1 mg/kg, q 24 h IM, begun in late January and continued until ovulation,[32] or 200 mg, q 24 h IM, begun in early February and continued until ovulation or for a maximum of 58 days,[33] advanced the first ovulation of the year by 21 d or 33 days, respectively. However, Donadeu and Thompson[34] administered sulpiride 1 mg/kg, q 24 h SC, for 32 days beginning in mid-January and failed to see an effect on ovarian activity or ovulation, indicating the importance of prolonged administration when treatment is initiated very early in the year in anestrous mares.

As previously stated, many factors undoubtedly affect the onset of cyclicity in a mare. For example, Gentry et al[5] fed mares to achieve a high or low body condition. Whereas those mares with low body condition ceased follicular activity and entered ovarian quiescence in the winter, all mares in the high body condition group except one continued to cycle or had significant ovarian activity. Therefore, it is not unreasonable to expect factors such as body condition,

nutrition, environmental temperature, photoperiod, and so forth, to affect the response to dopamine antagonists and play a role in the onset of cyclicity. Duchamp and Daels[35] first put mares under 14.5 hours of light beginning on January 10, for 2 weeks before administering sulpiride (1 mg/kg, q 12 h IM) until ovulation or for a maximum of 21 days. Treated mares ovulated almost 17 days earlier than controls. Nearly 73% ovulated within 28 days of the beginning of sulpiride treatment.

Kelley et al[36] showed that pretreatment with estrogen greatly enhanced the prolactin response to daily treatment with sulpiride. Mares received 10 injections of estradiol benzoate (11 mg, q 48 h IM) or vegetable oil as a control, beginning on January 11. Sulpiride treatment (250 mg, q 24 h SC) was begun 11 days after initiation of the estradiol treatment. Mares that received estradiol ovulated 45 days earlier than mares receiving sulpiride alone. Similarly, Mitcham et al[37] used estrogen to prime mares before treatment with domperidone. In January, mares in anestrus received a single injection of domperidone (3 g, in biodegradable particles, IM) either alone or after a single injection of estradiol cypionate (ECP) (100 or 150 mg IM), whereas another group also received progesterone (1.5 g, long-acting formulation, IM), and another group received ECP (150 mg IM) and progesterone (1.5 g, long-acting formulation, IM), without domperidone. Mares receiving ECP before domperidone ovulated earlier than those not receiving ECP. Of the mares that received ECP, 14 of 31 ovulated within 35 days, whereas of those that did not receive ECP, only three of 36 ovulated within 35 days, including none of nine that received domperidone alone. Timing of pretreatment with ECP relative to administration of domperidone (1, 6, or 11 days apart) was also assessed. Administration of domperidone 1 day after treatment with ECP provided the most positive response on prolactin compared with day 6 or day 11. The addition of progesterone did not affect the date of first ovulation. Likewise, estrogen and progesterone without domperidone did not advance ovulation. A second experiment examined the effect of varying doses of ECP (0, 75, or 150 mg IM) followed by 1.5 g or 3 g domperidone. Estradiol cypionate had a positive effect on advancing ovulation in domperidone-treated mares in a dose-independent manner.[37]

In a recent report, transitional mares with a follicle ≥25 mm received sulpiride (1 mg/kg, q 24 h IM) until ovulation or for a maximum of 21 days.[38] Mares receiving sulpiride ovulated 25 days earlier than did control mares. Of the mares receiving sulpiride, 46% ovulated within 20 days, and 85% ovulated within 30 days versus 27% and 37%, respectively, for control mares.

Mari et al[39] compared sulpiride (1 mg/kg, q 24 h IM) and domperidone (1 mg/kg, q 24 h PO) administered to anestrous mares for 25 days during February. Of the sulpiride-treated mares, three of 10 ovulated during treatment; another three ovulated within 7 days of the last sulpiride treatment, and two more ovulated within 14 days of the last treatment, for a total of eight of 10 ovulating within 38 days of the beginning of treatment. The remaining two ovulated more than 30 days after sulpiride treatment ended. Of the domperidone-treated mares, only two ovulated during treatment, whereas eight ovulated 64 to 108 days after treatment ended. The control mares ovulated 53 to 113 days after the treatment period. The authors concluded that sulpiride was effective in advancing ovulation, whereas domperidone was effective only in some mares. It is possible that the lack of effect with domperidone was due to the dose or route of administration in this study.

Most recently, Mitcham[c] compared domperidone (1.5 g in biodegradable microparticles, IM) with varying doses (0.75 and 1.5 g, IM) of a new, non-particle formulation of sulpiride in ECP-treated mares. Treatment with domperidone took place 1 day after treatment with 100 mg ECP; treatment with sulpiride took place 1, 6, and 11 days after treatment with 100 mg ECP. Domperidone-treated mares as well as control mares received vehicle on days 6 and 11. The addition of 50 mg thyroxin 6 days before ECP treatment was also used to assess the prolactin response to domperidone or sulpiride. The prolactin response was greatest in mares treated with 100 mg ECP and 1.5 g sulpiride formula. Mares that received domperidone or 0.75 g sulpiride did not ovulate earlier compared with controls; however, of the mares treated with the higher dose of sulpiride (1.5 g), seven of the nine ovulated earlier relative to controls. Previous results showed that the effect of sulpiride on prolactin is quick but does not persist as well as the effect of domperidone. Therefore, repeated injections of sulpiride provided maximal results. The response to domperidone was greater than in control mares but significantly lower than in mares treated with sulpiride. Thyroxin did not have an effect on either the prolactin response to dopamine antagonists or the mean date of first ovulation. The preparation of sulpiride in a non-particle, slow-release formula may be less expensive (non-particle compared with microparticles) and more useful for breeders in the future.

The differing results reported in the various studies that used dopamine antagonists may be due to any number of factors, including when treatment was initiated—during anestrus or transition; the route of administration—intramuscular, subcutaneous, or oral; the dose and frequency of administration, body condition, and adjunct treatments such as pretreatment with increased photoperiod or administration of estrogen. Treatments beginning when mares are in mid-late transition probably would require a shorter duration to achieve an effect compared with treatment begun during deep anestrus. If pretreatment with estrogen can significantly affect the results, it could prove to be a valuable ad-

junct therapy. Formulations that enable effective treatment with as few injections as possible would certainly meet with greater acceptance and improve client compliance compared with treatment regimens requiring daily injections for weeks or months.

Many recent advances have been made in the ability to induce earlier cyclicity in mares; however, no single protocol yet exists to provide an infallible method of bringing mares into cyclicity earlier and shortening the duration of the vernal transition. The use of artificial lights beginning in December remains a widely used and trusted tool for inducing earlier cyclicity, but mares still have the erratic signs of transition. The use of pharmacological agents such as GnRH analogues, progestogens, and dopamine antagonists has been successful, but with variable results. The most promising of these therapies is the use of a dopamine antagonist, either sulpiride or domperidone, after pre-treatment with estradiol. Although studies have clearly shown the value of dopamine antagonists for advancing the onset of cyclicity, work remains to be done to determine the best compound to use, the best formulation, the best route of administration, and the best adjunct therapy to achieve the greatest benefit while at the same time maximizing ease of use and minimizing cost.

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Hormones and Breeding

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1. Introduction

The administration of hormones to mares during breeding management is an essential tool for equine practitioners. Proper and timely administration of specific hormones to broodmares may be targeted to prevent reproductive disorders, to serve as an aid to treating reproductive disorders or hormonal imbalances, and to optimize reproductive efficiency, for example, through induction of estrus or ovulation. These hormones, when administered exogenously, act to control the duration and onset of the different stages of the estrous cycle, specifically by affecting duration of luteal function, hastening ovulation especially for timed artificial insemination and stimulating myometrial activity in mares susceptible to or showing delayed uterine clearance. In this discussion, we address the effects and potential indications for the different hormones available to the equine practitioner working with broodmare reproduction.

2. Prostaglandin $F_{2\alpha}$

The administration of natural or synthetic prostaglandin $F_{2\alpha}$ (PGF) analogues causes interruption of luteal function by luteolysis. The diestrus stage of the estrous cycle is then shortened leading to onset of estrus. Characteristics of estrus and ovulation are not different from that occurring in natural cycles. The inherent fertility of mares is not affected by PGF treatment to induce estrus. In other words, once luteolysis takes place, whether induced by PGF treatment or occurring naturally, the events that follow (estrus behavior, ovulation and fertility) are essentially similar or minimally affected (eg, decreased signs of behavioral estrus). Duration of diestrus and interovulatory intervals are shortened after PGF administration.[1] The equine corpus luteum (CL) is responsive to PGF luteolytic effects any day after ovulation; however, only CL >5 days are responsive to one bolus injection of PGF.[2,3] Luteolysis or antiluteogenesis can be reliably achieved in CL <5 days only if multiple PGF treatments are administered. For that reason, it became a widespread practice to administer PGF as a single bolus injection (subcutaneous or intramuscular) only after 5 to 6 days after ovulation. This common practice perhaps contributed to the prevailing assumption that PGF treatments have only significant effects in mares with CL >5 days.

Mares with a CL >5 days undergo luteolysis when treated with one single injection (subcutaneous or intramuscular) of 5 to 10 mg of dinoprost tromethamine or 250 to 500 μg of cloprostenol (synthetic PGF analogue). In the United States, dinoprost tromethamine[a] is the only PGF drug approved by the Food and Drug Administration (FDA) for use in horses, although the use of the racemic formulation

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of d,l-cloprostenol[b] is very common among practitioners working with horse breeding, especially because of its longer half-life than dinoprost tromethamine salt preparations—hours versus minutes, respectively. Conversely, in other countries (Europe, South America, etc), formulations of the synthetic analogue cloprostenol are approved and available for use in horses. In those countries, the racemic d,l-cloprostenol and the more potent formulation containing only the d-enantiomer cloprostenol[c] are available; only 25 to 37.5 μg per mare of d-cloprostenol is needed to induce luteolysis in mares at least 5 days after ovulation.[4]

3. Induction of Ovulation

The ability to induce ovulation is one of the most important strategies used in breeding management. The goal of inducing ovulation in breeding management of mares is to induce ovulation within 48 hours, preferably between 24 to 48 hours after treatment. In mares monitored closely by palpation per rectum and ultrasonography, ovulation occurs within 36 to 48 hours. This fact is particularly important for mares being managed for artificial insemination with frozen-thawed semen. For optimal chances to predict outcome and increase efficacy of the treatment, regardless of the ovulation-inducing agent being used, induction of ovulation should be attempted only in mares found unquestionably to be in estrus and with a growing follicle at least $\geq$30 mm in diameter. The most common hormones used to induce ovulation include human chorionic gonadotropin (hCG) and the gonadotropin-releasing hormone agonists (GnRH), which are discussed below.

4. Human Chorionic Gonadotropin

Human chorionic gonadotropin[d] has been widely and extensively used as an ovulation-inducing agent because of its luteinizing hormone (LH)-like activity. The hormone, hCG, is produced after being extracted from urine of pregnant women. Chemically, hCG is a glycoprotein composed of an α- and a β-subunit; the α-subunit is similar to that of human follicle-stimulating hormone (FSH) and LH; however, the β-subunit, despite containing the same amino acids present in the LH β-subunit, does have an additional group of 23 amino acids and a high degree of sialylation that confers hCG a longer half-life (several hours) than that of LH (20 to 30 minutes). The hCG hormone is then lyophilized and available in the United States in vials containing 10,000 international units (IU). Despite reports of mares being treated with hCG since 1939,[5] hCG is not approved by the FDA for use in horses. Nevertheless, a single bolus dose of 1500 to 3300 IU is typically sufficient to induced ovulation in mares during estrus, with distinct estrous uterine edema and with a viable, growing follicle >30 mm.[6] Mares with more than one presumptive preovulatory follicle do not need to receive a double dose or a second injection during that estrus. It is the author's clinical experience that mares that repeatedly ovulate two or three follicles respond to a single hCG treatment similarly to mares ovulating only one follicle during estrus. Because hCG is available in 10,000-IU vials, it is common to have equine practitioners administering bolus doses of 2000 or 2500 IU to optimize the use of each vial. Although the author has successfully used vials that have been reconstituted for several weeks, it is recommended that reconstituted vials be used within 2 weeks and kept refrigerated during that period. Intramuscular or intravenous routes can be used to administer hCG in mares, but it seems, anecdotally, that fewer ovulation failures are seen in mares receiving hCG intravenously than in mares receiving it intramuscularly. For example, intramuscular doses of 5000 to 10,000 IU should be avoided. Mares do have development of antibodies to the human hormone, but the presence of antibodies does not appear to interfere with ovulation.[7] Nevertheless, some authors do recommend not treating mares with hCG more than once or twice during the same breeding season, whereas others (including this author) have used it successfully (ovulation within 48 hours from administration) in mares treated five to 10 times during the same breeding season.

5. Gonadotropin-Releasing Hormones

Administration of simple gonadotropin-releasing hormone (GnRH) analogues (equivalent to the natural decapeptide GnRH) to cycling mares has failed to consistently induce ovulation. Whereas doses of 50 to 100 μg of GnRH are sufficient to induce ovulation in cattle, doses as high as 1 to 4 mg have failed to reliably induce ovulation in mares when administered once or twice daily during estrus. Furthermore, only pulsatile administration (every hour) of GnRH has been found to be effective in inducing ovulation in the mare; this method is, however, not feasible for widespread clinical application. Since the chemical structure and peptide identification was reported in 1971, more than 2000 GnRH analogues were synthesized in the following 15 years after the 1971 report. Scientists working in research and private pharmaceutical institutions shared two main aims: (1) to create an analogue with high affinity for GnRH receptor binding and (2) to avoid or minimize enzymatic degradation by proteolysis. The GnRH molecule is susceptible to cleavage and inactivation by hypophyseal endopeptidases, especially between amino acids in position 5 and 6 and between 6 and 7. Another enzyme, carboxypeptidase, targets the bond between amino acids in positions 9 and 10. For these reasons, several GnRH synthetic analogues used in human and veterinary medicine have (1) D-amino acid substitutions in position 6 (glycine) and (2) the amino acid in position 10 (another glycine) and its amide terminal replaced by an ethylamide residue that is linked to the amino acid 9 proline.[8]

Deslorelin

In contrast with the poor or limited efficacy of simple GnRH analogues, more potent GnRH synthetic analogues such as deslorelin have a predictable effect in inducing ovulation within 48 hours when administered as intramuscular injections or by means of subcutaneous implants. Deslorelin differs from the natural decapeptide GnRH in two amino acid substitutions: in amino acid position 6, where L-glycine has been replaced with the amino acid D-tryptophan, and in position 10, where glycine and its amino terminal have been replaced by N-ethylamide.

Ovulation rates after deslorelin administration are comparable with those resulting from hCG administration.[9] Commercially, deslorelin became available as implants containing 2.1 mg of deslorelin acetate.[d] After being first available in Australia and other countries, a deslorelin implant[e] became FDA-approved and available in the United States in 1998. After being on the market for approximately two breeding seasons, it was anecdotally reported that occasionally, mares treated with the implant had prolonged interovulatory intervals, and some actually had their reproductive cyclicity downregulated for months. When exploring this issue, McCue et al[10] reported normal interovulatory intervals in mares that had their implant removed once ovulation occurred. This phenomenon was later elucidated by the fact that prolonged delivery of deslorelin would induce ovulation but its continuous release would downregulate FSH and LH secretion from the pituitary, resulting in prolonged interestrous intervals. Removing the implant after ovulation thus prevented sustained release of deslorelin. Reportedly because of problems in manufacturing logistics, this deslorelin implant[e] has not been commercially available in the United States since the early 2000s. Interestingly, side effects related to sustained deslorelin release have not been reported in Australia, where the drug originated and still remains available there and in other countries.

For several years, deslorelin was available either through importation from countries that manufactured the deslorelin implant[e] or through compounding of injectable formulations of deslorelin (1.5 mg/mL; dose 1 mL per mare administered intramuscularly). Recently, an injectable controlled release form of deslorelin acetate[f] (1.8 mg/mL) was approved by the FDA in 2010 for induction of ovulation in mares and became commercially available in the United States in 2011. It contains 1.8 mg of deslorelin acetate equivalent to 1.7 mg of deslorelin in a sucrose acetate isobutyrate and propylene carbonate matrix that promotes controlled release of the drug. This formulation has been shown to induce ovulation at comparable rates of those from hCG or deslorelin implant administrations. In the only published peer-reviewed report on the efficacy of this FDA-approved deslorelin injectable formulation, 151 of 168 (~90%) mares treated in estrus with 1.8 mg of deslorelin acetate (1 mL IM) ovulated on average 41 hours after treatment, whereas 111 of 134 mares (~83%) treated with hCG (2500 IU IV) ovulated on average 44 hours after treatment.[11]

Table 1. Comparative Rank Order Potency of Synthetic GnRH Analogues in Relation to Natural GnRH

	GnRH	Buserelin	Deslorelin	Histrelin
Relative potency	1	20	144	210

Histrelin

Once an injectable form of deslorelin became FDA-approved and commercially available, some compounding pharmacies began to accept requests to compound another synthetic GnRH analogue, histrelin (or historelin). Histrelin is an even more potent GnRH analogue than deslorelin (Table 1), but with similar clinical efficacy in inducing ovulation in horses as deslorelin or hCG. Histrelin differs from the natural decapeptide GnRH in two amino acid substitutions. In amino acid position 6, where L-glycine has been replaced with the amino acid D-histidine, and in position 10, where glycine and its amino terminal have been replaced by N-ethylamide.

Recently, a sustained-release (biorelease) formulation of histrelin was found to effectively induce ovulation within 2 days after administration.[12] The efficacy of histrelin treatment (0.5 or 1.0 mg bolus injections) in inducing ovulation within 48 hours after administration has been found to be no different from that promoted by hCG or deslorelin. In a more recent study,[13] a sustained-release (biorelease) formulation of histrelin was also found to effectively induce ovulation within 2 days after administration. There were no differences among maiden, barren, and foaling mares treated throughout the ovulatory seasons; similarly, there were no significant differences for the two doses used in that study, 0.25 and 0.5 mg, administered as single intramuscular bolus injections to mares in estrus. The overall ovulation rate at 48 hours was 93% (43/46) and 85% (44/52) for doses of 0.5 mg and 0.25 mg of histrelin, respectively. In summary, there does not appear to be differences in the efficacy, relative to ability to induce ovulation, among deslorelin, histrelin, and hCG.

Buserelin

Buserelin is another GnRH analogue that also contains modification in the amino acid position 6 by having the natural occurring L-glycine replaced by a tertiary butylated D-serine and the amino acid 10 glycine and its amino terminal replaced by N-ethylamide, which is similar to the amino terminal modification present in deslorelin and histrelin.

The moderate potency of buserelin (Table 1) perhaps may explain why a single administration of a labeled dose of buserelin (40 to 100 μg per mare) is not sufficient to induce ovulation in mares. For many years, buserelin (not available in the United States, except through compounding pharmacies) has had limited use and efficacy in inducing ovulation only when used in daily or twice-daily injections for 2 to 4 days. This protocol probably has prevented veterinarians and horse owners from favoring its use in brood mare management. In contrast with this knowledge, Levy and Duchamp[14] (2007) recently showed that much higher doses than those originally used resulted in ovulation rates comparable to that of hCG. In that study, 6 mg of buserelin was injected subcutaneously to cyclic mares, resulting in 89% to 95% ovulation rates that did not differ from hCG administration (86%). Considering the relative potencies of deslorelin, histrelin, and buserelin (Table 1), we calculate that buserelin is at least 7 and 10 times less potent than deslorelin and histrelin, respectively. This may partially explain why in this study a buserelin dose ~60 times greater (6000 μg versus 100 μg) than that recommended in veterinary commercial preparations of injectable buserelin[g] was able to effectively promote ovulation in treated mares at a similar rate reported for hCG, deslorelin, and histrelin.

Veterinarians should use the FDA approved products for this indication. Certain compounded products can vary significantly in potency and stability. Consequently results may vary substantially. There can also be legal and ethical issues with using a compounded product when there are FDA approved products available in the appropriate dosage form and with the appropriate indication.

6. Ecbolic Hormones for Treatment of Delayed Uterine Clearance

In general, the use of ecbolic hormones to correct deficiencies or weakness in myometrial contractions commonly seen in mares with delayed uterine clearance and susceptible to endometritis are safely initiated at 4 to 8 hours after artificial or natural insemination to avoid interferences with sperm transport that could affect fertility. These treatments are then continued at the discretion of the prescribing veterinarian; ecbolics are often administered twice daily or more often if necessary and depending on the recorded response for an individual mare. Commonly used ecbolics in broodmares include oxytocin and PGF.

Oxytocin

Oxytocin is a nonapeptide synthetized in the magnocelullar neurons in the hypothalamus that extend their axons into the neurohypohysis, where it is stored until released into the blood stream for biological action. Oxytocin has several known physiologic effects, such as milk ejection, promoting maternal behavior, and inducing myometrial contractions. Because delayed uterine clearance is commonly seen in mares with persistent mating-induced endometritis, oxytocin is one important therapeutic option for treating abnormal intrauterine accumulation of fluid. Despite oxytocin's relatively short-life of ~7 minutes, it appears that its biological effect far exceeds this time. For that reason, treatment protocols varying from two to four daily treatments have been shown to be relatively efficacious in promoting uterine clearance. Boluses doses of 10 to 20 IU administered subcutaneously, intramuscularly, or intravenously are frequently used to treat mares with mating-induced intrauterine fluid accumulation. Oxytocin treatments are administered during estrus before or after ovulation, with treatments scheduled to not coincide with breeding to avoid interfering with sperm transport, especially if given shortly after artificial insemination, for example, <4 hours. Recently, our laboratory reported on the pharmacokinetics of carbetocin, a long-acting oxytocin analogue.[15] Carbetocin has a half-life of ~17 minutes. Carbetocin is available and approved for use in horses in several countries but not in the United States. Despite its longer half-life than oxytocin, multiple daily treatments may still be required for optimal effects in promoting uterine clearance as is required with oxytocin or PGF. A direct comparison between oxytocin's and carbetocin's ability to promote uterine clearance has not been reported for in vivo treatment in mares. Currently, it not known whether the reported prolonged half-life of carbetocin would translate into a greater and/or more productive biologic response than oxytocin in mares that may require prolonged myometrial stimulation. Steckler et al[16] have recently reported on the results of a comparison of oxytocin versus carbetocin in eliciting contractions in ex vivo uterine tissues collected from mares at different reproductive stages (eg, anestrus, estrus, and diestrus). The results indicated that the effect of oxytocin on equine myometrial tissue was greater during anestrus and diestrus than during estrus, whereas carbetocin appeared to elicit myometrial contractions independent of the reproductive status; however, there were no differences between their ability to induce myometrial contractions in the ex vivo strips of myometrial tissues.

Prostaglandin $F_{2\alpha}$

In addition to being known for its luteolytic actions, PGF is also an effective promoter of myometrial contractions. Because of its longer half-life than oxytocin, especially the synthetic PGF analogue cloprostenol (~2–3 hours), PGF has been favored by equine practitioners, especially for use in mares that anecdotally appear to be refractory or do not respond appropriately to oxytocin treatment. Cloprostenol does elicit less vigorous myometrial contractions than oxytocin but for prolonged periods. For example, one study reported that oxytocin induced strong myometrial contractions that lasted ~30 minutes,

whereas cloprostenol induced low-amplitude myometrial contractions that lasted 4 to 5 hours. This sustained effect is particularly desirable to enhance lymph flow and consistent intraluminal evacuation of accumulated fluid (inflammatory transudate). Despite its indisputable efficacy as an ecbolic hormone, the use of PGF during estrus, especially in the early post-ovulatory period, was found to transiently affect luteal function. Some studies confirmed this effect on luteal function that was followed by a resurgence in CL function.[17,18] It is unknown whether this early effect in suppressing luteal function would affect the fertility of treated mares. For this reason, if PGF is to be used as an ecbolic to treat delayed clearance during estrus and early postovulatory period, it is important that equine practitioners remain cognizant of its effects on luteal function. Most mares so treated should have resurgence in luteal function and not have deleterious effects on their luteal function and subsequent fertilities.[19] Moreover, our laboratory has recently reported that the effect of PGF administration on early luteal function can induce luteolysis or antiluteogenesis, depending on the duration of treatment after ovulation and on dosage used.[3] For these reasons, treatments with PGF during estrus should not continue beyond 24 hours after ovulation. Whenever indicated, low doses of PGF (eg, 62.5–125 μg cloprostenol or 1.25 mg dinoprost), which are capable of inducing uterine contractions, are recommended for treating mares with delayed uterine clearance.

7. Modulation of Mating-Induced Endometritis With Steroids

Endometritis after artificial insemination or mating is one important cause of infertility especially in mares susceptible to persistent mating-induced endometritis. In addition to therapies aimed at combating endometritis by use of intrauterine or systemic antibiotic treatment and stimulation of myometrial function through administration of ecbolic hormones (mainly oxytocin and PGF), another strategy is to control or modulate the exacerbated inflammation response seen in these mares. Dell'Aqua et al[20] first reported on the efficacy of prednisolone treatment of mares during estrus that benefited from the glucocorticoid modulatory effects on barren mares afflicted with endometritis. A significantly higher pregnancy rate was seen in barren mares treated 5 times with 0.1 mg/kg of prednisolone acetate (~50 mg per mare) administered every 12 hours with four treatments occurring before artificial insemination (AI) and one at AI. In 2008, Morris reported promising results used the same protocol described by the Dell'Aqua, except she used a dose of 200 mg of oral prednisolone per mare. More recently, Bucca et al,[21] reported on the use dexamethasone given as a single bolus administration of 50 mg per mare at time of breeding. In that study, the authors concluded that dexamethasone administered at the time of artificial insemination was safe and effective for modulating persistent mating-induced endometritis in susceptible mares; the glucocorticoid treatment decreased endometrial edema, accumulation of intraluminal uterine fluid and its turbidity, without altering the amount of polymorphonuclear cells seen in preparations obtained from endometrial cytology. Ferris and McCue[22] studied the effects of multiple (twice daily for 5 days) glucocorticoid treatments (prednisolone or dexamethasone in mares during early estrus. They reported that mares treated with dexamethasone showed reduced uterine edema and ovulation rate (40% versus 83%, respectively) than mares treated with prednisolone. In addition, only two of five dexamethasone-treated mares had unaltered LH hormonal profiles, whereas five of six mares treated with prednisolone had LH surges within normal limits. The differences noted between dexamethasone and prednisolone may derive from their differences in relative potencies in relation to cortisol (dexamethasone = 30; prednisolone = 4). Nevertheless, it is unlikely that a single treatment with dexamethasone as prescribed by Bucca and Carli[21] would result in ovulation failure as only mares with multiple, daily treatments with dexamethasone have been shown to have altered endogenous LH release that resulted in significant ovulation failures. It is important, however, to ensure that mares being treated with glucocorticoids during estrus are also treated with ovulation-inducing agents to prevent any potential negative effects of glucorticoid agents in the hypothalamic-hypophyseal axis that may result in anovulation. Bucca et al[23] provided supporting evidence to the efficacy of associating induction of ovulation with glucocorticoid treatment in mares susceptible to persistent post-mating endometritis. Mares treated with a single bolus intravenous injection of 50 mg of dexamethasone within 1 hour of breeding and concurrent induction of ovulation with 1500 IU of hCG exhibited normal ovulation rates (~97% of mares ovulated within 48 hours). The minimal dose of prednisolone and dexamethasone to combat inflammation in mares during estrus has yet to be determined.

8. Conclusions

Several pharmacologic agents are now available for use in the breeding management of broodmares. Strategic utilization of hormones to induce estrus and ovulation and to modulate/prevent inflammatory processes in mares susceptible to mating-induced endometritis and delayed uterine clearance can significantly affect the outcome of breeding by increasing the odds of timed ovulation and pregnancy.

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[a]Lutalyse, Pfizer Animal Health, Kalamazoo, MI 49001.
[b]Estrumate, Merck Animal Health, Summit, NJ 07091.
[c]Genestran, FORTE Healthcare Limited, Naul, Dublin, Republic of Ireland.
[d]Chorulon, Merck Animal Health, Millsboro, DE 19966.
[e]Ovuplant, Zoetis Canada, Kirkland, QC H9J 2M5 Canada.
[f]Sucromate is a product of and manufactured by Thorn BioScience, Louisville, KY, a division of CreoSalus; it is marketed by Bioniche Animal Health USA, Inc, Athens, GA 30601.
[g]Receptal®, Intervet Ltd., Upper Hutt, New Zealand.

Use of Prostaglandin $F_{2\alpha}$ for Controlling the Mare's Estrous Cycle

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1. Introduction

Prostaglandins belong to a group of modified long-chain fatty acids containing 20 carbons called eicosanoids. The cyclooxygenase pathway uses prostaglandin synthases to convert arachidonic acid into prostaglandins. Arachidonic acid is available through the hydrolysis of phospholipids present in the cell membrane. The breakdown of membrane phopholipids is catalyzed by the enzyme phospholipase A. Two isoforms of prostaglandin synthase exist: constitutive (cyclooxygenase-1) and inducible (cyclooxygenase-2). Systemic administration of prostaglandins (mainly prostaglandin $F_{2\alpha}$ [PGF] in animals) is associated with side effects affecting the central nervous system (incoordination, stupor, and ataxia) and the vascular system (contraction of smooth muscle of organs such as the stomach, intestines, and urinary bladder).

The use of injectable preparations of PGF has revolutionized the breeding management of horses and cattle since its identification as the main luteolytic hormone. Pharmacokinetics of PGF after intravenous administration of 5 mg per mare (large mixed breeds of large ponies) has been recently described as follows: apparent plasma clearance, 3.3 ± 0.5 L/h/kg; distribution half-life of 1.57 ± 0.26 minutes; elimination half-life of 25.9 ± 5.0 minutes; and maximum plasma PGF concentration of 249.1 ± 36.8 ng/mL.[1] The original studies pointed out that mares appeared to be more sensitive to exogenous PGF than cows. Indeed, an in vitro study has shown the affinity of equine luteal cell membrane preparations for PGF to be approximately 10 times greater than that for bovine luteal cell membrane preparations.[2] The relatively high affinity of mare corpus luteum (CL) to binding of PGF along with the relatively slow metabolic clearance documented in mares account for the greater sensitivity of mare CL to the luteolytic effect of PGF when compared with other domestic species.

The luteal phase of the equine estrous cycle can be reliably shortened by the administration of PGF, allowing mares to return to estrus at a relatively predictable time (on average 2–5 days after PGF administration). In horses, a single treatment with PGF (intramuscular or subcutaneous) will induce complete luteolysis if administered at least 5 to 6 days after ovulation. This fact led to the prevailing assumption that the CL is not responsive to PGF luteolytic effects before it is at least 5 days old, despite the fact that some initial studies reported that some mares were responsive to luteolytic effect

NOTES

of PGF administration when treated on day 3 after ovulation. In the United States, the natural analogue dinoprost tromethamine[a] is the only Food and Drug Administration–approved PGF for use in horses, although equine practitioners commonly use the synthetic analogue cloprostenol in breeding management, mostly owing to the longer half-life than its natural analogue. A review of the effects of PGF on luteal function and characteristics of the induced estrus and ovulation is presented in the subsequent sections. The use of PGF as an abortifacient and ecbolic for breeding management will not be presented.

2. Effects of PGF Administration on the Mare's Reproductive Cycle

Natural luteolysis begins approximately 14 days after ovulation in mares. In the decade of the 1970s, several studies investigated the effects of PGF treatment on blood progesterone concentration profile and effects of the length of diestrus and interovulatory intervals.[3–5] Most of these studies were based on examinations of serial blood samples taken before and after treatment with PGF or on the recording of the length of interovulatory intervals in treated and control mares. Studies on subsequent PGF-induced estrus, follicular dynamics, and ovulation were based mainly on findings of serial reproductive examinations by palpation per rectum. More recently, a significant wealth of information on the characteristics of luteal development and regression, follicle growth, and ovulation after PGF induced luteolysis became available with the advent of transrectal ultrasonography. The information gained with ultrasonography studies on mare reproduction contributed to the understanding of PGF actions on the mare's reproductive cycle and tract.[6,7]

Soon after PGF was shown to be the uterine luteolysin in cattle, sheep, and rats, Douglas and Ginther[3] published convincing evidence that exogenous (subcutaneous or intramuscular) or intrauterine administration of PGF had also luteolytic effects in mares. Since then, PGF and its synthetic analogues have been widely used for intensive broodmare management.[7] In the study by Douglas and Ginther,[4] mares received PGF on day 6 after ovulation. Previous studies demonstrated that intrauterine saline solution infusions 6 days after ovulation shortened the mare's estrous cycle. The shortened cycle was denoted by luteal activity interruption that terminated diestrus.[4] In that study, all mares treated with 1.25, 2.5, 5.0, or 10.0 mg of PGF (intramuscular) had shorter diestrus and shorter interovulatory intervals than did control mares (not treated with PGF). After that report, several other studies confirmed that PGF treatments not only shorten diestrus but also shorten interovulatory intervals. Despite the fact some mares may undergo complete luteolysis when treated on day 3 after ovulation, maximal response to one single-bolus intramuscular injection is expected when at least 5 days have elapsed from ovulation. Anecdotally, some equine practitioners report that whenever the day of ovulation is unknown, daily treatments of PGF are prescribed until treated mares show signs of behavioral estrus.

3. Luteolytic Doses of PGF Preparations

Dinoprost Tromethamine

For PGF tromethamine salt (PGF tham salt) preparations, 1.34 mg of the salt equals 1 mg of free acid PGF. Douglas and Ginther[3] reported that doses of 1.25, 2.5, 5.0, and 10.0 mg of PGF administered intramuscularly shortened the luteal phase of the estrous cycle. Mares in all treatment groups were in estrus 3 to 4 days after treatment. A single bolus dose of 1.25 mg of dinoprost tromethamine[a] per horse mare (~2.8 μg/kg for an average 450-kg mare) when administered between days 6 and 12 after ovulation has been shown to be luteolytic and induce normal ovulatory estrus periods, which in turn were followed by normal luteal function (diestrus).[8] Even doses as low as 0.5 mg per mare (~1.1 μg/kg) has been shown to affect luteal function; however, complete luteolysis (21/21 mares) was only achieved when mares were treated twice, 24 hours apart.[9] In that study, this low dose did not induce common side effects (sweating, colicky behavior) generally associated with PGF treatment. Most commercial preparations of dinoprost tromethamine, however, recommend a single intramuscular or subcutaneous bolus administration of 5 to 10 mg per mare (~11.1–22.2 μg/kg).

Cloprostenol

In contrary to several other countries, cloprostenol formulations are not Food and Drug Administration–approved for use in horses in the United States. Nevertheless, cloprostenol is widely used in the United States by equine practitioners because of the longer half-life and association with fewer side effects than dinoprost tromethamine. Cloprostenol is available as two optically active isomers (enantiomers), combined, d-cloprostenol and l-cloprostenol. The recommended luteolytic doses of these synthetic analogues are much lower than that recommended for the natural analogue dinoprost tromethamine. Luteolytic doses for d-cloprostenol are further lower than that needed for d,l-cloprostenol–induced luteolysis.[10] The dosage difference between these two cloprostenol analogues is explained by the fact that only the d-enantiomer is pharmacologically active (luteolytic). Most popular preparations of cloprostenol in the United States use the racemic mixture[b] (d- and l-enantiomers) at a dose of 250 to 500 μg per mare. In one study, doses as low as 25 μg of d,l-cloprostenol per mare successfully induced luteolysis.[11] In several countries, the more potent preparations with only the active d-cloprostenol enantiomer[c] are also available and labeled for use in horses. In a recent report, the bolus dose of

37.5 μg of d-cloprostenol[c] induced complete luteolysis, similar to that in mares receiving 250 μg of a d,l-cloprostenol preparation.[12] The recommended labeled doses for d-cloprostenol and d,l-cloprostenol are 37.5 μg per mare (0.5-mL injection volume) and 250 μg (1-mL injection volume), respectively, administered subcutaneously or intramuscularly.

4. Luteolytic Effects of PGF and Stage of the Estrous Cycle

The results presented in the early studies in the 1970s provided the basis for the assumption that PGF formulations would not induce luteolysis or affect CL function if administered before day 5 or 6 after ovulation. Interestingly, some of these studies reported that some mares actually responded to PGF-induced luteolysis when treated on day 3 after ovulation[3]; however, the notion that the early CL was not responsive to PGF administration remained ingrained in the scientific and veterinary professional community. In 1974, Thompson and Witherspoon[13] briefly reported another phenomenon that has recently gained attention: the ability of PGF to induce partial luteolysis followed by resurgence in CL function characterized by a transient increase in concentrations of blood progesterone. In that study, two mares receiving a relatively low dose of a synthetic PGF analogue 9 days after ovulation began to have a decrease in concentrations of plasma progesterone at 12 hours after PGF treatment, followed by a resurgence in progesterone concentrations at 48 hours after treatment; progesterone concentrations then remained at 30% to 50% of that before PGF treatment. More recently, 32 years from that initial report, Bergfelt et al[14] (2006) compared the pattern of luteolysis after PGF treatment as a single bolus injection on day 3 after ovulation with that of mares treated on day 10. In the day 3 group, 75% (12/16) of mares had CL resurgence. Among those, six mares with "minor" progesterone resurgence had treatment-to-ovulation intervals similar to that in control mares. In summary, CL resurgence after PGF treatment results in partial luteolysis of the CL. Partial luteolysis is evident by decreasing concentrations of blood progesterone and followed by CL function resurgence. The resurgence is denoted by a moderate increase in progesterone concentrations. Partial luteolysis followed by CL resurgence may occur after administration of sub-luteolytic boluses doses of PGF during mid diestrus,[13,15,16] or after administration of single injections at day 3 after ovulation.[14]

5. Effects of Exogenous PGF on Steroid and Gonadotropin Secretion

Administration of PGF in mares with a functional CL >5 days is followed by functional luteolysis (significant decrease in progesterone) 24 hours after treatment that is, however, preceded by an immediate, transient rise in progesterone shortly after PGF treatment. Noden et al[17] reported that functional luteolysis was preceded by a transient increase in progesterone, estradiol, and leuteinizing hormone at 10, 30, and 60 minutes after PGF treatment of diestrual mares. In a more recent study by Ginther et al,[18] administration of a single luteolytic intravenous bolus of PGF resulted in an immediate increase in circulating progesterone concentrations within 10 minutes after the bolus injection, accompanied by an increase in concentrations of follicle-stimulating hormone, leuteinizing hormone, and cortisol. Conversely, mares infused with PGF for 2 hours, mimicking a natural pulse of endogenous PGF action, did not show increases in the same hormones; however, both treatments, bolus injection and infusion, resulted in similar luteolytic effects. These effects on steroids and gonadotropin secretion associated with supraphysiologic doses of PGF may partially explain the results of one study that found that mares treated in estrus with a synthetic PGF, fenprostalene, had shorter estrus-to-ovulation intervals than did control mares.[19]

6. PGF Treatment and Antiluteogenesis

Recently, it has been reported that luteolysis or prevention of luteal formation may be accomplished with PGF administration beginning as early as the day ovulation is detected. This effect is dependent on the dose and frequency of PGF treatments. On the basis that the early developing CL <5 days is actually responsive to luteolytic effects of PGF, a series of experiments conducted in our laboratory produced data that support the hypothesis that the early developing CL is indeed responsive to exogenous PGF as early as within the first 24 hours from ovulation.[20,21] Because of this early luteolytic responsiveness to PGF administration before the CL is fully functional, we named this phenomenon as (PGF-induced) "antiluteogenesis." Mares treated once or twice daily for 3 days with 2.5 or 10 mg of PGF dinoprost failed to show a significant rise in concentrations of plasma progesterone during the treatment period. Approximately 60% of mares treated twice daily for 3 days with 10 mg of PGF had complete luteolysis; all mares receiving once-daily 2.5 mg of PGF for 3 days showed CL resurgence. Therefore, the antiluteogenesis effect of PGF is dependent on the dose and frequency of PGF treatments.

7. Clinical Applications of PGF in Broodmare Management

Use of PGF to Induce Luteolysis and Return to Estrus

Termination of the luteal phase ("short-cycling") with exogenous PGF may be attempted for planned breeding of a single mare or as an approach to synchronize estrus and ovulation in a group of mares. If reproductive examinations with palpation per rectum and transrectal ultrasonography are available, the predictability of onset of estrus and ovula-

tion increases. Prediction of the next ovulation in the PGF-induced estrus is not predictable as it is the return to estrus. For example, it has been shown that the diameter of follicles present in the ovaries at the time of PGF treatment may influence when the mare would ovulate.[22] When a relatively large follicle (≥35 mm) is present at the time of PGF administration, the onset of estrus and ovulation will depend on the follicular status (growing phase versus undergoing atresia). Accordingly, mares with follicles approaching the diameter of preovulatory follicles may come in estrus and ovulate within 2 to 5 days after PGF treatment, whereas the mean interval from treatment to ovulation in mares during mid diestrus and with follicles <25 mm may vary from 7 to 12 days from treatment.[22] For example, in some extreme instances, mares will ovulate in 2 to 3 days; mares ovulating within 48 hours from PGF treatment often show no signs of behavioral estrus. Conversely, large follicles present at the time of PGF treatment may be already undergoing atresia and will slowly regress, and the mare may not ovulate until 10 to 14 days after the treatment. In most cases, however, mares will come into estrus and the large follicles at the time of PGF treatment will continue to grow and ovulate within 4 to 6 days after PGF treatment.

Obviously, the prediction of PGF-induced estrual events requires that treated mares have an active corpus luteum at the time of administration. If reproductive examination is not available, horse owners may be instructed to administer a single dose of PGF 5 days after the mare ceases behavioral signs of estrus, or, alternatively, if teasing is not feasible, daily administration of a single PGF treatment may be prescribed until the mare shows signs of estrus or a reproductive examination by a veterinarian becomes available. Another alternative if veterinary assistance, or teasing information were not available, would be to recommend administration of a single dose of PGF at any given day and to repeat it in 5 days if the mare is not observed to be in estrus.

Use of PGF in Postpartum Mares

Several factors associated with complications during foaling could compromise the fertility of the mare's foal heat. For most mares with dystocia or retention of the fetal membranes, it may be prudent to not breed on the first estrus after parturition (foal heat). In this scenario, instead of having horse owners waiting for mares to come into their second postpartum estrus ("30-day heat"), one strategy would be to treat mares with PGF approximately 5 to 7 days after ovulation in the foal heat.

Use of PGF in Mares With Prolonged Luteal Phases

Occasionally, mares may have prolonged diestrus periods because of the presence of a persistent CL. Persistent CLs may occur in mares that failed to express their endogenous luteolytic mechanism (rare), or, more commonly, in mares that have early embryonic loss after maternal recognition takes place. In general, prolonged diestrus is often associated with another unique phenomenon of the mare's estrous cycle, the diestrus ovulation. Prolonged diestrus is diagnosed as a diestrus period lasting more than 16 days after ovulation. A single luteolytic dose of PGF (5–10 mg dinoprost) should induce mares to return to estrus.

Use of PGF in Estrus Synchronization

One of the most basic methods to attempt estrus synchronization is to treat mares with PGF and repeat the treatment approximately 2 weeks from the first injection. If teasing is available, mares can then be teased every other day beginning 2 days after PGF treatment. The efficacy of the use of PGF in estrus synchronization programs is greatly enhanced with the concomitant use of progestagens and estrogens.

8. Non-Reproductive Effects Associated With PGF Administration

In general, prostaglandins have significant effects on vascular and non-vascular smooth muscle, the central nervous system, and carbohydrate and lipid metabolism.[23] The administration of exogenous PGF is relatively safe, and doses 20 to 40 times greater than the therapeutic dose (typically 5–10 mg of dinoprost) do not elicit toxic effects.[24] Even doses up to 800 mg were not fatal to mares despite being associated with intense side effects such as recumbency; in that study, severe side effects subsided by 4 to 5 hours after PGF overdose treatment. This increased sensitivity is also reflected by the appearance of side effects after administration of a conventional luteolytic dose in mares in 20% to 40% of mares treated with PGF. Sweating, restless behavior, diarrhea, or even colic-like signs are commonly observed in mares but not in cattle. One of the most common side effects is a pronounced sweating seen within minutes after PGF administration. The results of most research studies indicate that equine sweating occurs by stimulation of adrenoreceptors on the sweat gland cells.[25] Adrenaline-induced sweating is primarily mediated by $\beta 2$ adrenoreceptors. Horses given PGF intramuscularly sweat but do not shiver, although shivering occurs in horses treated with adrenaline. This may explain why rectal temperature significantly decreased in horses after PGF administration.[24] Because concentrations of plasma adrenaline and noradrenaline become elevated after administration of PGF, it has been accepted that PGF-related sweating is associated with release of adrenaline from the adrenal medulla. Some mares may also have abdominal discomfort resembling colic-like symptoms. Abdominal discomfort is a result of hypergastromotility. Occasionally, some mares also show locomotor incoordination and ataxia.

These side effects typically subside within 20 to 30 minutes after PGF treatment. The appearance and duration of these aforementioned side effects appear to vary among mares. It is important to note that these side effects are dose-dependent and typically subside within the first hour after PGF treatment. Irvine et al[9] reported that the administration of two low doses of PGF 24 hours apart did not elicit any appreciable side effects, including elevation in heart rate.

9. Conclusions

Manipulation of the mare's estrous cycle with PGF is an important strategy in the breeding of mares. The CL is sensitive to PGF treatment throughout the whole estrous cycle. A single bolus injection of PGF can reliably induce luteolysis when administered in mares with a CL >5 days. Serial injections of PGF for several days beginning (q 12 h or q 24 h) as early as within 24 hours from ovulation will prevent CL formation (antiluteogenesis), as evidenced by the absence of a rise in progesterone. Not only diestrus is shortened in mares treated with PGF but interovulatory intervals are also reduced in relation to normal, untreated cycles. Estrus and ovulation occurring after PGF treatments are normal and the inherent fertility of mares treated is not affected.

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[a]Lutalyse, Pfizer Animal Health, Kalamazoo, MI 49001.
[b]Estrumate, Merck Animal Health, Summit, NJ 07901.
[c]Genestran, FORTE Healthcare Limited; Naul, Dublin, Republic of Ireland.

Prolonging Function of the Corpus Luteum to Suppress Estrus in Mares

Dirk K. Vanderwall, DVM, PhD, Diplomate ACT

The orally active synthetic progestin altrenogest is widely considered to be the "gold-standard" method of inhibiting estrous behavior in mares. However, its expense, need for long-term daily administration, safety concerns for personnel during handling, and increased public scrutiny regarding the use of exogenous steroids in performance horses have collectively prompted interest in the development of practical methods of prolonging corpus luteum (CL) function, which allows continued secretion of endogenous progesterone to keep mares out of heat naturally. Placement of an intrauterine glass ball has been the most widely used method of prolonging CL function, but variable efficacy and the potential for deleterious consequences associated with them (if not eventually removed), have stimulated interest in alternative methods of prolonging CL function. Currently, oxytocin treatment appears to be the most practical and efficacious alternative method of prolonging CL function. Additional methods include inducing a late-diestrus ovulation, intrauterine infusion of plant oils, and establishment of pregnancy followed by manual reduction of the conceptus after day 16 of gestation. Author's address: Department of Animal, Dairy and Veterinary Sciences, School of Veterinary Medicine, Utah State University, Logan, UT 84322; e-mail: dirk.vanderwall@usu.edu. © 2013 AAEP.

1. Introduction

A common complaint of horse owners, trainers, and veterinarians is variable performance of mares that is related to the estrous cycle.[1,2] In a survey of more than 750 veterinarians, approximately 90% had the clinical impression that the estrous cycle affected the performance of mares, and the most frequently reported clinical sign associated with an effect of the reproductive cycle on performance was attitude change, whereas other signs included tail-swishing, difficulty to train, squealing, "horsing," excess urination, kicking, and a decrease in performance.[1] As the preceding list of objectionable behaviors suggests, not all problematic behaviors are necessarily associated with estrus. In addition, it is important to note that some behaviors displayed by mares that are thought to be associated with estrus are in fact not estrous behaviors. They are instead signs of 1) submissive behavior, 2) urogenital discomfort, or 3) stallion-like behavior.[3,4] Of these, submissive behavior may be most easily confused with estrous behavior. Submissive behavior includes leaning away from perceived threats, swishing/ringing the tail, and actively squirting urine, which collectively can give the impression of estrus, whereas true estrous behavior is manifested by leaning toward the stallion (or other stimulus), a relaxed, lifting motion of the tail, stationary/squat-

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ting stance, and passive urination (full stream or small amounts in dribbles).[4] Urogenital discomfort may be the result of cystitis or renal disease, whereas stallion-like behavior may be associated with neoplasia of the ovaries or administration of exogenous anabolic steroids.

Because numerous factors (both reproductive and nonreproductive) can adversely affect performance, when evaluating an owner/trainer complaint of an estrous cycle-related behavior/performance problem in a mare, the first step is to determine if the problematic behavior is or is not related to a specific phase of the estrous cycle. To thoroughly evaluate the mare, additional expertise may be needed in the form of consultation with or referrals to behavior and/or reproduction experts, because a systematic team approach to evaluating and solving the problem may be most successful.[5]

If problematic behavior is consistently associated with estrus, it is necessary to determine if the underlying cause is physical or behavioral. Some mares that exhibit performance problems during estrus may be having pain in the periovulatory period that can vary from sensitivity to weight and/or manipulation of the back (caused by tack and/or rider) to overt colic-like symptoms.[6–9] Mares that have back pain and/or colic-like symptoms in the periovulatory period may benefit from the use of an ovulatory agent to reduce the time a large pre-ovulatory ovarian follicle is present or complete suppression of cyclical reproductive activity. In contrast to mares with an underlying physical cause of performance issues during estrus, some mares display such profound signs of estrus that the behavior itself impairs performance. For example, even under saddle, some mares may "break down" and show estrus in response to being around other horses and/or other stimuli.[4] For these mares, suppression of estrus is clearly warranted. In other mares, the condition may be much more subtle, causing owners and trainers to report that the mare is less cooperative or attentive during estrus, prompting them to pursue estrus suppression.[2] It is also common to suppress estrus when the signs of estrous behavior are simply perceived to be associated with performance problems or to preemptively block the behavior to preclude the possibility of an adverse effect of estrus on performance. When suppression of estrous behavior is desired, the following general methods have been used in mares: 1) administration of exogenous progesterone or synthetic progestins, 2) extending the duration of corpus luteum (CL) function, 3) downregulating ovarian follicular activity, and 4) ovariectomy (for complete review, see Vanderwall and Nie[10]).

When discussing methods of suppressing estrus, it is important to note that mares are unique among domestic animals, because in addition to ovarian-derived estrogen-induced signs of estrous behavior, many seasonally anovulatory and ovariectomized mares exhibit paradoxical estrous behavior associated with hormone secretion from the adrenal cortex.[11,12] The intensity of this type of "unseasonable" estrous behavior can be equivalent to the behavior intact cycling mares display during the initial and terminal days of estrus but is generally less intense than the behavior displayed near ovulation.[11] It has been postulated that behavioral receptivity to a stallion outside the breeding/ovulatory season that is independent of ovarian estrogen secretion may have developed as a means of maintaining social bonds between a harem stallion and his mares.[11,13] This phenomenon has important implications for the clinical management of estrous behavior in mares, since simply suppressing ovarian follicular activity or removing the ovaries may not ensure complete elimination of estrous behavior.

2. Exogenous Progesterone/Progestins

Historically, the use of exogenous progesterone/progestins has been the most widely used method of suppressing estrous behavior in mares. It was first demonstrated in the 1960s that daily intramuscular administration of 0.2 mg/kg progesterone in oil effectively suppressed signs of estrus in mares.[14] Although progesterone in oil is available from compounding pharmacies, the need for daily administration and the potential for soreness at the site of injection are limitations to its use. An alternative to daily administration of progesterone in oil is a compounded long-acting injectable formulation of progesterone containing a total dose of 1.5 g progesterone that will maintain blood levels of progesterone above 1.0 ng/mL for approximately 10 days,[15] which is a sufficient level of progesterone to block estrous behavior[14,16]; however, the potential for soreness at the injection site is a limitation to its use in performance horses.

In contrast to native progesterone, the orally active synthetic progestin altrenogest[a,b] is approved for suppressing estrus in mares and is widely considered the "gold-standard" method of inhibiting estrous behavior. Daily oral administration of altrenogest at a dose of 0.044 mg/kg is very efficacious for suppressing estrus in mares[17,18]; however, its expense, need for long-term daily administration, and safety concerns for personnel during handling and administration[19] are drawbacks to its use. As an alternative to oral administration, it was recently reported that intramuscular administration of a compounded preparation containing 225 mg or 450 mg of altrenogest in a sustained-release vehicle blocked estrous behavior for approximately 12 and 15 days, respectively, and administration of 500 mg altrenogest in microparticles suppressed estrous behavior for approximately 30 days.[20] Veterinarians should use FDA approved products for this indication. Certain compounded products can vary significantly in potency and stability. Consequently results may vary substantially. There can also be legal and ethical issues with using a compounded product when there are FDA approved products

available in the appropriate dosage form and for the appropriate indication.

Although there have been anecdotal reports on the use of synthetic progestins other than altrenogest for estrus suppression in mares, none have been found to be efficacious when rigorously tested.[10] Because of the ineffectiveness of other synthetic progestins, altrenogest remains the primary exogenous hormone used for suppression of estrus; however, because it is a steroid hormone, it is facing increased scrutiny because of public concern about the use of steroids in performance horses. Therefore, finding effective alternatives to the use of exogenous steroid hormones is particularly prudent at this time.

3. Prolonging Function of the Corpus Luteum

An alternative method of suppressing estrus that does not require administration of exogenous progesterone/progestins is prolonging function of the CL, which allows continued secretion of endogenous progesterone to suppress estrus naturally. In nonpregnant mares, the CL secretes progesterone for approximately 2 weeks after ovulation (ie, the duration of diestrus) and then stops functioning when the endometrium secretes prostaglandin F2α (PGF2α) during luteolysis.[21] As a result of luteolysis, progesterone levels fall below 1.0 ng/mL and the mare returns to estrus. Development of therapeutic strategies for prolonging function of the CL (ie, maintaining progesterone $>$1.0 ng/mL) as a means of long-term suppression of estrus has gained increasing interest because it obviates the need for administration of exogenous progesterone/progestins to mares. The remainder of this review will discuss various methods of prolonging CL function for suppression of estrus in mares.

4. Intrauterine Glass Ball

The most common method of prolonging CL function in mares has been intrauterine insertion of a glass ball (ie, marble). Nie et al[22] reported that placement of a 25- or 35-mm sterile glass ball into the uterine body immediately after ovulation resulted in prolonged CL function in seven of 18 mares (39%) that retained the glass ball after insertion (six of 12 mares expelled the 25-mm glass ball soon after insertion). In mares with prolonged CL function after placement of the glass ball, CL function was maintained for approximately 90 days, during which time progesterone levels remained above 1.0 ng/mL and estrous behavior was not exhibited. In nontreated control mares, spontaneous prolongation of CL function occurred in four of 32 mares (13%). Although placement of a glass ball appeared to be an efficacious means of blocking estrous behavior for an extended period of time, it should be noted that in addition to the 11 mares that retained the glass ball and never developed extended CL function (i.e., continued to cycle normally while the glass ball was in the uterine lumen), three of the seven mares that received a glass ball had one or two estrous cycles of normal duration after placement of the glass ball before CL function was prolonged. Therefore, on a "per-cycle" basis, the incidence of prolonged CL function was only 11% (7/62 cycles) in the mares that received a glass ball compared with 8% (4/50 cycles) in the nontreated control mares, which was not significantly different between groups.

More recently, Rivera del Alamo et al[23] examined the effect of intrauterine placement of a 20-mm, water-filled polyproplylene ball on the duration of CL function in mares with the specific objective of investigating two potential mechanisms by which CL function is extended: 1) the intrauterine device induces mild endometrial inflammation that completely blocks or markedly attenuates PGF2α secretion (ie, prevents high-magnitude luteolytic pulses) or 2) the physical presence of the device (movement and/or contact with the endometrium) directly mimics the inhibitory effect of a conceptus on PGF2α secretion. Corpus luteum function was extended in nine of 12 mares (75%) that received the intrauterine device with an average duration of 57 days compared with none of 12 control mares in which the average duration of CL function was 16 days. In six of the nine mares with extended CL function, small accumulations of intrauterine fluid ($\leq$10 mm $\times$ 20 mm) were identified during the luteal phase, but no neutrophils or bacteria were recovered on uterine swabs when they were examined during the subsequent estrus. In addition, changes in uterine biopsy scores for inflammation and glandular dilation before and after treatment were similar for control and uterine device mares (with or without extended CL function); therefore, there was no evidence that the intrauterine device induced an inflammatory response in the uterus. On the basis of intensive blood sampling and measurement of PGF2α metabolite (PGFM) levels in the systemic circulation, on days 11 to 16 after ovulation in four control and eight uterine device mares, PGF2α secretion was attenuated in mares with prolonged CL function, with the exception of two mares; one mare showed a single PGFM peak and another showed two isolated PGFM peaks. Because there was no evidence of inflammatory changes caused by the intrauterine device, the author concluded the physical presence of the device in the uterine lumen somehow mimicked the effect of a conceptus by impairing endometrial secretion of PGF2α. In a subsequent brief report, the same group determined the "embryo-mimicking" effect of the intrauterine device prevents up-regulation of endometrial gene expression of cyclooxygenase-2 (COX-2) in mares that develop prolonged CL function, which is the key regulatory enzyme in PGF2α synthesis/secretion.[24] In nonpregnant mares, endometrial COX-2 expression is upregulated on days 14 to 15, coincident with the onset of PGF2α secretion, whereas the expression of COX-2 remains at basal levels in pregnant mares.[25–27]



Fig. 1. Two glass balls manually recovered from the uterine lumen of a mare displaying signs of urogenital discomfort. The current owner was unaware these glass balls were present in the mare's uterus. It is not known whether both glass balls were inserted at the same time or if one ball was placed initially and then at a later date the second ball was inserted without knowledge of the first one. Both glass balls had noticeable pitting on their surfaces, indicating it was likely that particulate matter from the glass balls had been deposited in the uterine lumen. Photo courtesy of Dr Candace Jacobson.

Although placement of an intrauterine glass ball has been the most widely used method of prolonging CL function in mares, there have been reports of deleterious consequences associated with their use, such as spontaneous fragmentation of the glass ball in the uterine lumen.[28] Although that specific complication can be avoided by the use of a device made of an alternative material such as plastic,[23] it is important to note that regardless of the composition of the device, if it is not eventually removed (spontaneously or manually), some mares may retain the device for an extended period of time (ie, years), such that its presence in the uterine lumen may not be known to individuals working with the mare (Fig. 1). This has led to situations in which mares have been bred when an intrauterine device was present, and there are anecdotal reports of mares becoming pregnant despite the presence of an intrauterine device and subsequently aborting because the device compromised the pregnancy later in gestation. Because of potential complications such as these, there is a need for alternative methods of extending CL function that are practical, efficacious, and safe.

5. Oxytocin Treatment

In contrast to the use of an intrauterine glass ball, administration of exogenous oxytocin during diestrus is an alternative method of blocking luteolysis to prolong CL function. Endogenous oxytocin is involved in regulating PGF2α secretion from the endometrium during spontaneous luteolysis in the mare,[29,30] and, although administration of exogenous oxytocin to mares around the time of luteolysis (ie, days 11 to 15 after ovulation) stimulates an immediate onset of PGF2α secretion,[31–33] when oxytocin is administered starting in the mid-luteal phase prior to the expected time of luteolysis (ie, before day 10 after ovulation), it does not induce PGF2α secretion and often paradoxically disrupts luteolysis causing prolonged CL function.[32]

Experimentally, continuous infusion of oxytocin from day 8 to 20 after ovulation blocked luteolysis in four of five mares, whereas luteolysis occurred at the expected time in all four control mares that received saline infusion.[34] Although it successfully induced prolonged CL function, continuous infusion of oxytocin would not be a practical method of long-term suppression of estrous behavior. As an alternative, in a "proof of principle" study, we showed that twice-daily intramuscular administration of 60 units (3 mL) of oxytocin on days 7 to 14 after ovulation was an efficacious method of disrupting luteolysis, because it caused prolonged CL function through day 30 after ovulation in six treated mares, whereas six saline-treated control mares underwent luteolysis by day 16 after ovulation.[35] In a subsequent study, we compared the use of the same 60-unit dose of oxytocin given intramuscularly twice daily versus once daily on days 7 to 14 after ovulation and found that CL function was maintained for 50 days after ovulation in five of seven mares (71%) treated twice daily, five of eight mares (63%) treated once daily, and one of seven (14%) untreated control mares.[36] There was no difference ($P > 0.05$) in the proportion of mares with extended CL function between once- and twice-daily administration of oxytocin, whereas collectively oxytocin treatment increased ($P < 0.05$) the proportion of mares with extended CL function. Therefore, the oxytocin treatment protocol can be simplified to once-daily administration on days 7 to 14.

In a third study, our objective was to monitor CL function and estrous behavior in mares for 90 days after administration of 60 units of oxytocin once daily on days 7 to 14 after ovulation.[37] Two of nine control (22%) and six of nine oxytocin-treated (67%) mares had prolonged CL function ($P = 0.08$). The mean duration of CL function in the two control mares with prolonged CL function was 78 days and in the six oxytocin-treated mares was 69 days. In both of the control mares and one of the six oxytocin-treated mares with prolonged luteal function, estrus was not observed while progesterone remained above 1.0 ng/mL. For the remaining five oxytocin-treated mares with prolonged luteal function, weak estrus was occasionally observed during the period of elevated progesterone. Although oxytocin treatment effectively prolonged CL function for approximately 2 months in two thirds of the treated mares, enigmatically, weak estrus was occasionally observed in some mares during the period of prolonged CL function.

Other research groups have recently evaluated modifications to the oxytocin protocol for prolonging

CL function by altering the dose, route of administration, and/or duration of treatment. In 2012, Gee et al[38] reported that five of six mares (83%) treated with 10 units of oxytocin intravenously once daily on days 7 to 14 had prolonged CL function compared with only one of six (17%) and two of six (33%) mares treated with 10 units oxytocin or saline intramuscularly, respectively. The apparent inability of 10 units of oxytocin administered intramuscularly to prolong CL function indicates the threshold intramuscular dose of oxytocin needed to disrupt luteolysis and prolong CL function is between 10 and 60 units. In all of the oxytocin-treated mares that developed prolonged CL function, estrous behavior was inhibited throughout the period of extended CL function, demonstrating the clinical efficacy of oxytocin treatment for estrus suppression. In a 2013 study, Keith et al[39] initiated intramuscular treatment with 60 units of oxytocin on day 8 and compared three different durations of treatment (2, 4, or 6 days). Administration of oxytocin on days 8 to 10, 8 to 12, and 8 to 14 induced prolonged CL function in three of seven (43%), four of seven (57%) and six of seven (86%) mares, respectively, compared with none of seven (0%) control mares. The proportion of mares with prolonged CL function increased ($P < 0.01$) as the number of days of oxytocin administration increased, confirming the need to continue oxytocin treatment until the expected time of luteolysis (ie, day 14) for maximum effectiveness. Also in 2013, Bare et al[40] compared the estrous cycle characteristics of mares treated with oxytocin or the synthetic oxytocin analog carbetocin on days 7 to 14 after ovulation. Carbetocin has a circulating half-life after intravenous administration 2.5 times longer than oxytocin (17.2 minutes versus 6.8 minutes, respectively).[41] Oxytocin-treated mares received 60 units of oxytocin intramuscularly once daily, whereas carbetocin-treated mares received 1.19 mg carbetocin intramuscularly, which, on the basis of its pharmacokinetics, is equivalent to 60 units of oxytocin. Compared with nontreated control cycles, administration of oxytocin increased the inter-estrus and inter-ovulatory intervals ($P < 0.01$), whereas carbetocin shortened the inter-estrus and inter-ovulatory intervals ($P < 0.01$), essentially "short-cycling" the mares. Therefore, with the use of this dose and treatment schedule, carbetocin was not efficacious for prolonging CL function.

In nonpregnant mares, the ability of the endometrium to secrete PGF2α in response to oxytocin (endogenous or exogenous) increases markedly between days 10 and 15 after ovulation concomitantly with an increase in the concentration of oxytocin receptors[33,42] and PGF2α synthetic enzymes[25] in the endometrial cells. In contrast, before day 10, the concentration of endometrial oxytocin receptors[33,42] and PGF2α synthetic enzymes[25] are low, which effectively blocks the ability of oxytocin to stimulate PGF2α secretion. We hypothesized that when oxytocin treatment is initiated before day 10 after ovulation, it prevents luteolysis by inhibiting the rise in endometrial oxytocin receptor concentration that would otherwise permit endogenous oxytocin-induced PGF2α secretion at the time of luteolysis; however, there was no difference in endometrial oxytocin receptor concentrations between saline-treated control mares and oxytocin-treated mares on day 15, which does not support that hypothesis.[36] More recently, it was demonstrated that oxytocin treatment suppresses PGF2α secretion by preventing upregulation of endometrial gene expression of COX-2,[39] which, as noted previously, appears to be the mechanism by which intrauterine placement of a polypropylene ball inhibits luteolysis and prolongs CL function.[24] Also, it seems clear from the results of Bare et al[40] that carbetocin (with use of the dose and treatment schedule in their study) does not have the same effect.

Collectively, the studies described above provide convincing evidence that administration of exogenous oxytocin on days 7 (or 8) to 14 after ovulation is an effective method of disrupting luteolysis to prolong CL function that can be used as a means of suppressing estrous behavior in mares. An advantage of using oxytocin treatment to prolong CL function is that it can be readily reversed by simply administering a luteolytic dose of PGF2α to initiate resumption of cyclical reproductive activity, in contrast to the need to physically remove an intrauterine glass ball. Disadvantages of the oxytocin protocol include the need to determine the exact day of ovulation before initiating treatment and potential difficulties associated with its use in mares with a needle aversion (ie, "needle-shy"). Although the dose of oxytocin is fairly high (60 units), no difference in body temperature, heart rate, or respiratory rate was noted before and after treatment; however, mild sweating, urticaria around the injection site, and mild diarrhea were noted in some mares.[40] Notably, there was no evidence of abdominal cramping and/or transient signs of colic,[40] which suggests the myometrium is much less responsive to oxytocin during mid- to late-diestrus than it is during other physiologic states (eg, immediately post-partum).

6. Inducing a Late-Diestrus Ovulation

In 2006, Hedberg et al[43] described the results of a preliminary study in which their objective was to prolong the luteal phase in mares with the use of human chorionic gonadotropin (hCG) to induce a late-diestrus ovulation to produce a new CL that would be too immature to respond to the luteolytic effects of endogenous PGF2α secretion at the end of diestrus (ie, day 14 to 15 after the initial "primary" ovulation). Mares were randomly assigned to control ($n = 4$) and experimental groups ($n = 5$), and, beginning on approximately day 8 after ovulation (or last signs of estrous in 3 mares), their ovaries were examined with transrectal ultrasonography every other day to determine the size(s) of their diestrus follicles. When a diestrus follicle $\geq$30 mm was de-

tected, control mares were treated with saline and experimental mares were treated with 3000 IU hCG intramuscularly. After treatment, the mares were monitored with transrectal ultrasonography for up to 72 hours or until ovulation was detected, and then once weekly for 3 weeks. If a mare did not have a diestrus follicle ≥30 mm during the first diestrus period, she was monitored for a second, and if necessary, a third diestrus period.

Three of the nine mares had development of a follicle ≥30 mm during the first diestrus period, four mares during the second diestrus period, and one mare in the third diestrus period. One mare never had a diestrus follicle that was ≥30 mm during three diestrus periods and therefore could not be treated with hCG. Overall, three of the four mares (75%) treated with hCG ovulated within 72 hours after treatment with hCG, which resulted in luteal phases that lasted for 58 to 82 days after treatment. None of the control mares ovulated during the luteal phase; however, one control mare had a spontaneously prolonged luteal phase during both a nontreated cycle in which she never developed a diestrus follicle ≥30 mm (CL function was terminated with exogenous PGF2α) and during the subsequent cycle in which she was treated with saline when she had a large diestrus follicle (that did not ovulate).

Although, on the basis of this study, the use of hCG to induce a late-diestrus ovulation looks promising for prolonging CL function, it is important to note that some mares (five of nine) required multiple estrous cycles to develop a diestrus follicle ≥30 mm. In addition, one mare never developed a large diestrus follicle during the three cycles that were monitored, which precluded her from receiving the hCG treatment. Therefore, in addition to the effort (and expense) of monitoring mares to evaluate their suitability for treatment, the fact that some mares may not develop a diestrus follicle large enough to warrant treatment, the use of hCG to induce a late-diestrus ovulation does not appear to be a reliable, "on-demand" method of blocking estrous behavior in mares. It is interesting to note that although the use of hCG was efficacious for inducing ovulation of diestrus follicles ≥30 mm in diameter in the study by Hedberg et al,[43] previous work by Glazar et al[44] found that administration of the GnRH agonist deslorelin acetate failed to induce ovulation and/or luteinization of diestrus follicles >30 mm in diameter.

7. Intrauterine Infusion of Plant Oils

In 2011, Wilsher and Allen[45] reported that intrauterine infusion of 10 mg estradiol in 1 mL fractionated coconut oil on day 6, 8, 10, 12, or 14 after ovulation resulted in prolonged CL function in 25%, 75%, 92%, 83%, and 50% of treated mares, respectively; however, they also demonstrated that estradiol was not needed to induce prolonged CL function, because infusion of 1 mL fractionated coconut oil or peanut oil (neither containing estradiol) on day 10 induced prolonged CL function in 92% of the treated mares in both groups. In contrast to infusion of plant oils, infusion of mineral oil on day 10 did not reliably prolong CL function, whether it was administered alone or in combination with estradiol (17% and 25% prolonged CL function, respectively). The authors postulated that the fatty acid milieus in both plant oils modulated/attenuated synthesis and/or secretion of PGF2α at the expected time of luteolysis, resulting in prolonged function of the CL. Given the high proportion of mares in which prolonged CL function occurred when fractionated coconut oil or peanut oil was infused on day 10 (92% in both groups), infusion of plant oil appears to be a plausible method of prolonging CL function for estrus suppression, although additional work will be needed to fully develop a practical protocol for that purpose and to ensure there is no detrimental effect on subsequent fertility.

8. Pregnancy

Pregnancy is another means of suspending cyclicity by taking advantage of the natural ability of the conceptus to block luteolysis and maintain CL function/progesterone secretion. Although efficacious, this method has obvious disadvantages that may make it undesirable for many horse owners. In addition to the time and expense necessary to establish pregnancy is the need to eventually terminate a normally developing pregnancy (unless an offspring is ultimately desired). Lefranc and Allen[46] reported that manual transrectal rupture of the conceptus between days 16 and 22 of gestation in 11 mares resulted in continued CL function for at least 60 days in all of the mares, during which time they did not display estrous behavior. Although efficacious, as noted above, terminating a normal, healthy pregnancy may be untenable to many horse owners.

9. Conclusions

Because of drawbacks and concerns associated with the use of altrenogest for suppression of estrus in mares, there is increasing interest in the development of practical methods of prolonging CL function, which allows continued secretion of endogenous progesterone to keep mares out of heat naturally. Although placement of an intrauterine glass ball has been the most widely utilized method of prolonging CL function, their variable efficacy and potential for deleterious consequences (if not eventually removed), has prompted interest in the development of additional methods of prolonging CL function. Of the current alternatives to an intrauterine glass ball, oxytocin treatment appears to be the most practical and efficacious method of prolonging CL function. Administration of 60 units of oxytocin intramuscularly once daily on days 7 (or 8) to 14 after ovulation induces prolonged CL function in 60% to 70% of treated mares, which can suppress estrous behavior for approximately 2 months. Ad-

ditional methods of prolonging CL function in mares include inducing a late-diestrus ovulation, intrauterine infusion of plant oils, and establishment of pregnancy followed by manual reduction of the conceptus after day 16 of gestation.

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[a]ReguMate, Intervet, Millsboro, DE 19966.
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Equine Pregnancy and Clinical Applied Physiology

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1. Introduction

The ability to produce a viable foal is critical to the brood mare. Maintenance of pregnancy entails almost a year of physiological "work" on the part of the gravid mare. This article reviews the physiology of pregnancy and applies it in the veterinary management of the mare.

2. Basics of the Endocrinology of Normal Pregnancy, From Ovulation to Parturition

An understanding of the endocrinology of equine pregnancy is helpful when considering administration of supplemental hormones to pregnant mares. We will begin with a basic review[1,2] and continue on to applying this information in practical therapeutic situations.

Progesterone begins to rise after ovulation in diestrus, irrespective of pregnancy status, with the development of the corpus luteum (CL). This initial rise of progesterone is approximately linear over the first several days; by day 5 after ovulation, serum progesterone is up to approximately 4 ng/mL.[3] If the mare is pregnant, progesterone produced by this CL (or corpora lutea if the mare has double-ovulated) maintains the pregnancy. This CL is called the primary CL of pregnancy. Early progesterone secretion is essentially the same, irrespective of whether the mare is pregnant or is in diestrus. If pregnant, luteolysis does not occur as it would in the nonpregnant mare, and this primary CL is maintained and continues to secrete progesterone. Ginther[1] has termed these two phases of progesterone secretion by the primary CL as "output D" and "output 1," for diestrous production and first luteal response of pregnancy.

Embryo migration between days 11 to 15 is necessary for maternal recognition of pregnancy, which keeps the early pregnant mare from returning to estrus. The signal for maternal recognition of pregnancy is poorly understood in the mare, but embryo migration within the uterus is clearly required. At day 16 or 17 after ovulation, the mobile embryo stops migrating within the uterus, and fixation, normally at the base of one of the uterine horns, occurs. Fixation is thought to be caused by a combination of increasing embryo size (diameter) as well as increased uterine tone, possibly caused by estrogen secreted by the embryo. Maternal recognition of pregnancy by the equine uterus prevents prostaglandin release by the endometrium, thus allowing the continued function of the primary CL.[4] Interestingly, by approximately day 30, progesterone production from the primary CL decreases, resulting in somewhat lower circulating serum progesterone

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concentrations. Progesterone as low as 2.5 ng/mL can even be seen at this stage of normal pregnancy, although somewhat higher concentrations between 4 and 10 ng/mL are often considered the "normal range" for the first trimester.[3] Fluctuations in peripheral progesterone levels have been shown to occur throughout the day, though not with a specific diurnal pattern.[5]

In the pregnant mare, unique structures known as the endometrial cups form by approximately day 35 after ovulation.[1] The cells that form the basis of the endometrial cups are from the embryo, specifically from the trophoblast chorionic girdle. These embryonic cells invade the maternal endometrium in a ring-like fashion around the developing umbilicus at the base of the uterine horn. There may be as many as 30 or more grossly visible, small, raised, whitish endometrial cups on the endometrial surface. The embryonic cup cells produce a hormone called equine chorionic gonadotropin (eCG), formerly known as pregnant mare's serum gonadotropin. This hormone is first detectable systemically between days 35 to 40 of pregnancy. The cups are mature and robustly secreting eCG at approximately days 50 to 60, but they will subsequently undergo sloughing by days 100 to 150 in most mares. This hormone is stimulatory to the ovary and causes follicular development, followed by either ovulation or luteinization (without ovulation) of these follicles. New luteal structures, called supplementary corpora lutea, are thus produced. As the result of the increased number of corpora lutea now present on the ovaries, systemic progesterone rises. The primary CL is also stimulated by eCG; therefore it remains active and secretes even more progesterone as well. This resurgence phase of progesterone secretion by the primary CL is termed the "secondary luteal phase" or "output 2," whereas the production by supplementary corpora lutea is termed the "third luteal phase" or "output 3."[1]

To summarize, progesterone rises after ovulation (diestrous phase) as the result of the primary CL. Because of maternal recognition of pregnancy, the primary CL does not undergo luteolysis but continues to secrete progesterone after day 14 of diestrus (first luteal phase). Subsequent to the stimulation by eCG, the primary CL increases production (second luteal phase), combined with production from the supplementary corpora lutea (third luteal phase).[1,4]

Progesterone from ovarian sources is required for early pregnancy maintenance to day 45. If pregnant mares are ovariectomized between days 50 to 70, many but not all will abort. It is during this stage that the feto-placental unit begins taking over the role of pregnancy maintenance by ramping up its progestogen production. The production of feto-placental estrogens also begins at this time.[1,2]

The corpora lutea continue to produce large quantities of progesterone, with high systemic concentrations (approximately 10–15 ng/mL) peaking at approximately 60 to 120 days of gestation[6]; they are active until regression begins at approximately 150 to 180 days after ovulation. Actual progesterone is quite low in the pregnant mare after approximately 180 days of gestation. By this time, the feto-placental unit has taken over the production of all progestogens. The term "progestogen" means progesterone-like substances or metabolites. The term "progestin" is also often used in a similar fashion, or "progestin" may be reserved for medications having progesterone-like function. All ovarian luteal structures will regress by approximately 200 days of gestation.[1,2]

Feto-placental progestogens, especially 5-α-pregnanes, are detectable in maternal serum/plasma by 60 days of gestation when measured by means of advanced laboratory techniques (gas chromatography/mass spectrometry, which are not commercially available).[7] The substrate for these progestogens is maternal cholesterol. Cholesterol is metabolized to pregnenolone (P5) and then to 5-α-pregnanes and other progestogens. The sites of progestogen conversion include utero-placental tissues as well as the fetal gonads and adrenals. It is thought that the fetal adrenals play a critical role in progestogen metabolism through their production of P5. Total progestogen concentrations are relatively high in the second and third trimesters; they will then increase dramatically during the last few weeks of gestation and peak in the last days of gestation, only to drop precipitously near term.[1,2]

Although sometimes overlooked, estrogen plays an endocrinological role in early pregnancy. The embryo produces small amounts of locally active estrogens. However, it is not until approximately day 35 that systemic estrogen rises. The source of this estrogen is the ovary, more specifically, the corpora lutea and possibly follicles. The stimulation of the ovaries by eCG is responsible for the timing of this increase in estrogen. It appears that estrogen is not actually necessary for pregnancy maintenance, because ovariectomized mares administered only exogenous progestins will maintain pregnancy without the administration of estrogens.[1,2]

At approximately 80 days of gestation, the feto-placental unit increases the production of estrone, estradiol-17β, estradiol-17α, and the equine-specific estrogens equilin and equilenin. The gonads of both male and female fetuses produce estrogen precursors, such as dehydroepiandrosterone, which are then metabolized at the level of the placenta into the various estrogenic hormones. The fetal gonads are grossly hypertrophied during gestation when large quantities of these estrogen precursors are being made. High levels of these estrogenic compounds will be present throughout much of the remainder of pregnancy, decreasing only in the last 2 to 3 months of gestation. These estrogens are postulated to increase blood flow to the fetal compartment and to promote uterine tonicity.[1,2]

To summarize, during the second and third trimesters, the feto-placental compartment produces large amounts of progestogens and estrogens. Hormone production occurs through the teamwork of fetal adrenals and gonads as well as the placental tissues. Total estrogens gradually decline during the last trimester. Total progestogens continue to rise and increase greatly during the last several weeks of gestation, finally peaking before parturition.[1,2]

The equine mechanism of parturition is not completely understood, but general concepts have been elucidated and reviewed.[8,9] As discussed above, high progestogens and low total estrogens are the hormone milieu of late gestation. With the rapid fetal growth of late gestation, the uterus becomes "stressed" by physical stretch, making the myometrium more contractile. The effect of stretch is thought to be countered by the high and increasing progestogens that characterize late gestation. The high progestogens are necessary for continued uterine quiescence until just before the time of parturition. These progestogens promote enzymes that inactivate prostaglandins, thus "blocking" their ecbolic (contractile) effects.[8,9]

At the end of gestation, a dramatic change in the hormonal milieu occurs, and progestogens fall precipitously. This drop coincides with maturation of the equine fetal hypothalamic-pituitary-adrenal axis, which occurs very late in gestation and is necessary for the fetus to signal its preparedness for parturition. As noted above, equine fetal adrenals produce the precursor P5, which is metabolized by the placenta to progestogens. As fetal adrenocorticotrophic hormone (ACTH) rises, adrenal enzymatic pathways are modified, and concomitantly the fetal adrenal decreases P5 production and instead produces cortisol. Thus, less fetal adrenal P5 substrate is produced and progestogens fall, normally occurring as late as 1 to 2 days before parturition.[8,9]

We think of corticosteroids as being anti-inflammatory; however, in the late-term fetus, the recently produced fetal cortisol activates enzymes that actually synthesize more prostaglandins. As the degree of progestogen "block" falls, prostaglandins continue to rise and myometrial contractions begin to occur. In association with periods of evening myometrial contractions, estradiol-17β rises at night in the week before parturition. This estrogen increases uterine responsiveness to prostaglandins and may even promote further prostaglandin production. Finally, the neuroendocrine Ferguson reflex, resulting from fetal distention of the cervix and vagina, causes large amounts of oxytocin release. Oxytocin and prostaglandin peak with rupture of the chorioallantois ("water breaking") and expulsion of the fetus.[8,9]

3. Evidence-Based Applied Endocrinology and Practical Therapeutics

Progestogen supplementation can be used as treatment for early-bred or known pregnant mares if the mare's endogenous progesterone production is suspected of being low.[10] For instance, altrenogest or natural progesterone may be administered beginning in early diestrus (commonly at or after day 5 after ovulation, theoretically not to have the treatment interfere with embryonic passage through the oviduct). Ovariectomy studies with the use of progesterone supplementation have shown that serum progesterone concentrations of 2 ng/mL were considered the minimum endogenous amount necessary to support pregnancy. In addition, when concentrations above 4.0 ng/mL were maintained consistently, no study mares aborted.[11] Note that altrenogest does not cross-react with progesterone assays; therefore endogenous progesterone production may be monitored while mares are supplemented with this product.[12] A commercial altrenogest assay to determine circulating altrenogest levels is not readily available. If supplementing with a parenteral natural progesterone, a serum progesterone determination will show the cumulative effect of both the exogenously administered product and endogenous production by the mare.[13]

Little research has addressed the question of whether altrenogest administration suppresses endogenous progesterone production. Two studies, one in earlier gestation[12] and one in late gestation,[13] concluded that altrenogest administration did not suppress endogenous progesterone production.[12,13] When eight mares were administered 22 mg of altrenogest orally daily from day 40 to 105 of gestation, no changes in serum progesterone were identified when compared with the eight control mares.[12] In this study, progesterone was not monitored daily but only on days 40 to 46, 69 to 75, and 99 to 105 of gestation.[12] On the other hand, a recent study[14] that evaluated the effects of nonsurgical embryo transfer and altrenogest administration on serum progesterone concentrations found lower progesterone in altrenogest-treated mares. Altrenogest, 19.8 mg, was administered orally from days 6 to 21 after ovulation. In nonpregnant, sham embryo–transferred mares, progesterone was lower at postovulatory days 10, 12, and 13 in the eight mares administered altrenogest, when compared with the eight similar sham controls. In early pregnant mares (that did not have sham or actual embryo transfers performed), progesterone was lower at days 14 to 18 and 21 in the eight altrenogest-treated mares compared with the eight control pregnant mares.[14] The researchers speculated that the altrenogest may be feeding back negatively at the level of the pituitary, with subsequent decrease in luteinizing hormone release.[14] The first study[12] was performed during the period of endometrial cup secretion of eCG, a luteotrophic hormone, whereas the recent study was performed at stages without eCG support.[14] Thus, the specific hormonal milieu at 40 to 105 days of gestation may have stimulated enough endogenous progesterone production to minimize any potential negative

feedback effects of altrenogest. Often, when discontinuing altrenogest treatment, veterinarians will gradually lower, over a week or so, the daily altrenogest dose administered before its complete cessation. The theory behind this "weaning" practice is that it will allow time for the gravid mare's endogenous endocrine systems to gradually adapt to altrenogest withdrawal.

Endotoxin experiments provided good evidence for the practical use of altrenogest supplementation during early pregnancy, before formation of endometrial cups, in the mare.[15] Endotoxin stimulates endogenous prostaglandin release, thus causing luteolysis, with resulting pregnancy loss. Mares between 21 to 35 days of pregnancy were administered systemic endotoxin. All seven mares that were concomitantly administered 44 mg of altrenogest orally once daily until day 70 maintained their pregnancies. In mares that were administered altrenogest only to day 40 of gestation, six of seven mares had fetal death within 4 days of altrenogest cessation. Thus, altrenogest administered at the time of insult could maintain early pregnancy despite a toxic insult and subsequent luteolysis. In addition, the altrenogest treatment needed to be maintained until the feto-placental unit was producing adequate progestogen for subsequent pregnancy maintenance. The 44 mg of altrenogest per mare used in this study corresponds to a dose of approximately 0.088 mg/kg, which is commonly called a "double dose."[15] By comparison, the standard dose of altrenogest[a] labeled for suppressing estrus is 0.044 mg/kg ("single dose") orally every 24 hours.[16]

These same researchers also addressed whether flunixin meglumine could prevent luteolysis and maintain pregnancy in mares that were administered endotoxin.[17] Flunixin meglumine, a non-steroidal anti-inflammatory cyclo-oxygenase inhibitor, interferes with the production of prostaglandin. When flunixin meglumine was administered to mares between days 21 and 44 of gestation intravenously 10 minutes before endotoxin administration, endogenous progesterone production was maintained and none of the seven mares lost their pregnancy. In those mares in which flunixin meglumine was administered 1 hour after endotoxic insult, systemic progesterone fell to below 2 ng/mL for several days, and pregnancy was lost in one of three mares. When flunixin meglumine was administered 2 hours after endotoxic insult, progesterone fell to below 0.5 ng/mL, and all three pregnancies were lost. Likewise, the 12 pregnant mares administered only endotoxin had very low progesterone concentrations, and all lost their pregnancies. Thus, flunixin meglumine was most useful when administered before endotoxin was given, but it was also of benefit in maintaining pregnancy in two of three mares when administered within 1 hour of the insult.[17]

Other work supports the administration of progesterone or altrenogest to maintain pregnancy in the face of prostaglandin-induced abortion. In one study, six mares between 82 and 102 days of gestation were administered 250 μg of cloprostenol (q 24 h, IM) to induce abortion.[18] Systemic prostaglandin concentrations rose, whereas progesterone concentrations fell, and fetuses were expelled spontaneously by the third day of cloprostenol administration.[18] Subsequently, the ability of progesterone or altrenogest[19] and flunixin meglumine[20] administration to inhibit abortion induced by cloprostenol administration was examined. Mares were either administered progesterone 300 mg (q 24 h, IM) or 44 mg altrenogest (q 24 h, PO; "double dose") beginning either 18 or 12 hours, respectively, after the first cloprostenol injection. The progesterone regimen was used in eight mares between 98 to 153 days of gestation, and, of these mares, only three aborted. When altrenogest was used in similar fashion in mares between 93 to 115 days of gestation, none of the mares aborted.[19] In contrast, when 500 mg flunixin (q 8 h, IV) was administered beginning 15 minutes before the first daily cloprostenol injection, all mares aborted.[20] Thus, progesterone or altrenogest supplementation[19] but not flunixin administration[20] blocked cloprostenol-induced abortion at these gestational ages. Taken together, these studies support the concept that progestin supplementation can maintain equine pregnancy when the mares were submitted to prostaglandin F2α insults.[15,19]

If there is fetal demise after formation of the endometrial cups, the mare will normally have a phase of pseudopregnancy.[1] Even without a live pregnancy, the endometrial cups will not regress until approximately days 120 to 150 after ovulation. The cups continue to secrete eCG, subsequently causing the ovarian luteal structures to secrete progesterone, which inhibits resumption of cyclicity. Loss of pregnancy during the endometrial cup phase will generally prevent the mare from becoming pregnant again during that season. Therefore, in cases in which a pregnancy must be terminated early (ie, presence of twins), it is important to perform elective abortion before endometrial cup formation, which is approximately 35 days of gestation but subject to individual mare variation. Before eCG production, a single dose of prostaglandin administered to the pregnant mare will result in luteolysis and pregnancy termination. Return to estrus and ovulation are normal after this procedure when performed before the eCG phase. After eCG production is present, and hence after the formation of supplementary corpora lutea, multiple doses of prostaglandin are necessary to terminate pregnancy. Although some mares may return to estrus when fetal demise (termination or spontaneous) occurs during the eCG phase, others will not. The eCG production that continues after the fetal loss affects ovarian function, in which case follicles develop but then may luteinize, without ovulating properly.[1]

To illustrate the use of prostaglandin for elective pregnancy termination, 185 abortions were induced with once-daily intramuscular injections of cloprostenol administered during the period of eCG production.[21] Three to four daily injections were necessary. Reportedly, in 90% of the abortions, no manual veterinary intervention was necessary.[21] Exogenous prostaglandin treatment caused luteolysis and increased endogenous prostaglandin production, altogether causing uterine contractions, subsequent cervical softening, and expulsion of the fetus.[18]

Once eCG production subsides, multiple daily doses of prostaglandin are still necessary to achieve abortion. Approximately 66% to 75% of 21 pony and 25 horse mares, respectively, aborted within the first week of administration of daily prostaglandin (product not specified) when it was administered at 100 to 245 days of gestation.[22] Some mares required up to 4 weeks of daily prostaglandin to achieve abortion.[22] Later in gestation, at approximately 10 months of gestation until near to term, both natural prostaglandin and the analogue fluprostenol did not reliably cause parturition when administered several times in one day.[23] It is also worthwhile to note that, depending on stage of gestation, dystocia may occur.

4. Late-Gestation Progestogen and Estrogen Monitoring

Systemic diseases that affect the gravid mare can affect hormone production by the fetus and placenta. Serial serum progestogens can be periodically monitored in mares as an indicator of pregnancy health. Commercially available progesterone assays cross-react considerably with the pregnanes and other progestogens produced by the feto-placental unit. Thus, diagnostic laboratories will use their progesterone assay to measure total progestogens, which is the sum of both progesterone and other cross-reacting feto-placental progestogens.[24] Note that laboratory assays for progesterone vary in their ability to cross-react and quantify the various equine progestogens. Thus, total progestogens determined on a serum specimen from a gravid mare and assayed at two different laboratories, running two different progesterone assays, may show considerable differences in the total values. It is best to stay with one laboratory when submitting serial specimens for pregnant-mare total progestogen determination. Normal reference ranges have been determined by means of the assay at BET Laboratories in Kentucky.[3] Total progestogens normally ranged from 4 to 10 ng/mL until approximately 300 to 320 days of gestation. After this time, progestogens may increase to above 40 ng/mL.[3,25]

Total progestogen concentrations are generally performed in a serial fashion so that a trend of rising, stable, or falling, becomes evident.[25] Progestogens have been shown to rise abnormally and prematurely in the compromised mid- to late-gestation pregnancy.[26] In severe cases of pregnancy compromise, in which fetal death is imminent, acute decreases in progestogens have been identified both in naturally occurring pregnancy conditions[26] and in experimentally induced placentitis.[27] Increases in progestogens before that normally identified in the last several weeks of gestation are a cause for concern and indicate ongoing fetal stress and compromise, with premature activation of the fetal pituitary adrenal axis.[28] The mechanism for this occurrence is the increased fetal adrenal production of the precursor P5, which is metabolized by the placenta to more progestogens.[2] Response to treatment may be assessed indirectly. For example, if progestogens fall back to a normal range, then improvement in the clinical condition of the fetus and placenta is suggested. Alternatively, failure of progestogens to rise can be identified in mares with fescue toxicity or uterine pathology and prolonged gestation.[29]

Estrogen production can likewise be determined in serum obtained from the mare and used as an indicator of feto-placental health. Total estrogen concentrations may also be taken serially but can be helpful if only a single specimen is assayed.[25] As discussed above, the fetal gonads produce estrogen precursors that are then metabolized at the level of the placenta into various estrogenic hormones. Estrogens peak in the second trimester and then fall gradually.[1,2] Normal reference ranges have been determined by means of the assay at BET Laboratories in Kentucky. Total estrogens normally were at least 1000 pg/mL from approximately 150 to 300 days of gestation. After this time, estrogens will normally be lower as term approaches.[3]

Low total estrogens have been found in cases of placentitis. Retrospectively, Douglas[25] found that in naturally occurring cases of placentitis at gestational ages between 150 and 280 days, it was common to find both abnormally elevated total progestogens and low total estrogens in mares that aborted. Mares that maintained their pregnancy (with various treatments administered) had normally low progestogen and normally high estrogen.[25]

5. High-Risk Mares and Reproductive Hormone Supplementation

The discussions above lead to the question of hormone supplementation for the high-risk pregnant mare with a compromised feto-placental unit. Questions one might ask are: If progestogens are elevated in abnormal pregnancies, then why administer additional progestins to the mare? What is the argument for this administration? Furthermore, is progestin supplementation safe for the fetus?

The argument for administration of progestins to later-term pregnant mares with at-risk pregnancies is that the treatment maintains uterine quiescence. As was discussed above, progestogens continue to

rise in late pregnancy and help maintain uterine quiescence up until just before parturition.[8,9] In normal pregnancies, progestogens actually upregulate a placental enzyme, 15-hydroxy prostaglandin dehydrogenase that breaks down prostaglandins. Prostaglandin F will cause smooth muscle contraction. Thus, it is postulated that higher progestogens lower the effects of prostaglandins and quiet the myometrium.[9] In addition, progestogens have immune modulating effects critical for pregnancy maintenance.[30]

Although exogenous progestins were shown to be effective in maintaining early pregnancy in the face of attempted cloprostenol-induced abortion,[19] similar studies have not been performed in late pregnancy. However, the efficacy of progestin treatment has been demonstrated when used in combination with trimethoprim sulfamethoxazole (30 mg/kg, q 12 h, PO) and pentoxifylline (8.5 mg/kg, q 12 h, PO) to treat experimentally induced bacterial placentitis.[31] Altrenogest at a dose of 0.088 mg/kg (q 24 h, PO; "double dose") was used in both the placentitis[31] and cloprostenol studies.[19]

Regarding the safety of supplemental progestogen administration, safety studies presented on the altrenogest label[a] indicate that the only untoward sign was increased clitoral size in fillies born to normal mares that were administered 0.088 mg/kg (q 24 h, PO), beginning at 20 days of gestation and ending at 325 days.[32] Thus, it seems reasonable to discontinue altrenogest by approximately 325 days of gestation.

Altrenogest crosses the placenta and is present in the foal at birth.[33] A recent study compared parturition and foal health parameters in normal pony mares maintained on the 0.088 mg/kg dose of altrenogest through foaling and those that were not administered altrenogest. In six healthy pony mares supplemented with altrenogest through parturition, stage II active labor was more than 10 minutes in duration in four of the five treated mares, and one additional treated mare required a cesarean section. Foals from treated mares had lower respiratory rates and higher blood pH; one foal died at 30 minutes, and another needed intensive supportive care.[34] Finally, these researchers found that there were differences in the neutrophil-to-lymphocyte ratio, suggesting dysmaturity, in foals born to altrenogest-treated pony mares.[35]

Some progestins have been shown to alter mental arousal status. When progesterone was administered systemically to pregnant ewes, fetal lamb mental status was lessened as determined by monitoring of fetal neurologic and electromyographic responses.[36] Regarding its effect in horses, one case report describes a foal that became stuporous and obtunded after being administered the progesterone metabolite allopregnanolone.[37] However, it is currently not known if administration of progestins to the pregnant mare causes any noticeable alteration in fetal mental status. Interestingly, progesterone therapy has been used in humans with acute traumatic brain injury, in which case the progesterone is thought to have neuro-protective effects and to lessen cerebral edema.[38] Additionally, a rodent model of pediatric brain trauma with the use of progesterone as a treatment has shown some positive neuro-protective effects.[39] Finally, human pediatric studies evaluating progesterone treatment effectiveness in head trauma are planned or ongoing.[40,41]

Another consideration is whether administration of progestogens to the late-gestation pregnant mare can alter gestational length. Progestogens rise near term and then fall off precipitously in the last 1 to 2 days before foaling.[1,2] If progestogens are administered to the late-pregnant mare, could this treatment mimic what is happening in late gestation and inadvertently shorten gestational length? Only a few studies have addressed this pertinent question. When natural progesterone was administered daily to healthy mares from 318 days of gestation to foaling, gestational length was shortened from a mean of 344 days in nine untreated controls to a mean of 332 days in the nine treated mares.[42] Reportedly, these foals were healthy at birth.[42] As discussed above, six healthy, late-gestation pony mares were administered a double dose of altrenogest through foaling. The gestational length of ponies administered altrenogest tended to be shorter but was not statistically significant from the untreated controls.[34] Finally, groups of six pregnant pony mares were administered either altrenogest, progesterone, or placebo daily from 300 days of gestation for 10 days.[13] Gestational length was not different between groups, and all foals were reportedly healthy. In this study, progesterone supplementation increased total progestogens as measured in the mare serum, but altrenogest did not increase total progestogens.[13] With such small sample sizes, the possibility of normal gestational length variability negating any effects between groups might be considered. Additionally, if one considers that equine gestation is normally variable between 320 to 365 days, then there is the chance that progestin administration did nothing to shorten gestation in early studies. Nevertheless, the administration of progesterone or altrenogest did not reliably prevent equine parturition.

Limited work has been performed looking at the administration of estrogens to the pregnant mare. In one study,[25] clinical cases of mares assumed to have placentitis and medically managed with various antibiotic and medical regimens were either administered no estrogens or either estrogen cypionate or estradiol 17β. The mares were not administered progestins. The live foal rate of 70% was higher in the group given either of the two estrogen preparations compared with the live foal rate of 20% in mares not given estrogen. In this study, the live foal rate was 87% for a control group of normal

mares not affected by placentitis.[25] Further studies regarding estrogen treatment have not been performed. With such limited information available, general recommendations regarding estrogen administration cannot be made.

6. Dexamethasone Induction

Since the 1970s, it has been known that administration of high doses of dexamethasone to healthy, late-gestation mares will induce parturition.[42] Dexamethasone, 100 mg per mare (approximately 0.2–0.25 mg/kg; q 24 h, IM), from 321 to 324 days of gestation in 12 mares, shortened gestation and resulted in the birth of viable foals. Mean gestation length in treated mares was 328 days versus 340 days in controls. Parturition, fetal membrane expulsion, mammary gland development, and lactation were normal, and the mares remained healthy. Foals were described to be initially "weak in the fetlock area," but the problem resolved by 7 to 10 days of age. Although dexamethasone-induced foals were smaller, foal growth rate was similar to that in foals born to untreated mares.[42]

Similarly, in five healthy mares administered this same dexamethasone treatment at days 315 through 317 days of gestation, healthy foals were born at a mean gestational age of 322 days, compared with five control mares at a mean of 335 days.[43] One mare foaled within 15 hours of the last dexamethasone injection. A parturition range of 1 to 8 days after last dexamethasone administration was reported. This group did not find joint laxity or poor ossification of the foals' fetlock or carpal joints. Most (four of five) foals produced from the dexamethasone treatment group required administration of supplemental colostrum to ensure adequate passive transfer. The foals responded adequately to an ACTH challenge test. This group specifically noted that other than slight depression, mild inappetence, and poor mammary gland development at foaling, the mares were healthy and showed no evidence of laminitis. Maternal total progestogens rose, whereas cortisol and a metabolite of prostaglandin fell, during dexamethasone treatment. This group cautioned that although the foals and mares remained healthy during this treatment with dexamethasone, this treatment protocol could not be directly or safely extrapolated to the mare with a compromised pregnancy in which the fetal hypothalamic pituitary axis was already activated because of fetal distress.[43] In both studies,[42,43] administration of dexamethasone was begun before the spontaneous maturation and activation of the fetal hypothalamic pituitary adrenal axis.

The association between dexamethasone treatment and subsequent dystocia has also been reported.[44,45] In an early report, two healthy pony mares were administered 100 mg of dexamethasone (mean, 0.3 mg/kg; q 24 h, IM) for 3 or 4 days beginning approximately 344 to 347 days of gestation, and no outward signs of initiation of labor were identified. However, both mares subsequently had dystocia, with dead foals delivered. Both mares also retained their placentas. A third pony mare was administered 100 mg dexamethasone for 3 days beginning at 327 days. On day 331, oxytocin was administered, and, in this case, parturition was normal but the placenta was again retained.[44,45]

Clinical data are limited regarding the use of dexamethasone for induction of parturition in healthy mares, much less in compromised pregnancies. One study[46] used smaller doses of dexamethasone in mares with experimentally induced streptococcal placentitis. Several treatment approaches combining various combinations of trimethoprim sulfamethoxazole, aspirin, altrenogest, and dexamethasone were used in the mares once clinical signs consistent with placentitis had developed. Trimethoprim sulfamethoxazole (30 mg/kg, q 12 h, PO) and dexamethasone were administered to one group of six light horse mares. Dexamethasone was given as follows: 40 mg, 35 mg, 25 mg (q 24 h, IV), with each successively lower dose administered for 2 days. Four of the six mares delivered viable foals at a gestational age of approximately 308 ± 4 days (mean ± SEM). Likewise, four of six mares administered only trimethoprim sulfamethoxazole had viable foals but at approximately 319 ± 5 days. It is not clear from the report if both groups of mares were inoculated at similar gestational ages. In this study, aggressive antibiotic treatment was deemed most useful in improving fetal outcome. Reportedly, the mares were not systemically ill and did not show signs of laminitis.[46] High-dose dexamethasone treatment has been reported to cause laminitic complications in mares affected by bacterial placentitis.[b]

7. Conclusions

Studies to elucidate equine fetal physiology and that of normal pregnancy continue to this day. In addition, work on pathological mechanisms of diseased pregnancy and how to best treat the high-risk gravid mare is ongoing. Not everything is known, and pieces of the puzzle are being added. There is often no easy answer to the difficult clinical presentation. My hope is that this article has provided you with knowledge that will help you in your clinical decision-making.

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[a]Regu-Mate Solution (altrenogest) Intervet, Inc, Middlesboro, DE 19709. *A new generic formulation of altrenogest came out in the USA market last year. Altresyn manufactured by Ceva Animal Health, LLC, Lenexa, KS 66215.*

[b]LeBlanc MM. Gainesville, FL (personal communication) 2013.

How to Take Radiographs of the Metacarpophalangeal/Metatarsophalangeal Joint (Fetlock Joint)

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1. Introduction

The fetlock joint is one of the most common radiographed areas of the horse. It is a relatively simple joint made up of the third metacarpal/metatarsal bone (cannon bone), the proximal phalanx (P1), and the paired proximal sesamoid bones. To accurately assess pathology within the joint, quality radiographic images must be obtained.

2. Materials and Methods

Currently in the author's practice, digital radiography is used exclusively. The techniques will vary, depending on which system being used (digital, computed, or plain radiography). It is possible to obtain excellent radiographs with any form of image acquisition as long as proper technique is used. In the author's experience for fetlock radiography, the patient is usually not sedated; however, if the patient is difficult, the author will use anywhere from 2.5 mg to 5 mg of detomidine IV. A good handler is very important because they will properly position the horse so that image acquisition is easier. Radiation safety is always practiced by everyone involved wearing lead gowns and thyroid protectors and the plate holder wearing lead gloves.

3. Results

Standard views of the fetlock include five views (Figs. 1–4). The standard views taken follow: dorsal to palmar elevated 10° to 15° (DP), standing lateral to medial view (SLM), flexed lateral to medial view (FLM), dorsomedial (30°) to palmarolateral oblique view, elevated 10° to 15° (DMPLO), and a dorsolateral (30°) to palmaromedial oblique view, elevated 10° to 15° (DLPMO).

Special Views

As mentioned earlier, there are some additional views of the fetlock that are taken to highlight specific anatomic areas of concern. Tangential fetlock DP views are used to highlight portions of the articular surface of the distal metacarpus/metatarsus not seen on the standard DP projection. The most common is a flexed DP elevated at 10° to 15° (FDP) of the fetlock (Fig. 5). More extreme tangents are sometimes necessary, such as the dorsodistal-palmaroproximal DP view, which is taken in the weight-bearing position 15° below horizontal (Fig. 6). Another specialty view is the palmar 45° proximolateral-palmarodistal medial oblique view (Pal 45° PrL-DiMO, Fig. 7). This view can also be taken from the contra-axial side of the limb.

NOTES



Fig. 1. Dorsal to palmar view of fetlock, elevated 15° to project joint space.



Fig. 3. Standing lateral to medial view with excellent overlap of sesamoids and visualization of mid-sagittal ridge.



Fig. 2. Poor-quality DP caused by lack of 15° elevation, which leads to lack of projection of joint space.



Fig. 4. Poor-quality SLM caused by improper angle in the medial to lateral and proximal to distal direction.



Fig. 5. Oblique views are similar on the lateral and medial aspects. A 30° angle (from lateral) projects dorsal P1 and divides sesamoids; 15° elevation allows visualization of palmar/plantar condyle of cannon bone.



Fig. 7. Flexed lateral to medial view straight through the joint, excellent sesamoid overlap, and visualization of mid-sagittal ridge.

4. Discussion

Factors Affecting Radiograph Quality

Some factors that result in poor-quality radiographs are poor positioning, dirt/mud/water on the horse, motion on the radiograph, and poor exposure. These examples can lead to a misrepresentation of the horse and an incorrect diagnosis. Motion makes the radiograph difficult to interpret accurately (Fig. 8). Poor positioning can result in hidden pathology because the proper area is not highlighted (Fig. 9). Dirt, mud,



Fig. 6. Poor quality oblique as the radiograph beam is oriented in too much of a dorsal to palmar/plantar direction.



Fig. 8. Poor-quality FLM because the radiograph beam is not straight through the joint, the sesamoids do not overlap, and there is no visualization of the mid-sagittal ridge.



Fig. 9. The flexed DP highlights the distal palmar/plantar condyles of the cannon bone. This view is used to detect condyle fractures on the palmar aspect that are not seen easily on the standard DP view. Image was taken in the non–weight-bearing position.



Fig. 10. Dorsodistal-palmaroproximal DP of the fetlock. Despite superimposition of the sesamoids over the fetlock joint, a short, incomplete (dorsal to plantar) condylar fracture can be seen that was not apparent on the standard DP view.



Fig. 11. Palmar 45° PrL-DMiO. This view is used to highlight the abaxial surface of medial sesamoid bone. This projection can be used for either sesamoid.



Fig. 12. Motion. Flexed lateral to medial view with motion. The image becomes "blurry," and subtle detail and lesions may be missed.



Fig. 13. Poor positioning. This oblique is too DP and therefore does not allow proper visualization of the condyles or dorsal P1.



Fig. 14. Dirt results in an artifact that can resemble pathology if present over an area of concern. It is very important to clean the area of interest before taking radiographs.



Fig. 15. Poor exposure. This underexposed image results in a lack of anatomic detail and also highlights the water present on the horse. Water artifact has the same effect as dirt and could easily be misinterpreted as a lesion; therefore, limbs should be dried before imaging.

and water on the horse cause artifacts on the radiographs and can make it difficult to differentiate true lesions from artifact (Figs. 10 and 11). Poor exposure can be the result of underexposure (light image) or overexposure (dark image). Digital images can compensate for some exposure problems by altering the brightness and contrast (windowing) (also see Figs. 12–15). However, gross inaccuracies in exposure cannot be corrected, especially overexposure.[1–3]

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How to Interpret Radiographs of the Fetlock and Pastern Joints of the Young Performance Horse

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1. Introduction

Routine radiographic evaluation of young performance horses has become commonplace in Thoroughbreds but also occurs in other performance breeds. This practice has resulted in the frequent discovery of radiographic abnormalities (RA) that are often clinically silent but can cause concern in buyers and sellers of young performance stock. The prevalence of radiographic developmental orthopedic disease in 6-month-old horses has been reported to be 25% in Warmbloods, 41% in Standardbreds, and 34% in Thoroughbreds (TB).[1] In Thoroughbred yearlings intended for racing, 86.3% exhibit RA;[2] in young Standardbreds, 42%;[3] in yearling Warmblood horses, 69.5%;[4] and in 1- and 2-year-old Quarter Horses intended for cutting, 89%.[5] The fetlock joints are often reported as the joints most commonly affected by RA. Predicting the significance of RA to later athletic performance can be challenging and frustrating for buyers and sellers, which is further complicated by the possibility of treatment, including surgery, on prognosis (Table 1[2,4,6–13]).

2. Identifying RA

Because radiographic images are reviewed in sets of projections of one joint, this paper will discuss abnormalities by each projection. Readers are, of course, responsible for reading the entire image. However, there are areas that should receive the closest scrutiny because of a greater frequency of abnormalities. These areas will be emphasized and the most common RA in young horses described. In the following section, the RA that is best detected on a specific projection will be listed in bold. This RA should be confirmed on other projections when possible. Finally, readers should always consider three general factors when reviewing radiographic images:

(1) Make sure that the films are of the correct horse.
(2) Make sure the date is appropriate and consistent.
(3) Make sure all required projections are present and of acceptable quality.

NOTES

Fetlock Radiographic Abnormality	Breed	Prevalence	Reference
RA (front fetlocks)	Thoroughbred	41%	2
RA (front fetlocks)	Warmblood	9.5–19%	4, 6
RA (hind fetlocks)	Thoroughbred	21%	2
RA (hind fetlocks)	Warmblood	13.7–23%	4, 6
RA (hind fetlocks)	Quarter Horse	44%	5
RA (hind fetlocks)	Standardbred	18%	3
RA in all four fetlocks	Standardbred	13–18%	3, 7
Dorsal proximal P1 fragments; front	Thoroughbred	0.7–4%	2, 8, 9
Dorsal proximal P1 fragments; front	Quarter Horse	1%	5
Dorsal proximal P1 fragments; hind	Thoroughbred	3.3	8
Dorsal proximal P1 fragments; hind	Quarter Horse	1%	5
Proximal palmar P1 fragments	Thoroughbred	0.4–2%	2, 8, 10
Proximal plantar P1 fragments	Thoroughbred	4.7–6.3%	8, 10
Proximal plantar P1 fragments	Quarter Horse	1.0	5
Proximal plantar P1 fragments	Standardbred	17–23%	3, 11
Lucencies or fragments dorsal sagittal ridge	Thoroughbred	0.4–28%	2, 8
Lucencies or fragments dorsal sagittal ridge	German Coldblood	54%	12
Changes to shape, dorsal sagittal ridge	Thoroughbred	20–27%	2, 8, 9
Supracondylar lysis (forelimb)	Thoroughbred	0.1–4.8%	2, 8
Abnormal length or shape to PSB	Thoroughbred	3.8–5.6%	2, 8
PSB fracture	Thoroughbred	1–1.5%	2, 8
PSB fracture	Quarter Horse	1%	5
PSB enthesophyte (front, hind)	Thoroughbred	1.2%, 39%	8, 9
PSB lucency (all four limbs)	Thoroughbred	5.5–13.6%	2, 8
>2 PSB vascular channels	Thoroughbred	33.5–50%	2, 8
Subchondral bone cyst distal MC/MT3	Thoroughbred	0.4–0.8%	8, 10, 13

Table 1. Prevalence of Fetlock Radiographic Abnormalities. P1 indicates first phalanx; PSB, proximal sesamoid bones; MC/MT3, third metacarpus or metatarsus.

Standard radiographic projections of the fetlock joint (5 for forelimb, 4 for hind limb, flexed lateral is optional for hind limb)

Dorsopalmar/plantar projection, elevated 15°
Dorsolateral (30°) to palmaromedial oblique (elevated 10°)
Dorsomedial (30°) to palmarolateral oblique (elevated 10°)
Lateral to medial projection of the fetlock joint
Lateral to medial (flexed) projection of the fetlock joint

Dorsopalmar/plantar projection, elevated 15° of the fetlock joint (Fig. 1).

At the public auction of Thoroughbreds, the pastern joint is included on the DP projection, therefore common RA in the pastern and the fetlock will be discussed. On this projection, the areas of greatest concern are the distal condyles of the third metacarpus/metatarsus (MC/MT3) and the distal P1 as well as the opposing articular surfaces on the proximal first phalanx (P1) and second phalanx (P2). The distal border of the proximal sesamoids should also be closely scrutinized.

Abnormalities best identified on the fetlock DP view are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Lucencies in distal MC/MT3, proximal P1, distal P1, or proximal P2**
(2) Enthesophytes of distal aspect of proximal sesamoid bones (PSB)
(3) Osteochondral fragments distal to the base of the proximal sesamoid bones
(4) Osteochondral fragments at the articular margin of proximal P1
(5) Lucencies in the sagittal ridge of MC/MT3
(6) Peri-articular osteophytes affecting the medial and lateral joint margins

Lateral to medial projection of the fetlock joint (Fig. 2).

Areas of greatest concern on this projection include the proximal dorsal margin of P1, the dorsal sagittal ridge, the apex and base of the proximal sesamoid bones, the palmar/plantar fetlock joint recess, and the proximal palmar/plantar margin of P1.

Abnormalities best identified on the fetlock lateral to medial projection are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Axial dorsal-proximal P1 fragments: in young horses, these fragments are often axial in the sagittal groove and do not project well on oblique views**
(2) **Irregularities or fragments of the sagittal ridge**
(3) Fragments of the base of the sesamoid bones

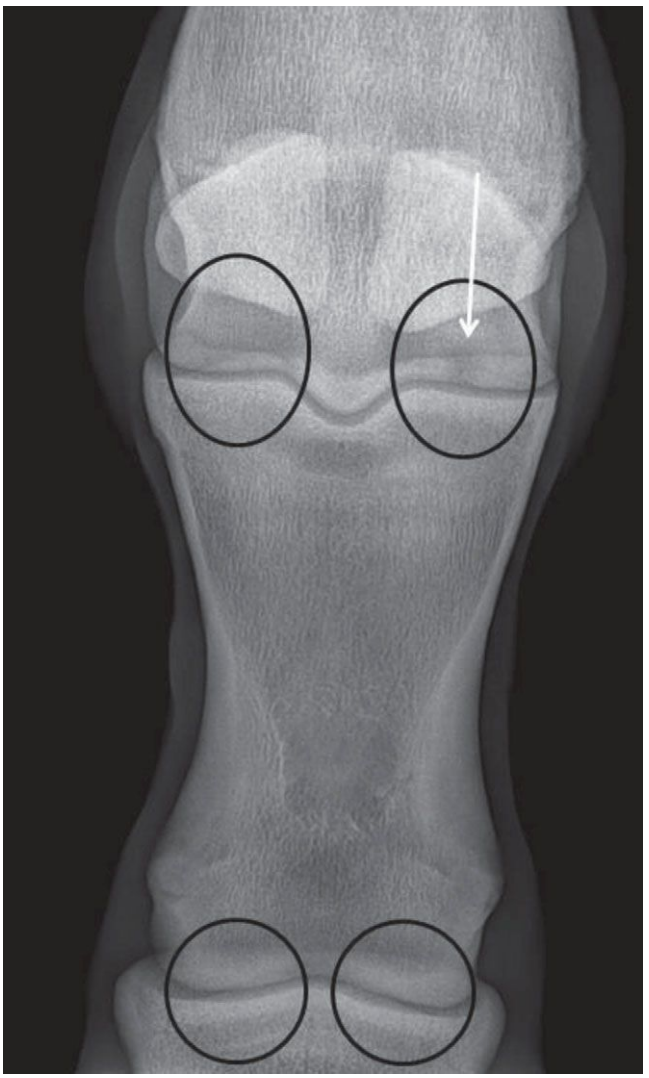


Fig. 1. Fetlock joint dorsopalmar/plantar projection, elevated 15°. This horse has a lucency in the distal metacarpus (arrow). This radiograph will be discussed by the panel as Case 1.

(4) **Supracondylar lysis of MC/MT3**
(5) Apical sesamoid fragments
(6) Palmar/plantar proximal P1 fragments



Fig. 2. Fetlock joint lateral to medial projection. This horse has moderate (3- to 4-mm) supracondylar lysis of the distal metacarpus (arrow). This radiograph will be discussed by the panel as Case 2.

Lateral to medial (flexed) projection of the fetlock joint (Fig. 3).

The area of greatest concern on this projection is the distal aspect of MC/MT3, particularly the sagittal ridge, and **will detect fragments and lucencies in the mid-sagittal ridge.**

Case Prognosis Summary

Case 1. Lucency in the Distal Metacarpal Condyle

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No		owner	buyer	
Western performance	No		owner	buyer	
English performance	No		owner	buyer	
General purpose	No		owner	buyer	

Case Prognosis Summary

Case 2. Moderate Supracondylar Lysis of the Distal Metacarpus

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No			owner/buyer	
Western performance	No			owner/buyer	
English performance	No			owner/buyer	
General purpose	No			owner/buyer	

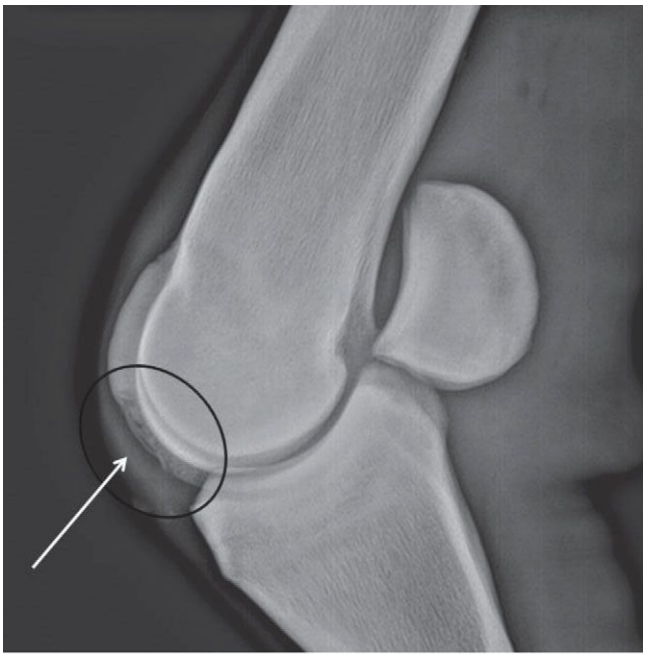


Fig. 3. Fetlock joint flexed lateral to medial projection. This horse has large irregularity in the middle of the sagittal ridge. This radiograph will be discussed by the panel as Case 3.



Fig. 4. Fetlock joint dorsolateral (30°) to palmaromedial oblique (elevated 10°). This horse has a large lateral proximal P1 palmar process fragment with two smaller intra-articular fragments. This radiograph will be discussed by the panel as Case 4.

Dorsolateral (30°) to palmaromedial oblique (elevated 10°) and dorsomedial (30°) to palmarolateral oblique (elevated 10°) projections (Fig. 4).

Areas of greatest concern on these projections are the proximal-dorsal aspect of P1, the abaxial aspect of the superimposed sesamoid, the apex and suspensory branch attachment of the palmar/plantar silhouetted sesamoid, and proximal P1.

Abnormalities best identified on the fetlock oblique projections are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Abaxial proximal-dorsal P1 osteochondral fragments**
(2) **Sesamoid fractures: apex, abaxial, mid-body, base**
(3) **Sesamoid enthesophytes (proximal or distal)**
(4) **Osteochondral fragments at the palmar/plantar margin of proximal P1**
(5) **Irregular or incompletely ossified palmar/plantar eminences**
(6) Irregularities or osteochondral fragments of the dorsal lateral/medial abaxial aspect of distal MC/MT3
(7) Lucencies or fragments in the palmar/plantar condyle of MC/MT3
(8) Distal MC/MT2 and four fractures

Case Prognosis Summary

Case 3. Large Irregularity in the Middle Aspect of the Sagittal Ridge

Performance Goal	Surgery Yes/No/Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No		owner/buyer		
Western performance	No		owner/buyer		
English performance	No		owner/buyer		
General purpose	No		owner/buyer		

Case Prognosis Summary

Case 4. Three Proximal P1 Palmar Process Fragments

Performance Goal	Surgery Yes/No/Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes		owner	buyer	
Western performance	Yes		owner/buyer		
English performance	Yes		owner/buyer		
General purpose	Maybe		owner/buyer		



Case 5. Multiple large fragments at the base of the sesamoid.



Case 6. Lucency with fragment palmar metacarpus.

3. Determining the Significance of an RA

Several studies of young stock have examined associations between fetlock and pastern RA and performance.[2,3,14–17] These studies can be challenging to interpret because they have many confounding factors, often have a selection bias, frequently struggle to achieve statistical significance, and have incomplete outcome information, usually racing data only. Also, retrospective performance studies of young stock vary in the classification of RA and have in general not been able to account for lesion size, the severity of associated changes (sclerosis, periosteal reaction), and the position in the joint, usually because of small numbers of affected horses. Further complicating the analysis is the impact of having multiple affected limbs or more than one kind of RA. However, there are several publications that focus on the treatment of a specific fetlock lesion, and these studies have been able to associate poorer prognosis with some lesion characteristics.[18–22] Clinicians must judge young stock with RA on the basis of careful reading of available studies, experience with RA in a given performance discipline, and evaluation of the appearance and clinical presentation of a specific RA.

Case Prognosis Summary

Case 5. Multiple Large Fragments, Base of Sesamoid

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No			owner	buyer
Western performance	No			owner	buyer
English performance	No			owner/buyer	
General purpose	No		owner/buyer		

Case Prognosis Summary

Case 6. Lucency With Fragment Palmar Metacarpal Condyle

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes		owner	buyer	
Western performance	Yes		owner	buyer	
English performance	Yes		owner	buyer	
General purpose	No		owner/buyer		



Case 7. Irregular surface to proximal medial sesamoid and small chip fracture of axial base.



Case 8. Large fragment at proximal MSR and irregular dorsal surface to distal metacarpus.

4. RA in the Fetlock or Pastern That Have Been Associated With Poorer Racing Outcomes Excluding Monetary Outcomes

Values are statistically significant at $P \leq 0.05$. RA include the following.

- Greater than 4-mm supracondylar lysis of MC3[14]
- Enthesophyte formation on proximal sesamoids (fore or hind)[14]
- Midline pastern joint lucencies (large)[16]
- Abnormal shape of PSB[2]
- Sesamoid fracture (forelimb)[2]
- Large-defect sagittal ridge MT3[2]
- Enlarged (>2 mm) vascular canals in PSB[17]

5. Trends Associated With Poorer Racing Performance

Values are significant at $P < 0.2$. Trends include the following.

- Fragment dorsal-proximal palmar P1[14]
- Sesamoid of abnormal shape[14]
- Fragment proximal-palmar P1[14]
- Abnormal vascular channels in the proximal sesamoid bones[2,14]
- Defect of sagittal ridge MC3[2]
- PSB lucency[2]
- Fragment plantar P1[2]

Case Prognosis Summary

Case 7. Irregular Abaxial Surface of PSB and Small Chip Fracture, Axial Base

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes		owner/buyer		
Western performance	No		owner/buyer		
English performance	No		owner/buyer		
General purpose	No		owner/buyer		

Case Prognosis Summary

Case 8. Large Fragment at Proximal Sagittal Ridge and Irregular Surface to Abaxial Metacarpus

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes			owner	buyer
Western performance	Yes			owner/buyer	
English performance	Yes			owner	buyer
General purpose	Yes		owner/buyer		



Case 9. Small osteochondral fragment dorso-proximal P1 and small apical fracture of proximal sesamoid.

6. Prognosis for Fetlock RA After Treatment

Prognoses for fetlock RA after treatment include the following.

- Sesamoid fracture; forelimbs have reduced racing (55%) versus hind limb (86%); medial forelimb fractures have the worst prognosis (44%)[18]
- Fragment removal dorso-proximal P1; 73% of TB return to racing, 25% at a lower class[19]
- Fragment removal palmar/plantar proximal P1; 76% to 91% return to racing[20,21]
- Debridement of subchondral bone cyst, distal MC3; 80% successful[22]



Case 10. Large circular lucency palmar margin of sesamoid.

7. Cases

The summary prognosis charts for Cases 1 through 10 summarize the opinion of the author and are predicated on the lesion being detected in a yearling intended for one of the four disciplines. The focus is on performance and not resale blemishes. The prognosis given to a horse owner and the prognosis given to a potential buyer, if different, is indicated.

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Case Prognosis Summary

Case 9. Small Fragment, Dorso-Proximal P1 and Small Apical Fracture PSB

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes	owner/buyer			
Western performance	Maybe	owner/buyer			
English performance	Maybe	owner/buyer			
General purpose	No	owner/buyer			

Case Prognosis Summary

Case 10. Large Lucency, Palmar Abaxial Margin of Medial Sesamoid

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No		owner	buyer	
Western performance	No		owner/buyer		
English performance	No		owner/buyer		
General purpose	No		owner/buyer		

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Radiography of the Carpus and Hock

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1. Introduction

Radiography of the carpus and hock are routine procedures performed by most equine veterinarians. When evaluating a performance issue or obtaining radiographs for purchase examination, use of the proper technique to obtain radiographs of the carpus and hock is essential. Use of the proper technique produces radiographs of diagnostic quality, allowing for a detailed analysis and interpretation of the joints and associated structures. Poor-quality radiographs make interpretation much more challenging. Many excellent textbooks and review articles are available for a more in-depth discussion of this topic.[1,2]

General Comments

High-quality radiographs are a sum of various components that must come together effectively to create diagnostic images. Some important factors are as follows.

- Safety first. Do not forget that radiographic procedures involve ionizing radiation that is not seen or felt. Follow all safety protocols to minimize exposure to the horse, handlers, and those in the area.
- Know your equipment. A thorough understanding of the generator and imaging system is imperative to obtain quality images. Not all systems function the same, and a complete understanding of how a particular system works in conjunction with the generator is imperative.
- Develop a technique chart. A technique chart for the various anatomic regions that is based on the available equipment will improve image quality. Although most digital systems are very tolerant of poor technique, they will excel when proper techniques are used.
- Know your anatomy and do not be afraid to experiment. Knowledge of the anatomy of the region allows for better radiographs. Sometimes, routine positioning will not highlight a problem as well as does a novel position. In particular, specialized oblique projections can be extremely useful.
- Develop a routine. Repetitive, practiced motions result in better, high-quality images. Although not everyone's routine will be the same, staying with a routine will help to obtain consistent, high-quality images.
- Use physical markers. Whereas most digital systems provide digital markers for laterality, relying on these markers can be risky. If the digital plate is positioned in a way not envisioned by the programmers, the laterality may be mis-marked. Physical markers that are properly placed allow for quick and easy con-

NOTES

Table 1. Routine Radiographic Projections of the Carpus

Projection	Racing (Thoroughbred)	Western Performance (Quarter Horse)	English Performance (Warmblood)	Arabian
Dorsal-palmar	X	X	X	X
Lateral-medial	X	X	X	X
Dorsal 35° lateral-palmar medial oblique	X	X	X	X
Dorsal 25° medial-palmar lateral oblique	X	X	X	X
Flexed-lateral	X	±	±	±
Dorsal proximal–dorsal distal oblique–distal row of carpal bones	X	–	–	–
Dorsal proximal–dorsal distal oblique–proximal row of carpal bones	±	–	–	–
Dorsal proximal–dorsal distal oblique–distal radius	±	–	–	–

X indicates strongly recommended on all examinations; ±, should be considered if clinically warranted; –, indicates not routinely performed but may be considered if clinically indicated.

formation of laterality. Additionally, the application of radio-opaque markers directly on the skin or region of interest can help to identify lesions on challenging cases.

The Carpus

The carpus is essentially round, from an external standpoint, and this allows relatively free access to all sides of the limb for excellent evaluation of the bony structures within the knee. Views will often depend on the age, breed, and use of the horse. A list of the "routine" projections and the relative frequency of the views on the basis of use is provided in Table 1.

Examples of routine radiographic projections of the carpus are shown in Figs. 1–6.

The views shown in Figs. 7 and 8 would be considered specialized views. Good information is given in these radiographs, but would you call them routine?

The Tarsus

The hock is essentially round, from an external standpoint, with a protuberance at the proximal calcaneus. Similar to the carpus, there is relatively free access to all sides of the limb for excellent evaluation of the bony structures. The views will depend on the age, breed, and use of the horse. A list of the routine projections by discipline is provided in Table 2.

Examples of routine radiographic projections of the tarsus are shown in Figs. 9–12.

Specialized views are shown in Figs. 13 and 14.

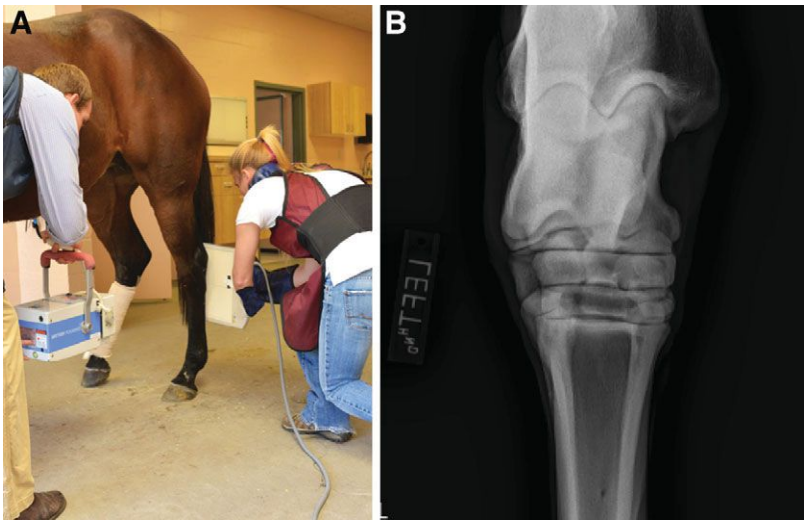


Fig. 1. Routine radiographic projection of the carpus: Dorsal-palmar view.

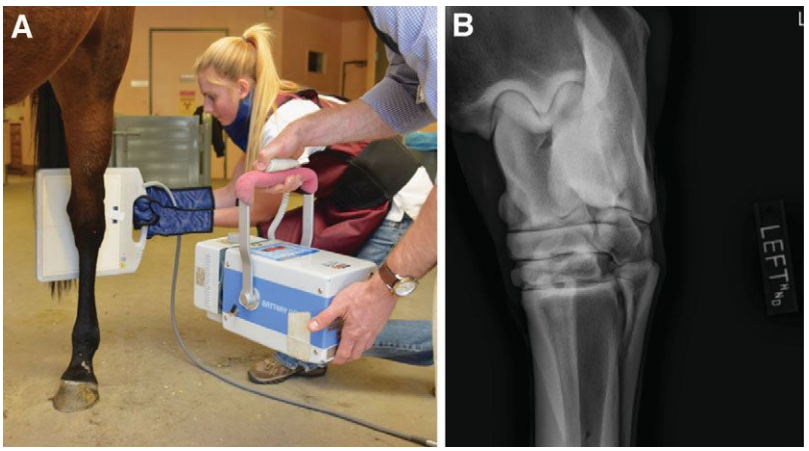


Fig. 2. Routine radiographic projection of the carpus: Flexed lateral view.

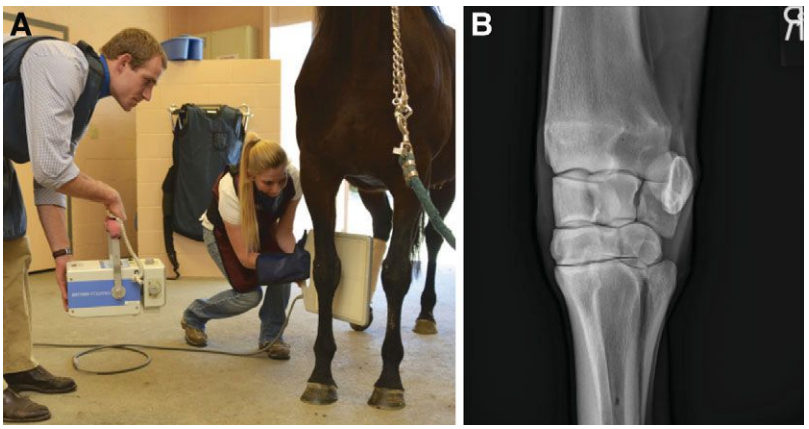


Fig. 3. Routine radiographic projection of the carpus: Dorsal 35° lateral–palmar medial oblique view.

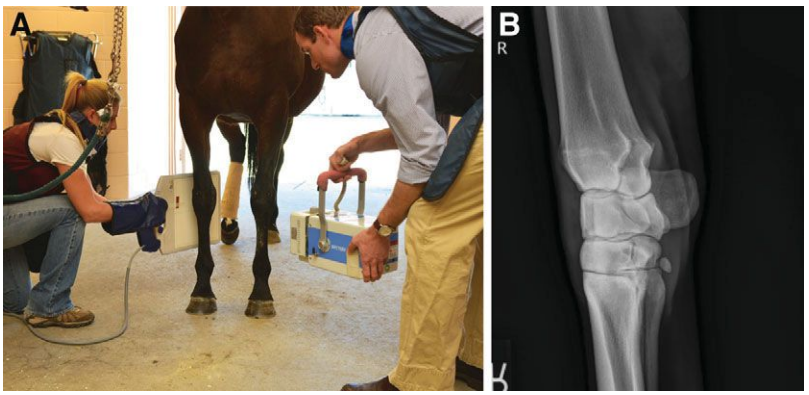


Fig. 4. Routine radiographic projection of the carpus: Dorsal 25° medial–palmar lateral oblique view.

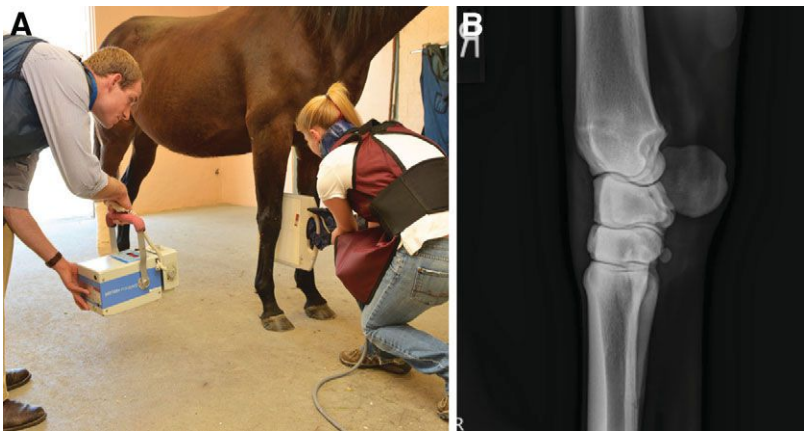


Fig. 5. Routine radiographic projection of the carpus: Lateral-medial view.

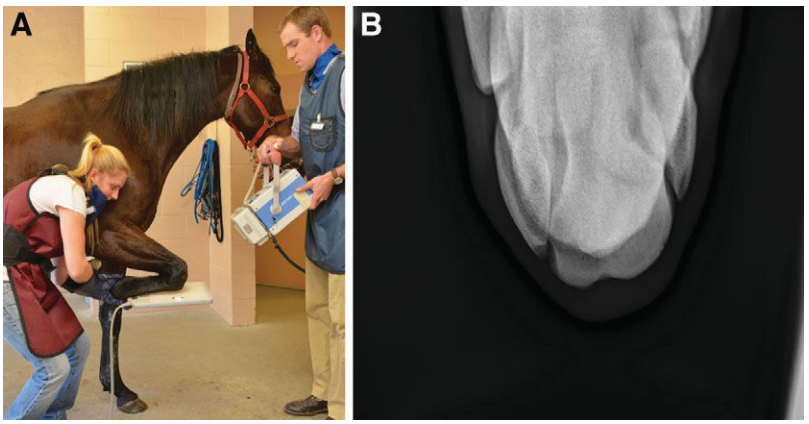


Fig. 6. Routine radiographic projection of the carpus: View of the dorsal proximal–dorsal distal oblique–distal row of carpal bones.

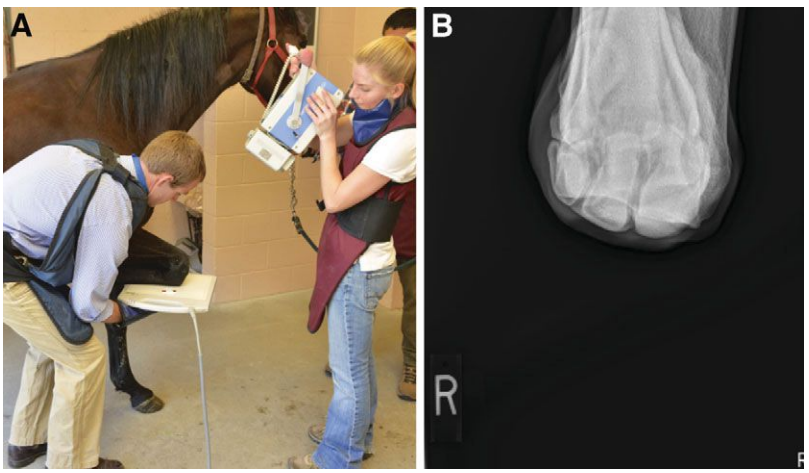


Fig. 7. Specialized view: dorsal proximal–dorsal distal oblique–proximal row of carpal bones.

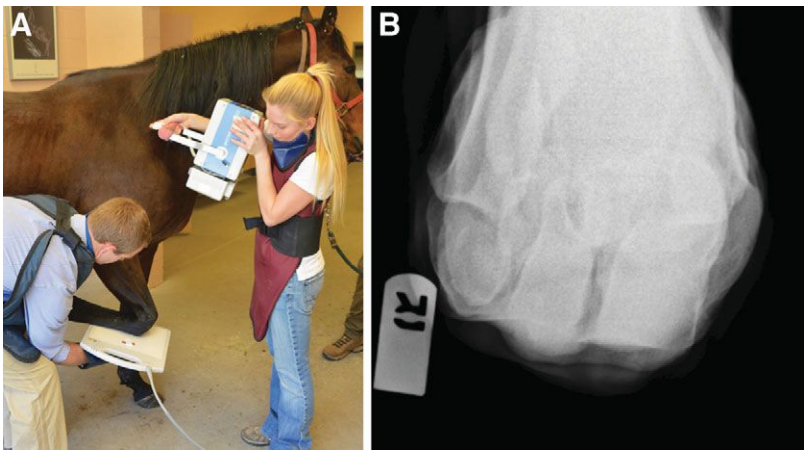


Fig. 8. Specialized view: dorsal proximal–dorsal distal oblique–distal radius.

Table 2. Routine Radiographic Projections of the Tarsus

Projection	Racing (Thoroughbred)	Western Performance (Quarter Horse)	English Performance (Warmblood)	Arabian and General Purpose	Other
Dorsal-plantar (dorsal 10° lateral–plantar medial oblique)	X	X	X	X	X
Lateral-medial	X	X	X	X	X
Dorsal 45° lateral–plantar medial oblique	X	X	X	X	X
Dorsal 65° medial–palmar lateral oblique	X	X	X	X	X
Flexed-lateral	–	–	–	–	–
Flexed caudal proximal-caudal distal oblique (Sustentaculum Tali Skyline projection)	–	–	–	–	–

X indicates strongly recommended on all examinations; –, indicates not routinely performed but may be considered if clinically indicated.

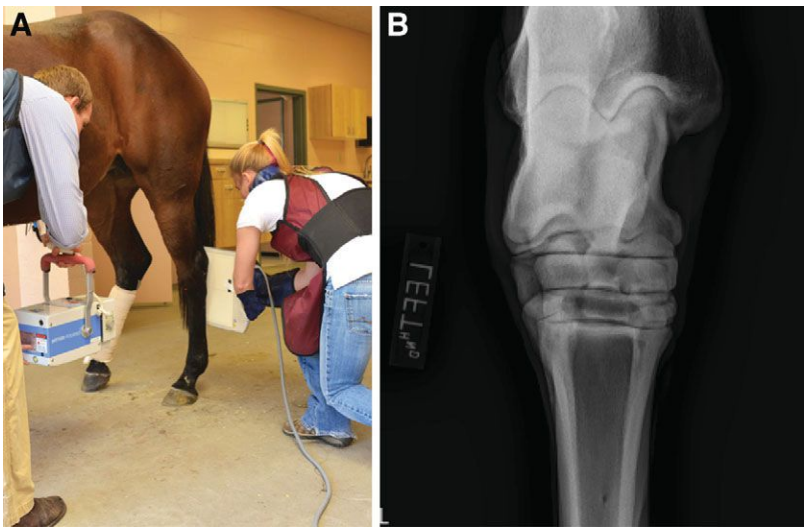


Fig. 9. Routine radiographic projection of the tarsus: Dorsal-plantar view.

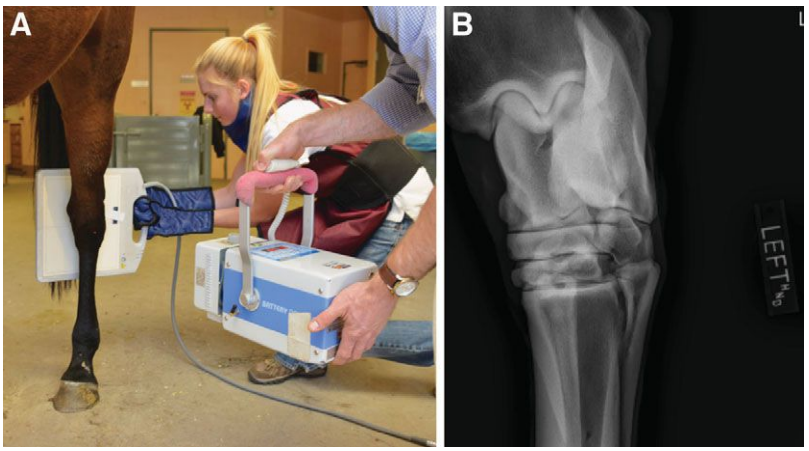


Fig. 10. Routine radiographic projection of the tarsus: Dorsal 45° lateral–plantar medial oblique view.

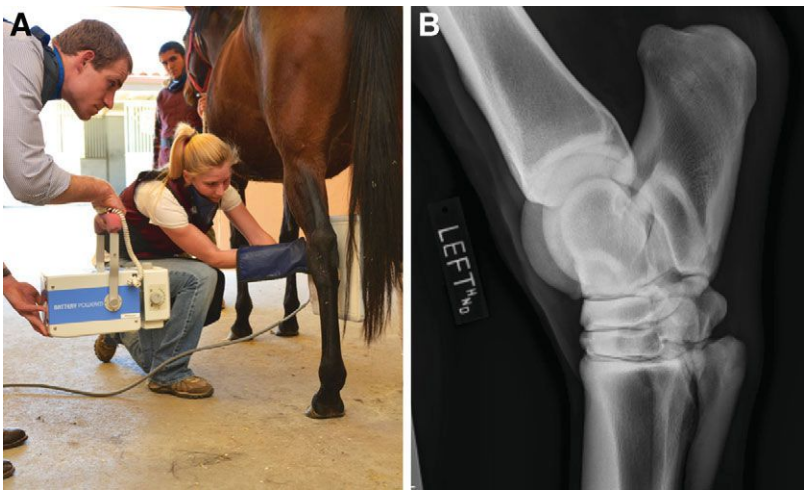


Fig. 11. Routine radiographic projection of the tarsus: Lateral-medial view.

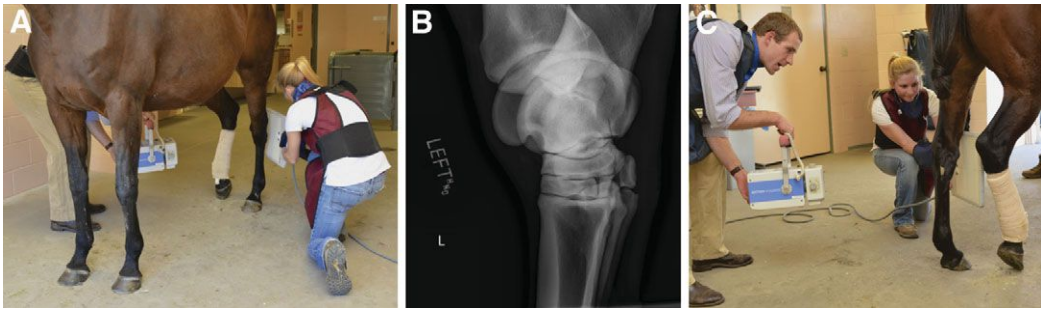


Fig. 12. Routine radiographic projection of the tarsus: Dorsal 65° medial–palmar lateral oblique view.

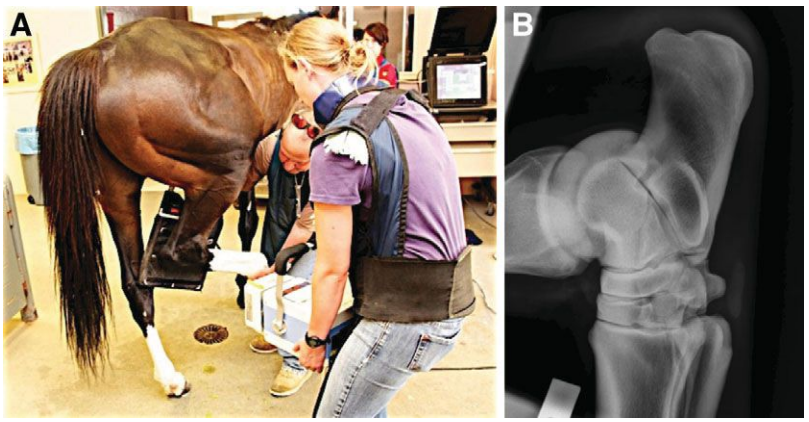


Fig. 13. Specialized view: Flexed lateral.

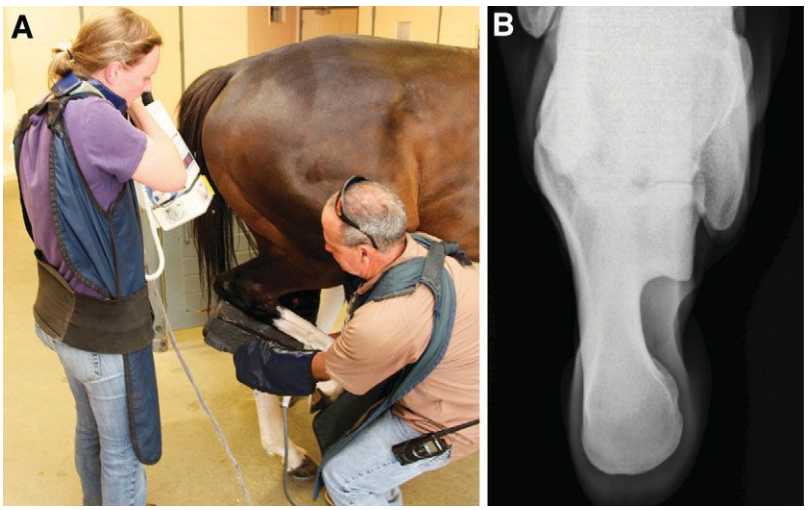


Fig. 14. Specialized view: Flexed caudal proximal-caudal distal oblique (Skyline projection of calcaneus).

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How to Interpret Radiographs of the Carpus and Tarsus of the Young Performance Horse

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1. Introduction

Routine radiographic evaluation of young performance horses has become commonplace in Thoroughbreds but also occurs in other performance breeds. This practice has resulted in the frequent discovery of radiographic abnormalities (RA) that are often clinically silent but can cause concern in buyers and sellers of young performance stock. The prevalence of radiographic developmental orthopedic disease in 6-month-old horses has been reported to be 25% in Warmbloods, 41% in Standardbreds, and 34% in Thoroughbreds.[1] In Thoroughbred yearlings intended for racing, 86.3% exhibit RA[2]; in young Standardbreds, 42%[3]; in yearling Warmblood horses, 69.5%[4]; and in 1- and 2-year-old Quarter Horses intended for cutting, 89%.[5] Predicting the significance of RA to future performance can be challenging and frustrating for buyers and sellers, which is further complicated by the possibility of treatment, including surgery, on prognosis.

2. Identifying RA

Because radiographic images are reviewed in sets of projections of one joint, this paper will discuss abnormalities by each projection. Examiners are responsible for reading the entire image; however, there are areas that are more commonly affected by RA, and they should receive the closest scrutiny. These will be denoted by black circles on the radiographic images included in these proceedings. RA best detected on a specific projection will be listed in bold, but should be confirmed on other projections when possible. Finally, readers should always consider three general factors when reviewing radiographic images:

(1) Make sure that the films are of the correct horse.

(2) Make sure the date is appropriate and consistent.

(3) Make sure all required views are present and of acceptable quality.

3. Carpus

Most radiographic abnormalities in the carpus of young horses can be imaged on three projections: dorsolateral (35°) to palmaromedial oblique, dorsomedial (25°) to palmarolateral oblique, and flexed lateral to medial. A skyline of the distal row can add information if injury to the dorsal surface of the carpal bones is suspected. At Thoroughbred sales, this view is used only for horses that have raced and is not routine for younger stock. Traditional descriptions of carpal views also include a dorsopalmar

NOTES



Fig. 1. Carpus dorsolateral (35°) to palmaromedial oblique projection. This image demonstrates remodeling of the distal radial and proximal third carpal bones (white circle). This radiograph will be discussed by the panel as Case 1.



Fig. 2. Carpus dorsomedial (25°) to palmarolateral oblique projection. This image demonstrates remodeling of the distal intermediate carpal bone (white circle). This radiograph will be discussed by the panel as Case 2.

and standing lateral projection, but, in the interest of controlling cost and radiation exposure, are not included in sale radiographs.

Dorsolateral (35°) to Palmaromedial Oblique Projection of the Carpus (Fig. 1).

The areas of greatest concern on this view are the dorsomedial aspect of the distal radius and the proximal radial, distal radial, and proximal third carpal bones. The radial articulation of the accessory carpal bone, the palmar aspect of the ulnar carpal bone, and the palmar pouch of the middle carpal joint are also of interest.

Abnormalities best identified on the dorsolateral (35°) to palmaromedial oblique are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Osteochondral fragments or remodeling of the distal radial carpal bone**

(2) **Osteochondral fragments or remodeling of the proximal third carpal bone**

(3) **Osteochondral fragments or remodeling of the distomedial radius**

(4) Osteochondral fragments or remodeling of the proximal radial carpal bone

(5) Fractures of the radial articulation of the accessory carpal bone

(6) Palmar fragments in the middle carpal joint

(7) Ulnar carpal bone lucencies or axial fragments

Dorsomedial (25°) to Palmarolateral Oblique Projection of the Carpus (Fig. 2).

The areas of greatest concern on this view are the dorsolateral aspect of the distal radius and the proximal intermediate, distal intermediate, and proximal third carpal bones. The ulnar carpal bone and the palmar pouch of the middle carpal joint are also of interest.

Abnormalities best projected on the dorsomedial (25°) to palmarolateral oblique projection of the carpus are listed below. If the RA is in bold type, it is the best projection to detect that RA.

Case Prognosis Summary

Case 1. Remodeling of the Distal Radial and Proximal Third Carpal Bones

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes				owner/ buyer
Western performance	Yes			owner/ buyer	
English performance	Yes			owner/ buyer	
General purpose	Yes			owner	buyer

Case Prognosis Summary

Case 2. Remodeling of the Distal Intermediate Carpal Bone

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No			owner/ buyer	
Western performance	No		owner/ buyer		
English performance	No		owner/ buyer		
General purpose	No		owner/ buyer		

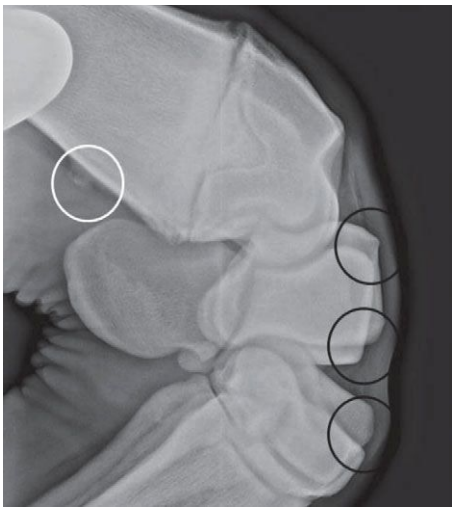


Fig. 3. Carpal flexed lateral to medial projection. This image demonstrates a small osteochondroma on the caudal radius (white circle). This radiograph will be discussed by the panel as Case 3.

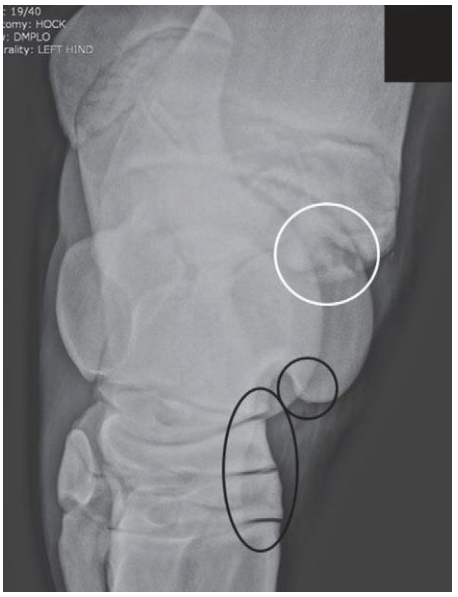


Fig. 4. Tarsal dorsomedial (65°) to plantarolateral oblique projection. This image demonstrates a DIRT lesion composed of multiple fragments (white circle). This radiograph will be discussed by the panel as Case 4.

(1) **Osteophytes on the proximal intermediate carpal bone**
(2) **Osteophytes or remodeling on the distal intermediate carpal bone**
(3) **Osteochondral fragments from the distocentral radius or proximal intermediate carpal bone**
(4) **Osteochondral fragments from the proximal third carpal bone**
(5) Osteochondral fragments from the plamar aspect of the radial carpal bone

Flexed Lateral to Medial Projection of the Carpus (Fig. 3).

The areas of greatest concern on this view are the proximal dorsal margin of the radial and intermediate carpal bones, the distal dorsal margin of the radial and intermediate carpal bones, the proximal dorsal margin of the third carpal bone, and the caudal aspect of the distal radius.

Abnormalities best projected on the flexed lateral to medial projection of the carpus are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Osteochondral fragments or remodeling of the distal radial carpal bone**
(2) **Osteochondral fragments or remodeling of the distal intermediate carpal bone**
(3) **Osteochondral fragments or remodeling of the proximal third carpal bone**
(4) **Osteochondroma formation on the caudal radius**

Carpal abnormalities that are believed to affect later athletic performance are not common and are generally reported to have a <7% prevalence in sales yearlings (Table 1). Lucencies in the ulnar carpal bone are detected with greater frequency but are generally considered insignificant blemishes.

Case Prognosis Summary

Case 3. Small Osteochondroma on the Caudal Distal Radius

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No		owner/ buyer		
Western performance	No		owner/ buyer		
English performance	No		owner/ buyer		
General purpose	No		owner/ buyer		

Case Prognosis Summary

Case 4. DIRT Lesion With Multiple Fragments

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes	owner/ buyer			
Western performance	Yes	owner/ buyer			
English performance	Yes	owner/ buyer			
General purpose	Maybe	owner/ buyer			

Table 1. Prevalence of Carpal Radiographic Abnormalities in Young Performance Horses

Carpal Radiographic Abnormality	Breed	Prevalence	Reference
Fragment	Thoroughbred	0.7–2.2%	2, 5, 8
Osteophytes	Thoroughbred	1.1–3.3%	2, 6–8
Osteophytes	Quarter Horse	3.5%	9
Enthesophyte	Thoroughbred	2.6%	2
Ulnar carpal bone lucency	Thoroughbred	8.3–22.2%	2, 6, 7
Dorsomedial carpal disease	Thoroughbred	2.7%	6
Dorsomedial carpal disease	Quarter Horse	6.4%	9
Subchondral cyst	Thoroughbred	0.2–0.3%	6, 8
Accessory carpal bone fracture	Thoroughbred	0.40%	6



Fig. 5. Tarsal dorsolateral (10°) to plantaromedial projection. This image demonstrates mild tarsocrural effusion (white arrows) and a large medial malleolar lucency with fragments (white circle). This radiograph will be discussed by the panel as Case 5.



Fig. 6. Tarsal lateral to medial projection. This image demonstrates a large enthesophyte on MT3 at the dorsal margin of the tarso-metatarsal joint and another on the distal aspect of the medial trochlear ridge (white circle). This radiograph will be discussed by the panel as Case 6.

4. Tarsus

Similar to the carpus, most radiographic abnormalities of the tarsus of young horses can be imaged on three projections: dorsomedial (65°) to plantarolateral oblique, dorsolateral (10°) to plantaromedial, and lateral to medial. Traditional descriptions of tarsal views include a lateral to medial oblique pro-

Case Prognosis Summary

Case 5. Large Medial Malleolar Lucency With Multiple Fragments

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes		owner/ buyer		
Western performance	Yes		owner/ buyer		
English performance	Yes		owner/ buyer		
General purpose	Yes		owner/ buyer		

Case Prognosis Summary

Case 6. Two Enthesophytes: Dorsal Margin of MT3 at the Tarsometatarsal Joint and Distal Aspect of the Medial Trochlear Ridge

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No		owner/ buyer		
Western performance	No		owner/ buyer		
English performance	No		owner/ buyer		
General purpose	No		owner/ buyer		

Table 2. Prevalence of Tarsal Radiographic Abnormalities in Young Performance Horses

Tarsal Radiographic Abnormality	Breed	Prevalence	Reference
Any	Warmbloods	31.4%	4
Any	Quarter Horse	68.2%	9
OCD of DIRT	Thoroughbred	3.4–5%	2, 5, 7, 8
OCD of DIRT	Quarter Horse	3.8%	2, 6–8
OCD of DIRT	Standardbred	14.5–17.7%	10, 11
OCD of medial malloleus of tibia	Thoroughbred	0.4–1.8%	2, 5–8
OCD of medial malloleus of tibia	Quarter Horse	1.10%	9
OCD of medial malloleus of tibia	Standardbred	2.5%	10, 11
OCD of lateral malloleus of tibia	Thoroughbred	0.5%	2
OCD medial trochlear ridge of talus	Thoroughbred	0.1–2.4%	2, 5, 7, 8
OCD medial trochlear ridge of talus	Quarter Horse	0.8%	9
OCD lateral trochlear ridge of talus	Thoroughbred	1–2.6%	2, 5, 7, 8
OCD lateral trochlear ridge of talus	Quarter Horse	2.3%	9
OCD lateral trochlear ridge of talus	Standardbred	1.6–3.8%	10, 11
Dorsal osteophytes PIT, DIT, TMT	Thoroughbred	20.1–25%	2, 5, 7, 8
Dorsal osteophytes PIT, DIT, TMT	Quarter Horse	47.9%	9
Fractures	Thoroughbred	0.1%	2
Lucency DIT, TMT	Thoroughbred	7.30%	5
Wedging of T3 or T central	Thoroughbred	0.6–1.6%	5–7
Wedging of T3 or T central	Quarter Horse	6.9%	9

OCD indicates osteochondrosis; DIRT, distal intermediate ridge of talus; PIT, proximal intertarsal joint; DIT, distal intertarsal joint; TMT, tarsometatarsal joint; T3, third tarsal bone; T central, central tarsal bone.



Case 7. Small DIRT fragment and large distal lateral trochlear ridge fragment.



Case 8. Remodeling of the dorsal margins of the central and third tarsal bones and proximal MT3.

Case Prognosis Summary

Case 7. Small DIRT Fragment and Large Distal Lateral Trochlear Ridge Fragment

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes	owner/ buyer			
Western performance	Yes	owner/ buyer			
English performance	Yes		owner/ buyer		
General purpose	Yes	owner/ buyer			

Case Prognosis Summary

Case 8. Remodeling of the Dorsal Margins of the Central and Third Tarsal Bones and Proximal MT3

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No		owner/ buyer		
Western performance	No			owner/ buyer	
English performance	No			owner/ buyer	
General purpose	No		owner/ buyer		



Case 9. Ulnar carpal bone lucency with fragment.



Case 10. Remodeling of the distal radial carpal bone.

jection, but, in young horses, this is of minimal additional benefit.

Dorsomedial (65°) to Plantarolateral Oblique Projection of the Tarsus (Fig. 4).

The areas of greatest concern on this view are the distal intermediate ridge of the tibia (DIRT), the lateral trochlear ridge of the talus, and the dorsolateral margin of the proximal and distal intertarsal and tarsometatarsal joints.

Abnormalities best projected on dorsomedial (65°) to plantarolateral oblique projection of the tarsus are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Osteochondral fragments on the distal intermediate ridge of the tibia**

(2) **Defects and osteochondral fragments of the lateral trochlear ridge of the talus**

(3) Osteoarthritis and fractures of the central and third tarsal bones

Dorsomedial (10°) to Plantarolateral Projection of the Tarsus (Fig. 5).

The areas of greatest concern on this view is the medial malleolus. However, tarsocrural effusion can also be best detected on this view, which can increase scrutiny of other typical locations of abnormalities, and, rarely, bony injury to the proximal metatarsus can be detected.

Abnormalities best projected on the dorsolateral (10°) to plantaromedial projection view of the tarsus are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Lucencies and osteochondral fragments on the axial aspect of the medial malleolus**

(2) **Tarsocrural effusion**

(3) Plantar damage on MT3 at suspensory origin

Lateral to Medial Projection of the Tarsus (Fig. 6).

The areas of greatest concern on this view are the distodorsal tibia, the dorsal aspect of both trochlear ridges, and the dorsal aspect of the proximal and distal intertarsal and the tarsometatarsal joints.

Case Prognosis Summary

Case 9. Ulnar Carpal Bone Lucency With Fragment

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No	owner/ buyer			
Western performance	No	owner/ buyer			
English performance	No	owner/ buyer			
General purpose	No	owner/ buyer			

Case Prognosis Summary

Case 10. Remodeling of the Distal Radial Carpal Bone

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Maybe		owner	buyer	
Western performance	No		owner/ buyer		
English performance	No		owner/ buyer		
General purpose	No		owner/ buyer		

Abnormalities best projected on the lateral to medial projection of the tarsus are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Degenerative joint disease of the distal intertarsal and tarsometatarsal joints**

(2) **Small axial DIRT fragments**

(3) Enthesophytes of proximodorsal MT3

(4) Osteochondral fragments in the proximal intertarsal joint

Tarsal abnormalities that are believed to affect later athletic performance are fairly common (31–68%) and the frequency appears to vary by breed (Table 2).[6–11]

5. Determining the Significance of an RA to Performance

Some studies of young stock have included RA of the carpus and tarsus in their attempts to associate young horse RA and performance.[2,12] These studies can be challenging to interpret by strict statistical methods ($P < 0.05$) because they have many confounding factors and incomplete outcome information, usually racing data only. Because of the overall low numbers of affected horses, subclassifying lesions (size, severity, number of limbs affected, etc) to determine prognosis is usually not possible. However, these studies do have important information to help guide veterinarians examining young performance horses. RA in the carpus or tarsus that have been associated with poorer racing outcomes ($P \leq 0.05$) include dorsal medial intercarpal joint disease and osteophytes in distal intertarsal or tarsometatarsal joints.[12] RA in the tarsus or carpus that demonstrate a tendency ($P < 0.2$) for poorer racing performance include carpal osteophytes, fracture of accessory carpal bone, change of medial trochlear ridge of talus,[12] and osteochondrosis of DIRT or medial malleolus.[2]

There also are multiple publications that focus on the treatment of a specific carpal or tarsal lesion, and some of these studies have been able to grade lesions and document that severity or size can affect prognosis.[13–17] Clinicians must judge young stock with RA on the basis of careful reading of available studies, experience with RA in a given performance discipline, and evaluation of the appearance and clinical presentation of a specific RA.

6. Prognosis for Carpal or Tarsal RA After Treatment

- Arthroscopy for osteochondral chip fractures of carpus: 68% race at level equal to or better than previous, 11% at lower level; greater cartilage damage results in lower success.[13]
- Arthroscopy for lucency of third carpal bone (Standardbreds); 75% race at equal level, 14% at lower.[14]
- Arthroscopy for lucency of distal radial carpal bone; 68% race at level equal to or better than previous, 12% at lower.[15]
- Arthroscopy for tarsocrural osteochondrosis (Standardbreds); 75% race after surgery.[16]
- Arthroscopy for tarsocrural osteochondrosis (Standardbreds);43% race at 3 years of age after surgery; Thoroughbreds, 78% race at 3 years of age.[17]

7. Cases

The summary prognosis charts (1–10) with each image are predicated on the lesion being detected in a yearling intended for one of the four disciplines. The focus is on performance and not resale blemishes. The prognosis given to a horse owner and the prognosis given to a potential buyer, if different, is indicated.

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How to Take and Interpret Radiographs of the Equine Stifle

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1. Introduction

Stifle radiography in the young performance horse is a fairly routine procedure for most equine practitioners. The primary reasons for taking radiographs of the stifle include radiographic evaluations for purchase, stifle joint effusion, lameness associated with the stifle, evaluation after a traumatic event associated with the stifle, and comparison of the perceived normal stifle to the affected stifle. Acquisition of stifle radiographs is not always intuitive for the beginner. A thorough knowledge of the 3D anatomy of the stifle as well as the anticipated pathologies of the stifle are necessary to properly position the x-ray generator and detector plate. Proper positioning results in acquisition of radiographs with sufficient quality to allow the veterinarian to make an informed diagnosis.[1–4]

2. Immature Horse

From birth until ~36 months of age, the stifle matures from a structure with six ossification centers: metaphysis and distal epiphysis of the femur, proximal epiphysis and metaphysis of the tibia, tibial tuberosity (apophysis), and patella. The lateral and medial trochlear ridges of the femur are initially fairly equal in size and the proximal portions of these ridges often have an irregular contour and opacity that must be differentiated from a suspected septic joint. This irregularity generally resolves by 3 to 5 months of age.

The distal femoral physis generally closes by 24 to 30 months of age. The physis between the tibial tuberosity and the tibial epiphysis tends to close by 9 to 12 months, and the physis between the tibial tuberosity and the tibial metaphysis closes at 30 to 36 months. The proximal tibial physis, between the proximal tibial epiphysis and the tibial metaphysis, closes at 24 to 30 months of age.

3. Stifle Joint Pathology

Osteochondrosis is reportedly the most common abnormality in the young performance horse stifle, affecting the trochlear ridges of the femur, the femoral condyles and the patella, and less frequently, the proximal tibia. Osteochondritis dissecans (OCD) of the trochlear ridges involves primarily the lateral trochlear ridge with the medial trochlear ridge less commonly affected. The articular surface of the patella also is a common site for OCD. Subchondral bone cysts are primarily observed in the medial femoral condyle, with much less frequency in the patella and even less frequently in the lateral femoral condyle.

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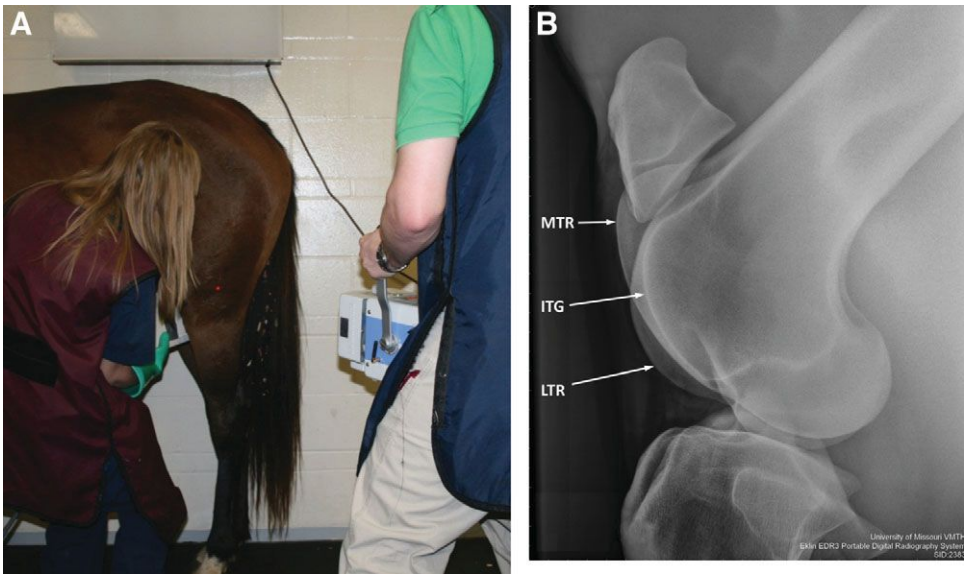


Fig. 1. (A) Positioning for lateromedial view. (B) Apparently normal lateromedial radiographic view of a mature Thoroughbred gelding highlighting the medial trochlear ridge (MTR), the lateral trochlear ridge (LTR), and the intertrochlear groove (ITG).

Other abnormalities include osteoarthritis, tumoral calcinosis (calcinosis circumscripta), collateral and patellar ligament desmitis, meniscal injuries, injuries to bone and soft tissues from acute

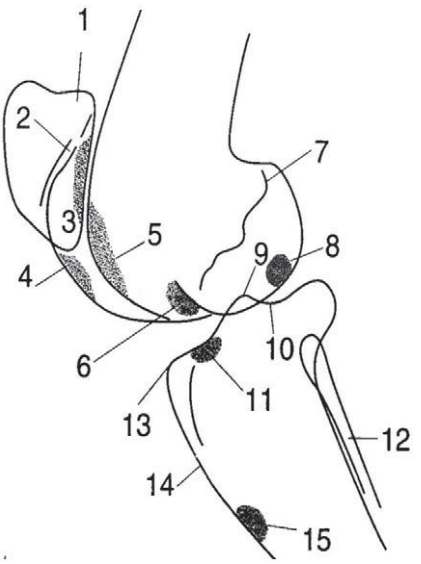


Fig. 2. Line drawing of lateromedial radiographic view identifying anatomical structures of importance. (1) Base of patella; (2) periarticular and subchondral areas of the patella*; (3) apex; (4) medial ridge of femoral trochlea*; (5) lateral ridge of femoral trochlea*; (6) area of extensor fossa*; (7) distal femoral physeal scar; (8) femoral condyles*; (9) tibial spine (medial intercondylar eminence); (10) tibial condyle; (11) area of attachment for the cranial ligaments of the menisci; (12) fibula; (13) tibial tuberosity; (14) tibial crest*; (15) areas of attachment for the tendon of semitendinosus muscle. *Common sites of pathology noted on lateromedial view. Adapted from O'Brien.[3]

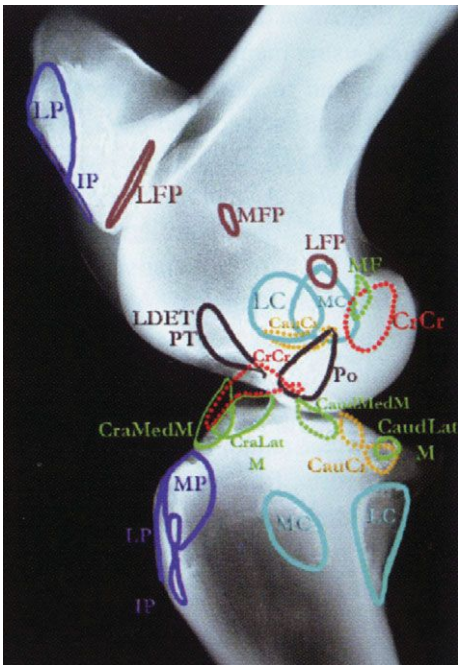


Fig. 3. Lateromedial radiographic map of ligament and tendon attachments to the stifle. Structures attaching within the intercondylar area of the tibia and the femur are indicated with dotted lines. From Maulet et al.[4] LP indicates lateral patellar ligament; IP, middle patellar ligament; MP, medial patellar ligament; LFP, lateral femoropatellar ligament; MFP, medial femoropatellar ligament; LC, lateral collateral ligament; MC, medial collateral ligament; CraLatM, cranial ligament of the lateral meniscus; CraMedM, cranial ligament of the medial meniscus; MF, meniscofemoral ligament of the lateral meniscus; CrCr, cranial cruciate ligament; CauCr, caudal cruciate ligament; Po, origin popliteus muscle; PT, origin of peroneus tertius; LDET, origin of long digital extensor; FP, femoropatellar joint capsule; LatFP, lateral femorotibial joint capsule; MedFT, medial femorotibial joint capsule.

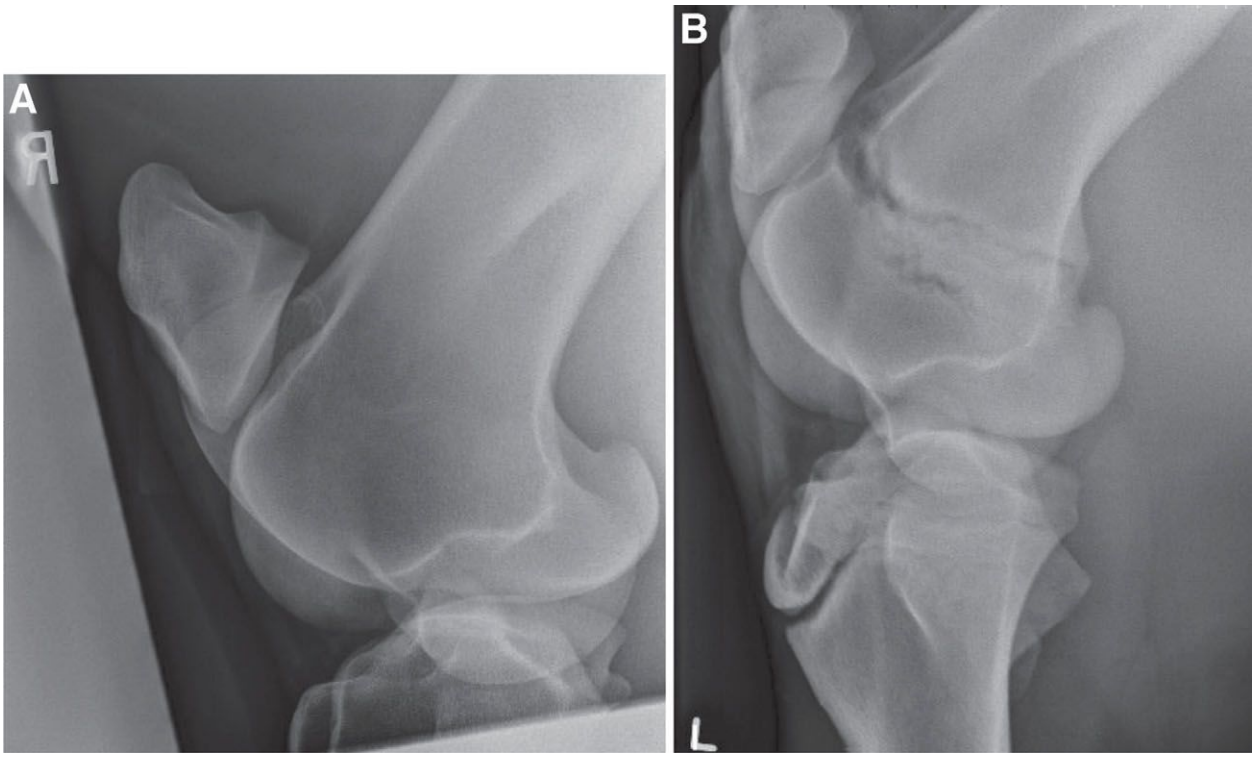


Fig. 4. A and B, Typical poor positioning for lateromedial view. Beam is directed at a proximal to distal angle instead of horizontal.

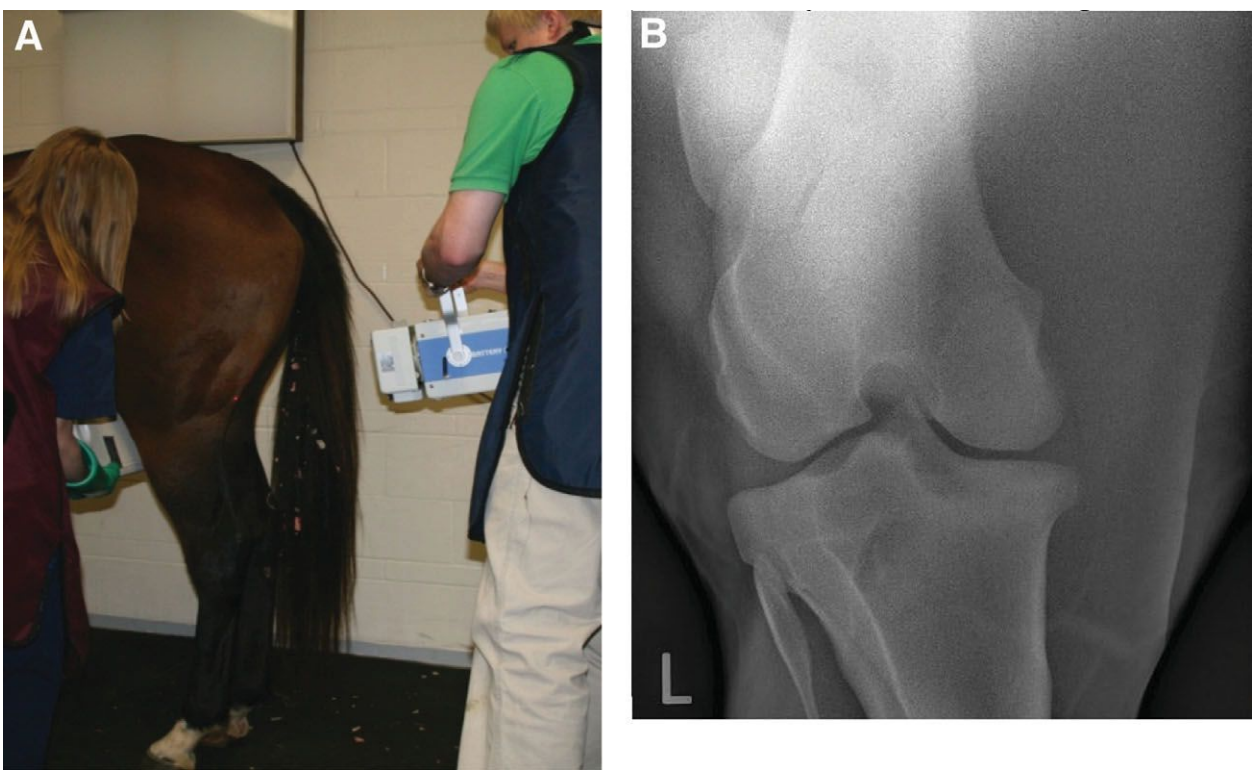


Fig. 5. (A) Positioning for caudocranial view. (B) Apparently normal caudal-cranial radiographic view of a mature Thoroughbred gelding.

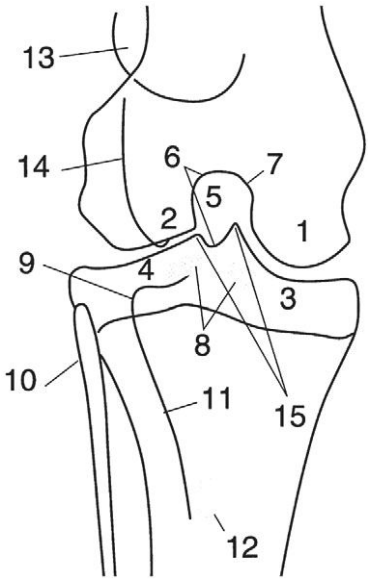


Fig 6. Line drawing of caudocranial radiographic view identifying anatomical structures of importance. (1) Medial femoral condyle; (2) lateral femoral condyle; (3) medial tibial condyle; (4) lateral tibial condyle; (5) intercondylar fossa of the femur; (6) areas of attachment of the cranial cruciate ligament; (7) area of attachment of the caudal cruciate ligament; (8) areas of attachment for the cranial ligaments of the menisci; (9) tibial tuberosity; (10) fibula; (11) tibial crest; (12) attachment site for the semitendinosus tendon; (13) patella; (14) lateral ridge of the trochlea of the femur; (15) intercondylar eminences: medial (spine) and lateral. Adapted from O'Brien.[3]

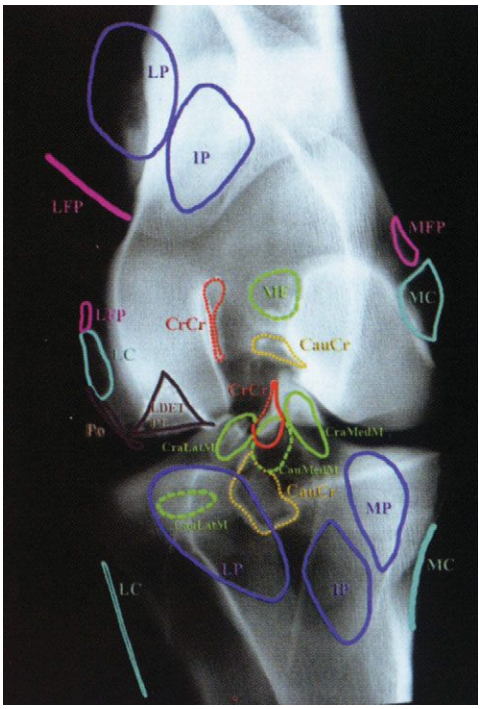


Fig. 7. Caudocranial radiographic map of ligament and tendon attachments to the stifle. Structures associated with the caudal half of the femur or tibia are indicated with dotted lines. From Maulet et al.[4] LP indicates lateral patellar ligament; IP, middle patellar ligament; MP, medial patellar ligament; LFP, lateral femoropatellar ligament; MFP, medial femoropatellar ligament; LC, lateral collateral ligament; MC, medial collateral ligament; CraLatM, cranial ligament of the lateral meniscus muscle; CraMedM, cranial ligament of the medial meniscus; MF, meniscofemoral ligament of the lateral meniscus; CrCr, cranial cruciate ligament; CauCr, caudial cruciate ligament; Po, origin popliteus muscle; PT origin of peroneus tertius; LDET, origin of long digital extensor; FP, femoropatellar joint capsule; LatFP, lateral femorotibial joint capsule; MedFT, medial femorotibial joint capsule.

traumatic events, and other miscellaneous sclerosis and lucencies of the trochlear ridges and proximal tibia).

4. Radiographic Views

There are three standard radiographic views that are commonly acquired for most stifle evaluations, including the lateromedial, caudal to cranial (10–20° proximodistal), and 30° caudolateral-craniomedial oblique. Optional views that are not acquired routinely, but are necessary views to highlight specific anatomic structures, include the patellar skyline, flexed lateromedial, and 30° to 45° caudomedial-craniolateral oblique views. In addition, special oblique projections can be very useful for specific joint abnormalities when the standard radiographic views do not sufficiently demonstrate the abnormality.

Lateromedial View

The horse should be positioned with both hind limbs fully weight-bearing with the limb to be imaged slightly caudal to the contralateral limb (Figs. 1–4). The x-ray beam should be directed horizontally, centered on the femorotibial joint, caudal and distal to the apex of the patella. The beam will most commonly be directed ~10° to 20° caudal to cranial to the frontal plane to accommodate for the typical hind limb anatomy of most horses. As a rule of thumb, align the beam parallel to the heel bulbs of the limb to be imaged.

The lateromedial view highlights the medial and lateral ridges of the femoral trochlea, the intertrochlear groove, the periarticular and subchondral areas of the patella, the tibial crest, the tibial plateau and areas of attachment of the cranial cruciate ligament and the cranial ligaments of the menisci, the extensor fossa, and the femoral condyles.

Caudal to Cranial View (10–20° Proximodistal)

The horse should be positioned with both hind limbs fully weight-bearing. The center of the beam should be directed at the soft tissue indentation at midline and the angle of the beam directed 10–20° proximodistally (Figs. 5–8).

30° Caudolateral-Craniomedial Oblique

The horse should be positioned with both hind limbs fully weight-bearing. The center of the beam

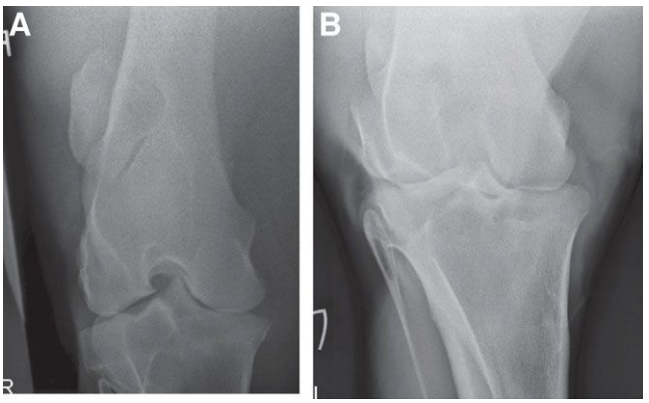


Fig. 8. A and B, Typical poor positioning for caudocranial view. Beam is directed horizontal rather than proximal to distal resulting in failure to achieve separation to evaluate femorotibial joint spaces.

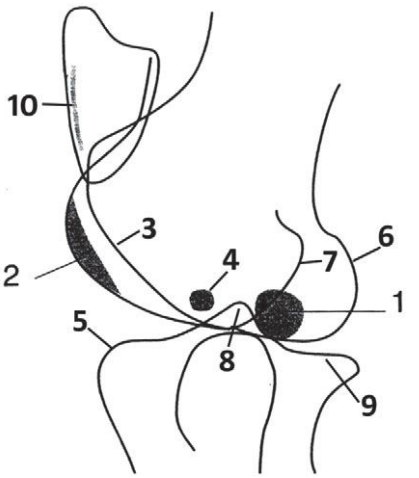


Fig. 10. Line drawing of a 30° caudolateral-craniomedial oblique view. The view is taken primarily to evaluate (1) site of typical subchondral cyst in medial femoral condyle; (2) typical area for OCD of the lateral trochlear ridge of the femur; (3) medial ridge of the femoral trochlea; (4) area of the extensor fossa; (5) tibial tuberosity; (6) medial femoral condyle; (7) lateral femoral condyle; (8) spine of the tibia; (9) medial condyle of the tibia; (10) attachment site for the biceps femoris and lateral patellar ligament. Adapted from O'Brien.[3]

should be directed on the femorotibial joint, ~10 cm caudal to the cranial aspect of the limb. The x-ray beam should be directed horizontal to the ground or slightly proximal to distal 10°–15° (Figs. 9–13).

Patellar Skyline (Cranioproximal-Craniodistal Oblique View)

The patellar skyline view is taken with the stifle flexed and the tarsus either flexed or extended. The cassette is held approximately horizontal with its caudal edge against the cranial aspect of the tibia, centered just proximal to the tibial crest. Depending on the horse, the beam angle may range from a proximal 10° lateral-distal medial oblique direction to a 30°–40° proximocranial-distocaudal direction. Adducting the flexed limb may facilitate positioning by rotating the stifle outwards.

The indications for the patellar skyline view include assessment of a suspected patellar fracture, and to better assess a known patellar fracture or suspected osteochondrosis of the patella or lateral trochlear ridge of the femur (Figs. 14 and 15).

Flexed Lateromedial View

Positioning is similar to the lateromedial view. The degree of flexion of the stifle can vary from just slightly flexed with the horse resting the toe on the ground to moderate flexion. The beam is directed horizontal, centered on the patella (Figs. 16 and 17).

The indications for the flexed lateromedial view include a better assessment of the apex of the patella, to

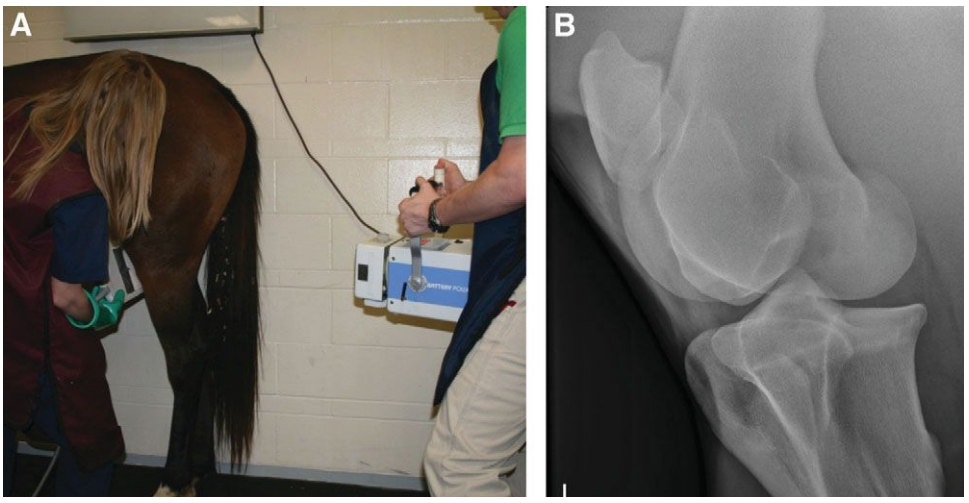


Fig. 9. (A) Positioning for 30° caudolateral-craniomedial oblique view. (B) Apparently normal 30° caudolateral-craniomedial oblique radiographic view of a mature Thoroughbred gelding.

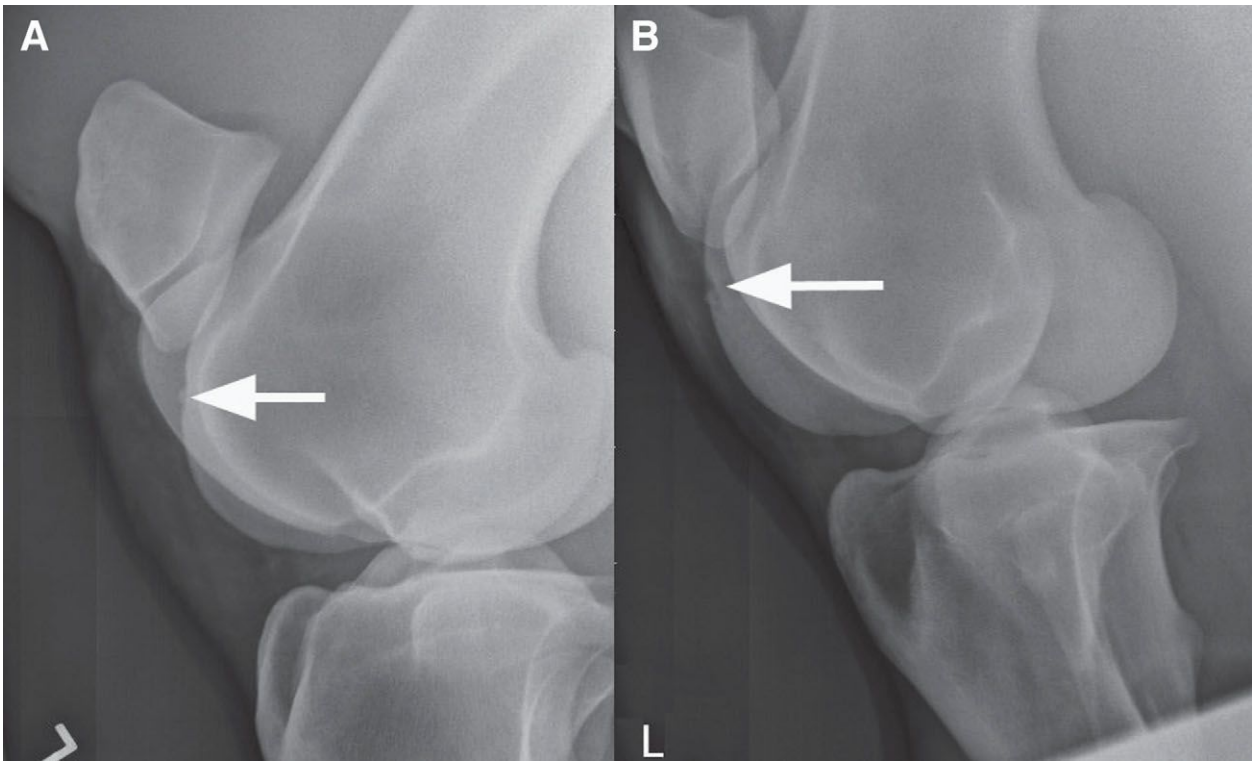


Fig. 11. (A) Lateromedial view and (B) 30° caudolateral-craniomedial oblique view of mild osteochondral defect (arrow) in the lateral trochlear ridge in an 18-year-old Warmblood gelding.

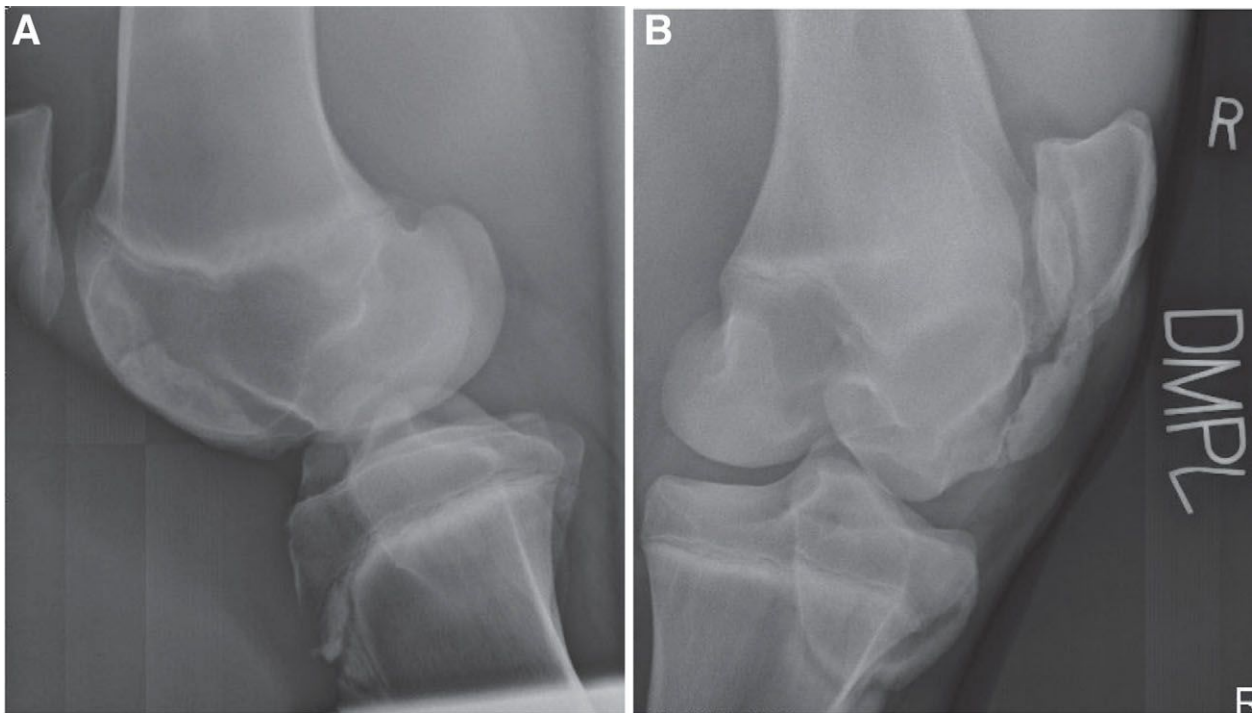


Fig. 12. (A) Lateromedial view and (B) 30° caudolateral-craniomedial oblique view of a large osteochondral fragment of the lateral trochlear ridge of the femur in a 15-month-old Standardbred filly.

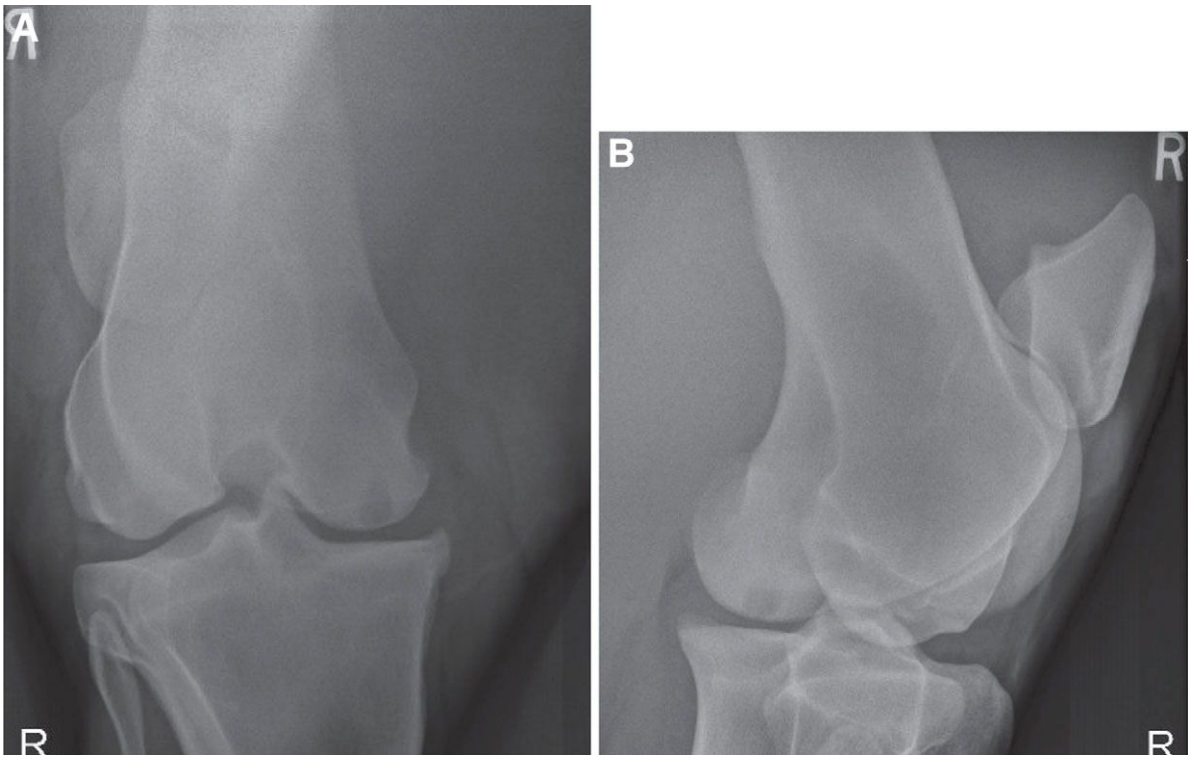


Fig. 13. (A) Caudo-cranial and (B) 30° caudolateral-craniomedial oblique view of small subchondral bone cyst of the medial femoral condyle with secondary mild osteophyte on medial tibial condyle in a mature Quarter Horse gelding.

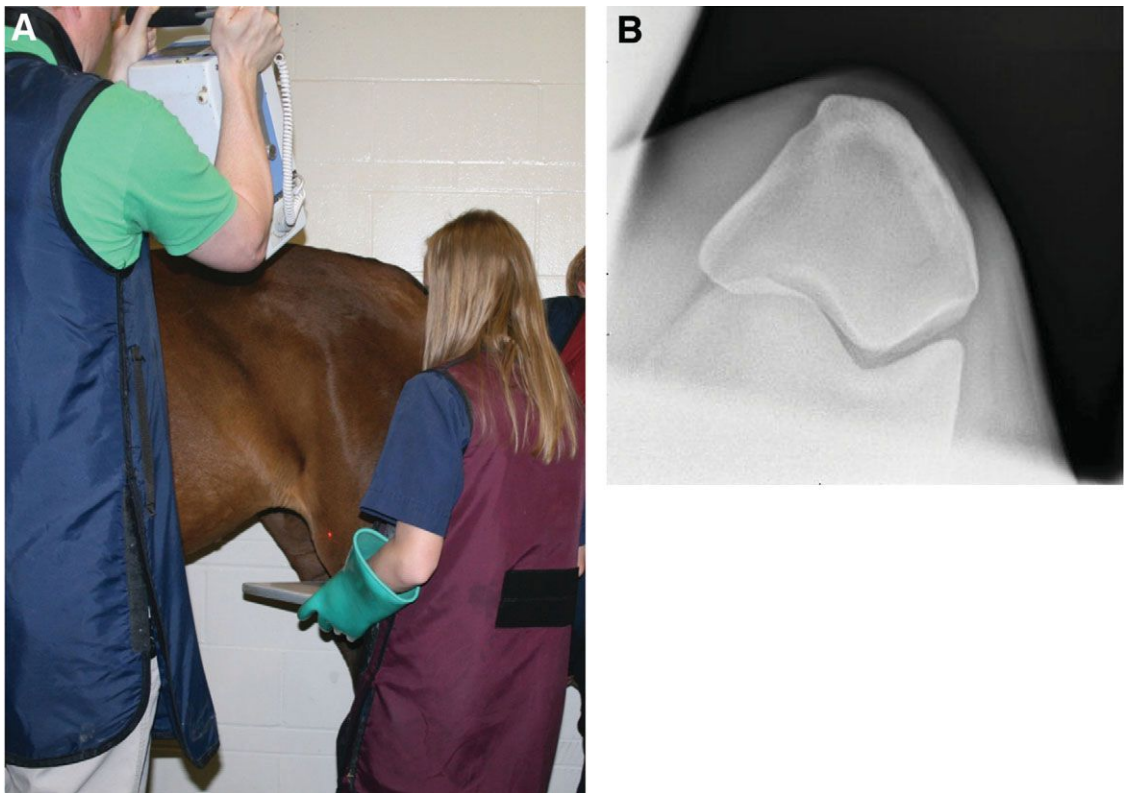


Fig. 14. (A) Positioning for patellar skyline view. (B) Apparently normal patellar skyline radiographic view of a mature Thoroughbred gelding.

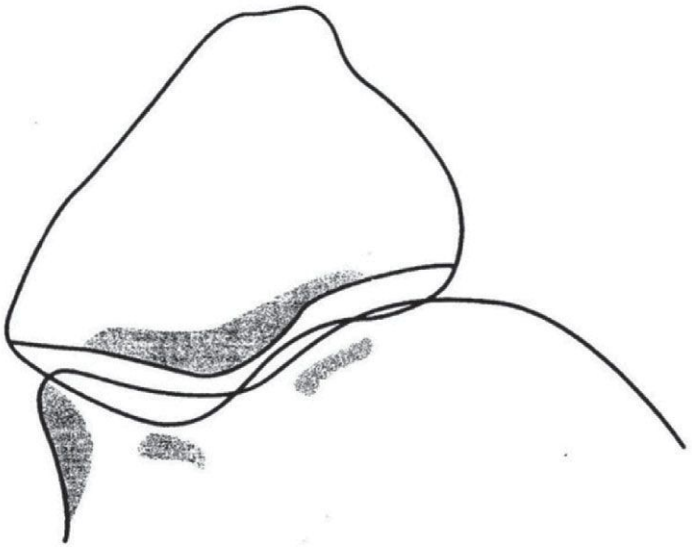


Fig. 15. Patellar skyline (cranioproximal-craniodistal oblique view). Shaded areas are common sites of subchondral lucency with/without sclerosis. Adapted from O'Brien.[3]

separate and better assess the articular surfaces of the patella and the proximal aspects of the trochlear ridges of the femur, and to better as-sess the attachment sites of the cranial ligaments of the menisci and cranial cruciate ligament in the region of the cranial aspect of the intercondylar eminences.

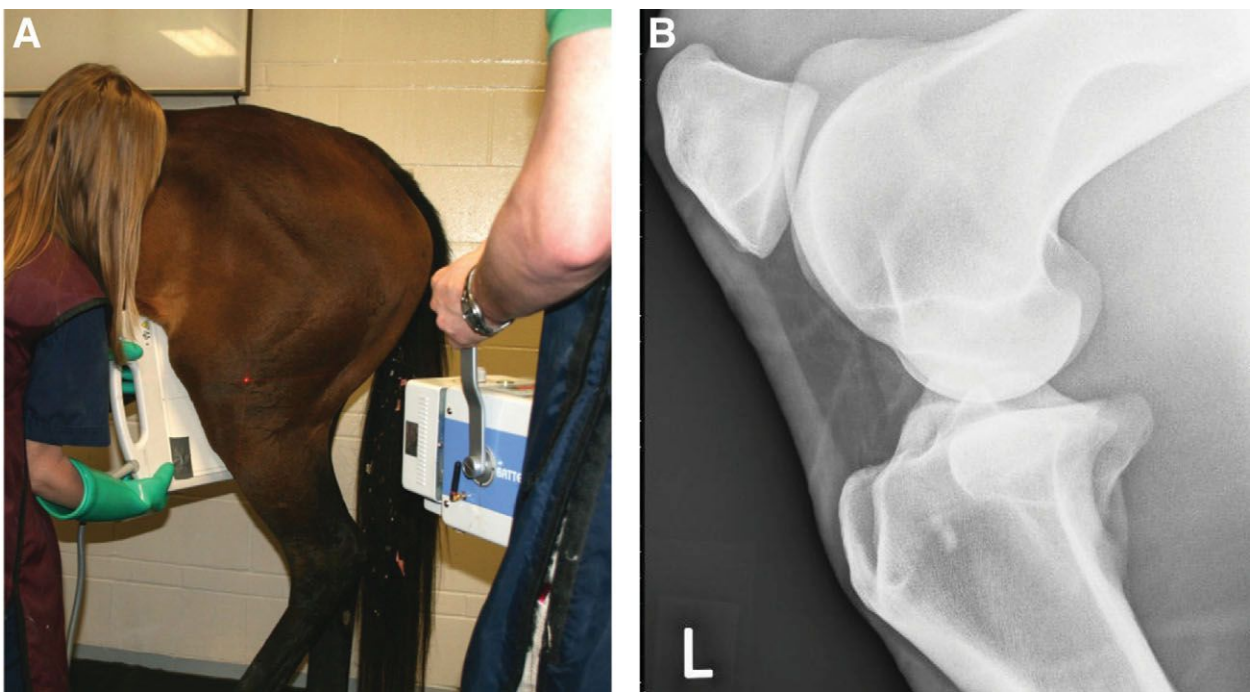


Fig. 16. (A) Positioning for flexed lateromedial view. (B) Apparently normal flexed lateromedial radiographic view of a mature Thoroughbred gelding.

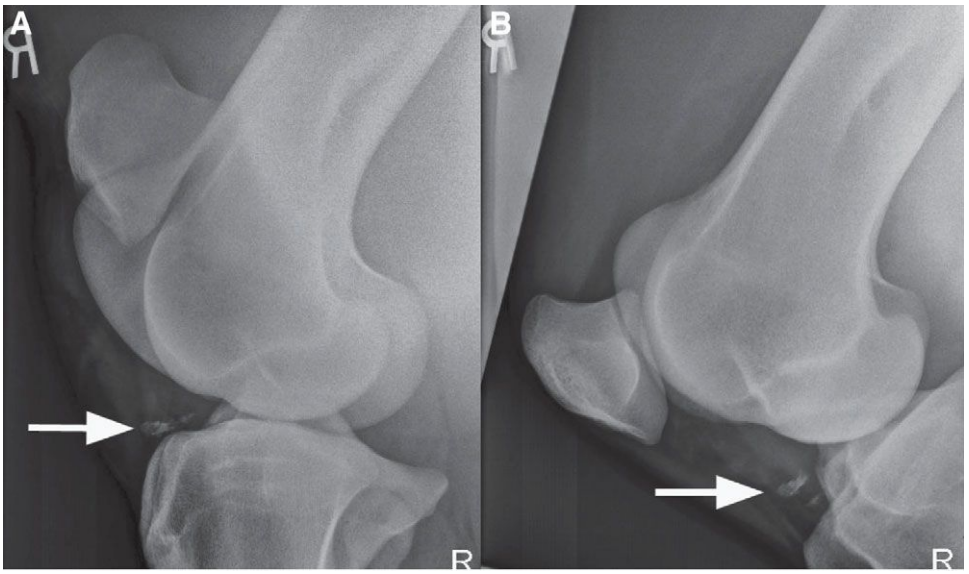


Fig. 17. (A) Lateromedial and (B) flexed lateromedial view of calcification (arrow) of the cranial attachment of the medial meniscus in a 3-year-old Standardbred filly.

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How to Interpret Radiographs of the Stifle Joint of the Young Performance Horse

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1. Introduction

Routine radiographic evaluation of young performance horses has become commonplace in Thoroughbreds (TB) but also occurs in other performance breeds. This practice has resulted in the frequent discovery of radiographic abnormalities (RA) that are often clinically silent but can cause concern for buyers and sellers of young performance stock. The prevalence of radiographic developmental orthopedic abnormalities with or without clinical significance in 6-month-old horses has been reported to be 25% in Warmbloods, 41% in Standardbreds, and 34% in TB.[1] In TB yearlings intended for racing, 86.3% exhibit RA[2]; in young Standardbreds, 42%[3]; in yearling Warmblood horses, 69.5%[4]; and in 1- and 2-year-old Quarter Horses intended for cutting, 89%.[5] Predicting the significance of these RA to later performance can be challenging and frustrating for buyers and sellers, which is further complicated by the possibility of treatment, including surgery, on prognosis.

2. Identifying RA

Because radiographic images are generally reviewed in sets of projections of a joint, this paper will discuss abnormalities by projection. Examiners are of course responsible for reading the entire image; however, there are areas that are more commonly affected, and they should receive the closest scrutiny. RA best detected on a projection are listed in bold but should be confirmed on other projections when possible. Finally, readers should always consider three general factors when reviewing radiographic images:

(1) Make sure that the films are of the correct horse.
(2) Make sure the date is appropriate and consistent.
(3) Make sure all required views are present and of acceptable quality.

Standard radiographic projections of the stifle joint (3 views)

Lateral to medial, 20° caudoproximal-craniodistal (CC20°), and caudolateral to craniomedial 20° oblique

Lateral to medial projection (Fig. 1).

Areas of greatest concern in the lateral projection of the juvenile stifle include the trochlear ridges, the

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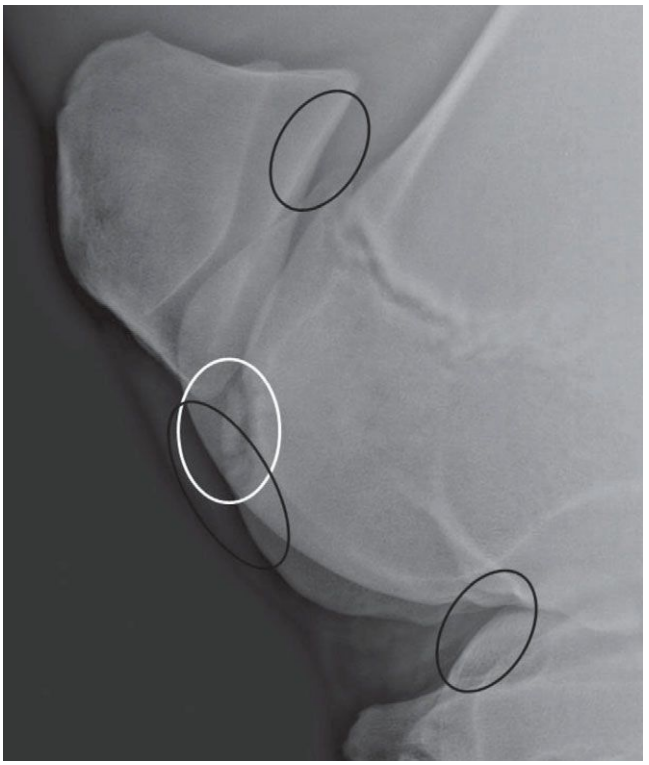


Fig. 1. Lateral to medial projection of the stifle joint. This horse has a lucency with fragments in the proximal LTR (white circle). This radiograph will be discussed by the panel as Case 1.

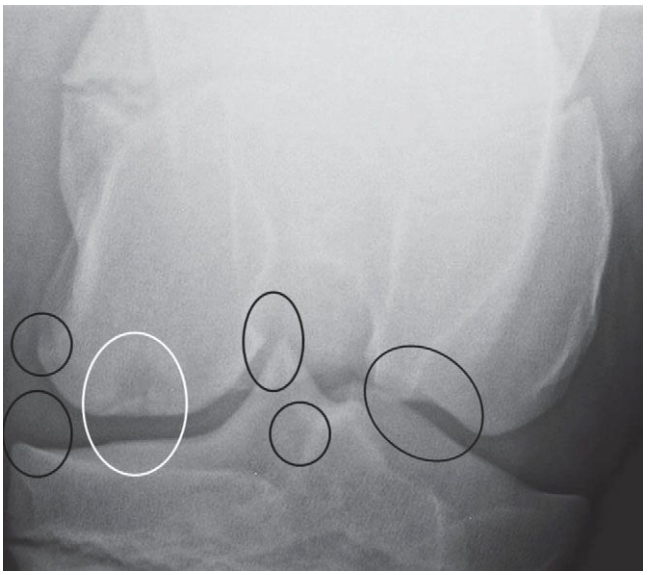


Fig. 2. Caudal to cranial elevated 20° projection of the stifle joint. This horse has an irregular lucency in the MFC (white circle). This radiograph will be discussed by the panel as Case 2.

articular surface of the patella, and the cranial proximal surface of the tibia. The caudal aspect of the stifle joint is often not included on the lateral to medial projection, which is acceptable if it is well projected on other views (Fig. 1).

Abnormalities best assessed on the stifle lateral to medial projection are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Osteochondral fragments or lucencies in the lateral trochlear ridge (LTR)**
(2) **Osteochondral fragments or lucencies in the medial trochlear ridge**
(3) Osteochondral fragments or lucencies in the articular surface of the patella
(4) Osteochondral fragments or lucencies in the proximal tibia

Twenty-Degree Caudoproximal-Craniodistal Projection of the Stifle Joint (Fig. 2)

The areas of greatest concern on the 20° caudoproximal-craniodistal projection of the juvenile stifle are the medial femoral condyle, the proximal medial tibial plateau, the medial intercondylar eminence of the tibia including the attachment of the cranial menisco-tibial ligament, the proximal lateral tibial plateau, and the lateral femoral condyle.

Abnormalities best assessed on the stifle 20° caudoproximal-craniodistal projection are listed below. If the RA is in bold type, it is the best projection to detect that RA.

Case Prognosis Summary

Case 1. Lucency With Fragments in Proximal LTR

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes	owner/ buyer			
Western performance	Yes	owner/ buyer			
English performance	Yes	owner/ buyer			
General purpose	Maybe	owner/ buyer			

Case Prognosis Summary

Case 2. Irregular Lucency in Medial Femoral Condyle

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes		owner	buyer	
Western performance	Yes		owner	buyer	
English performance	Yes		owner	buyer	
General purpose	Maybe		owner/ buyer		

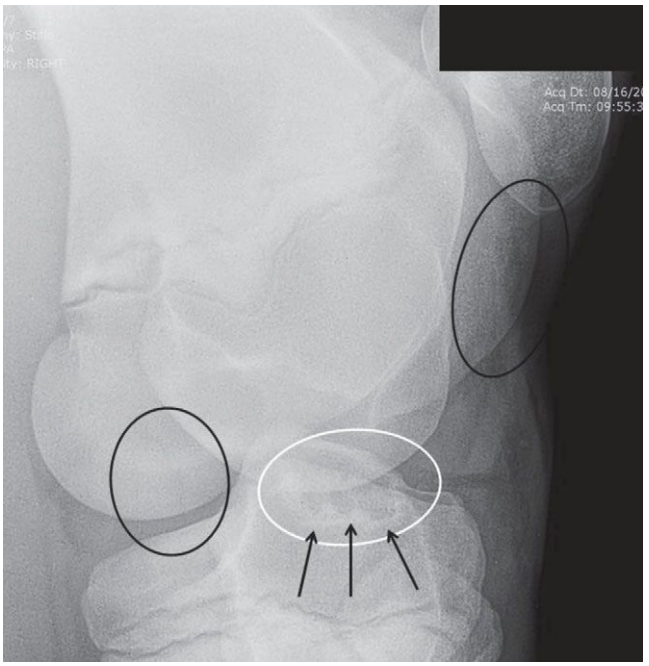


Fig. 3. Caudolateral to craniomedial 20° oblique projection. This horse has a large irregular lucency in the proximal-lateral tibial plateau (white circle and arrows). This radiograph will be discussed by the panel as Case 3.

(1) **Lucencies and sclerosis in the medial femoral condyle (MFC)**
(2) **Osteophytes on the medial intercondylar eminence, proximal medial tibial plateau, or proximal abaxial articular margin of the MFC**
(3) Lucencies and sclerosis in the medial tibial plateau
(4) Lucencies and sclerosis in the lateral tibial plateau
(5) Lucencies and sclerosis in the lateral femoral condyle
(6) Sclerosis at the attachment of cranial menisco-tibial ligament

Case Prognosis Summary

Case 3. Large Irregular Lucency in the Proximal Tibia

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Maybe				buyer/ owner
Western performance	Maybe				buyer/ owner
English performance	Maybe			buyer	owner
General purpose	Maybe			buyer	owner



Case 4. Lucencies of the lateral trochlear ridge and patella.

Caudolateral to Craniomedial 20° Oblique Projection of the Stifle Joint (Fig. 3)

The areas of greatest concern on this view include the medial femoral condyle, the medial tibial plateau, the proximal lateral tibial plateau, and the LTR.

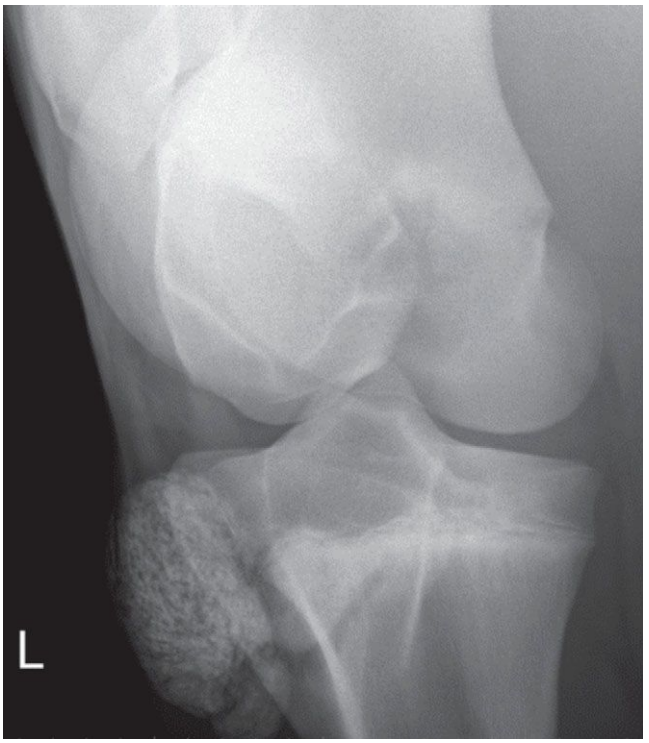
Abnormalities best assessed on the caudolateral to craniomedial 20° oblique projection are listed below. If the RA is in bold type, it is the best projection to detect that RA.

(1) **Lucencies and sclerosis in the medial femoral condyle**
(2) **Osteochondral fragments or lucencies in the LTR**
(3) Lucencies and sclerosis in the medial tibial plateau
(4) Lucencies and sclerosis in the lateral tibial plateau

Case Prognosis Summary

Case 4. Lucencies of the Lateral Trochlear Ridge and Patella

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes			buyer	owner
Western performance	Yes			buyer	owner
English performance	Yes			buyer	owner
General purpose	Maybe			buyer	owner



Case 5. Calcinosis circumscripta.



Case 6. Large LTR lucency with fragments.

Stifle radiographic abnormalities are common in young performance stock and have been reported to have a prevalence as high as 45% in 1- to 2-year-old Quarter Horses at auction.[5] Table 1 summarizes the reported prevalence by breed.[2,4–11]

3. Determining the Significance of an RA to Performance

There are several studies of young stock that have examined associations between stifle RA and performance.[2,12–16] These studies can be challenging to interpret by strict statistical methods ($P < 0.05$) because they have many confounding factors, including varying definitions of RA and incomplete outcome information, usually racing data only. Because of the low numbers of affected horses, subclassifying lesions (size, severity, number of limbs affected, etc) to determine prognosis is usually not possible. However, these studies do have important information to help guide veterinarians examining young performance horses. Some stifle RA in these studies have been associated with poorer racing outcomes ($P \leq 0.05$): debrided MFC subchondral bone cyst (SBC) (versus sibling controls) and large articular communication of MFC SBC after debridement[16] and osteochondrosis (OCD) of LTR and MFC SBC >6 mm in depth.[2] RA in the stifle associated with a tendency ($P < 0.2$) for poorer racing performance include changes in the LTR of the femur and fragmentation of the distal patella.[12]

Case Prognosis Summary

Case 5. Large Calcinosis Circumscripta, Proximal Tibia, Dorso-Lateral

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No	owner/ buyer			
Western performance	No	owner/ buyer			
English performance	No	owner/ buyer			
General purpose	No	owner/ buyer			

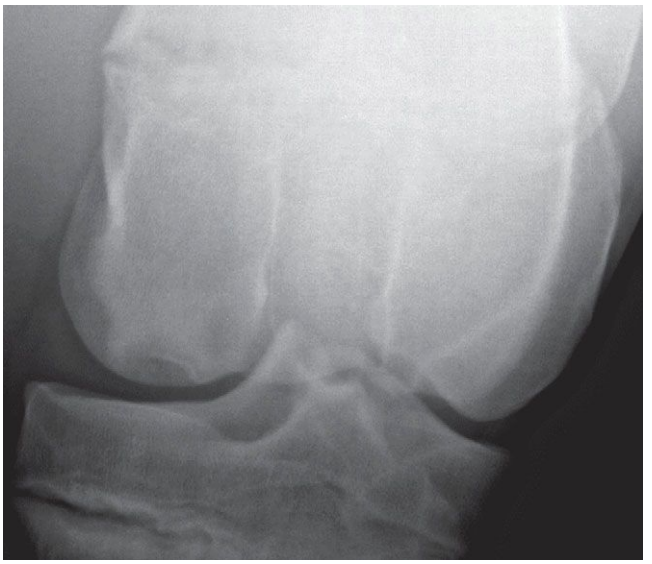
Case Prognosis Summary

Case 6. Large LTR Lucency With Fragments

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Maybe			owner	buyer
Western performance	Maybe			owner	buyer
English performance	Maybe			owner	buyer
General purpose	Maybe			owner	buyer



Case 7. Flattened proximal LTR.



Case 8. Shallow and wide MFC lucency with deeper sclerosis.

Many publications focus on the treatment of a specific stifle lesion, and some of these studies have been able to grade lesions and document an effect of severity or size on prognosis. Clinicians must apply clinical judgment on the basis of their experience with RA in young horses, carefully interpret available studies, and closely evaluate the appearance and clinical presentation of a specific RA.

Table 1. Prevalence of Stifle Radiographic Abnormalities in Young Performance Horses

Stifle Radiographic Abnormality	Breed	Prevalence	References
Lateral trochlear ridge RA	Thoroughbred	36%	6
Lateral trochlear ridge OCD	Thoroughbred	0–4%	2, 7–9
Lateral trochlear ridge OCD	Standardbred	10%	10
Femoropatellar joint RA	Warmblood	39%	4
Lucencies in medial femoral condyle	Thoroughbred	16%	9
RA in medial femoral condyle	Quarter Horse	41%	5
Subchondral bone cyst medial femoral condyle	Thoroughbred	1.7–3.6%	2, 8, 9
OCD any location	Thoroughbred	3.8–8.0%	2, 7–9,11
RA any location	Quarter Horse	43%	5

Case Prognosis Summary

Case 7. Flattened Proximal LTR

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No		owner/ buyer		
Western performance	No		owner/ buyer		
English performance	No		owner/ buyer		
General purpose	No		owner/ buyer		

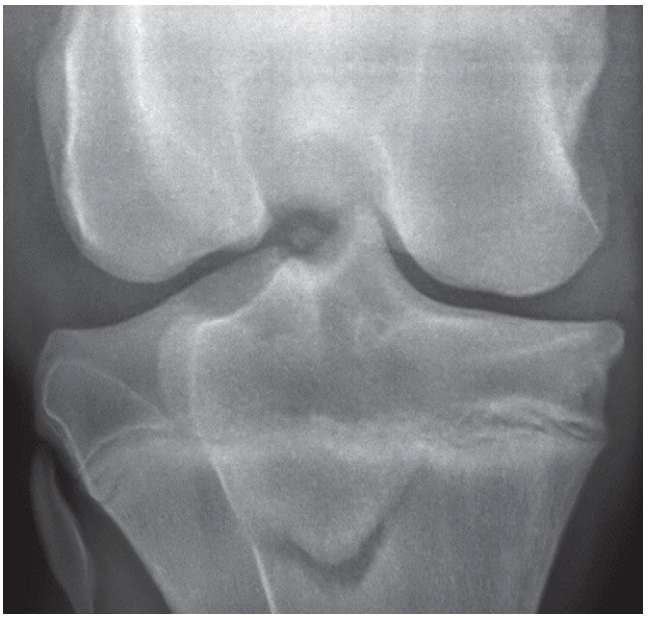
Case Prognosis Summary

Case 8. Shallow and Wide MFC Lucency With Deeper Sclerosis

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes		owner	buyer	
Western performance	Yes		owner	buyer	
English performance	Yes		owner	buyer	
General purpose	No		owner/ buyer		



Case 9. Large MFC subchondral bone cyst.



Case 10. Fragment between intercondylar eminences.

4. Prognosis for Stifle RA After Rest or Surgery

Prognoses include the following:

- Femoropatellar OCD after arthroscopic debridement (multiple breeds): 64% returned to intended use, higher success rate was achieved with lesions <20 mm, higher success rate achieved if operated as 3-year-old versus yearling.[16] Warmbloods had 66% full return, 83% if a return to lesser activity was included; deeper lesions led to a worse prognosis.[17]
- Femoropatellar OCD after debridement of loose pieces and reattaching loose osteochondral flaps with absorbable pins, 95% were successful.[18]
- Subchondral bone cysts of the medial femoral condyle after 6 months' rest, 50% full recovery, with an additional 20% improved.[19]
- Subchondral cystic lesions of medial femoral condyle after debridement: 64% to 74% returned to soundness.[20,21] Articular openings >15 mm had a worse prognosis.[21]
- Subchondral bone cyst injected with corticosteroids: 67% successful. Unilaterally affected horses had higher success (90%) and those without DJD also had a better rate of success (87%).[22]
- Subchondral bone cysts debrided and filled with bone substitute, growth factors, and chondrocytes: 74% were successful.[23]

5. Cases

The summary prognosis charts (cases 1–10) with each image are predicated on the lesion being detected in a yearling intended for one of the four disciplines. The focus is on performance and not resale blemishes. The prognosis given to a horse owner and the prognosis given to a potential buyer, if different, is indicated.

Case Prognosis Summary

Case 9. Large MFC Subchondral Bone Cyst

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	Yes		owner	buyer	
Western performance	Yes		owner	buyer	
English performance	Yes		owner	buyer	
General purpose	Yes		owner	buyer	

Case Prognosis Summary

Case 10. Fragment Between Intercondylar Eminences

Performance Goal	Surgery Yes/No/ Maybe	Excellent Prognosis	Good Prognosis	Guarded Prognosis	Poor Prognosis
Racing	No		owner	buyer	
Western performance	No		owner	buyer	
English performance	No		owner	buyer	
General purpose	No		owner	buyer	

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Fatal Musculoskeletal Injuries of the Metacarpophalangeal and Metatarsophalangeal (Fetlock) Joints in California Racehorses: One Hundred Thirty-Nine Cases

Erin McKerney, DVM*; Elizabeth Collar, DVM; and Susan M. Stover, DVM, PhD, Diplomate ACVS

Fetlock injuries comprise half of fatal musculoskeletal injuries among California racehorses. Most injuries have evidence of pre-existing lesions that likely predispose to catastrophic injury. Knowledge of these lesions will enhance early detection to aid in the prevention of fatal injuries. Authors' address: University of California, Davis, School of Veterinary Medicine, One Shields Avenue, Davis, CA 95616; e-mail: erinmckerney@gmail.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Catastrophic fetlock injuries are common in racehorses. Repetitive loading and fetlock hyperextension associated with training and racing subject fetlock-supporting musculoskeletal structures to degenerative and adaptive changes. These changes can weaken key structures, thus predisposing the fetlock region to catastrophic fracture.

2. Materials and Methods

A retrospective analysis of postmortem examination records was conducted for deceased California racehorses that incurred a fatal fetlock injury during the period of July 2011 to December 2012. Cases were categorized by injury site, fracture configuration, and pre-existing lesions associated with the site of injury.

3. Results

Fatal fetlock injuries comprised 50% of total injuries. Proximal sesamoid bone (PSB) fracture was the most common cause of fetlock injury. Sixty-six percent of fetlock injuries included a grossly visible pre-existing lesion, with lesions observed in 63% of fractured PSBs. The most common lesion was subchondral discoloration and porosity at the abaxial aspect of the medial PSB.

4. Discussion

The high prevalence of California racehorses that are euthanized because of fetlock breakdown injuries makes possible pattern recognition of fracture configuration and pre-existing lesions for the discovery of clinical correlates that can aid in early detection and catastrophic injury prevention.

Acknowledgments

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Research Abstract

NOTES

How to Radiograph and Diagnose Fractures of the Tibia in the Field

Patrick Worden, DVM*; John Peloso, DVM, MS, Diplomate ACVS; and Rodney Legg

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1. Introduction

Displaced and non-displaced fractures and proximal physeal fractures of the tibia are occasionally encountered in practice.[1] Stress fractures of the tibia are one of the most common causes for hind limb lameness in the young Thoroughbred racehorse.[2–5] Although making a diagnosis of a displaced fracture is self-evident, non-displaced and physeal fractures of the tibia often cause non–weight-bearing lameness with a variable degree of swelling and few other clinical signs, making diagnosis a challenge.[2]

Stress fractures of the tibia occur most commonly in 2-year-old or unraced Thoroughbred racehorses.[3–6] They often give minimal to no clinical evidence of their presence with the exception of lameness and a recent history of high-intensity exercise (racing or breezing). Diagnosis of stress fractures of the tibia in its upper portions of the limb is a clinical challenge because the site of pain is rarely amenable to identification through palpation or regional anesthesia. Historically, nuclear scintigraphy has been the imaging modality of choice for the diagnosis of stress fracture of the tibia, given its location on the upper portion of the limb and the high sensitivity of nuclear scintigraphy.[3–6]

Because nuclear scintigraphy is an expensive imaging modality, the initial diagnosis is often not performed, and follow-up scintigraphic examinations are rarely performed. Digital radiography has become commonplace in equine practice over the last 6 years; therefore, collecting excellent quality images of the tibia has become a reality. In one study of 51 cases of tibial stress fracture, 33% (17 cases) were diagnosed through the use of radiographs alone.[3] Making an initial diagnosis of a tibial fracture and return to exercise recommendations can be tailored to patient needs on the basis of the results of serial radiographic examinations and not standardized group recommendations.

Radiography of the tibia is often frustrating and typically inconclusive for several reasons. The technique is infrequently performed; therefore, questions regarding radiographic technique, plate positioning, and inexperience create uncertainty. The concave anatomy of the caudal aspect of the pelvic limb created by the musculature and the proximal tibia makes positioning of the cassette a challenge. Fine-quality, motion-free radiographs are essential if subtle findings such as endosteal sclerosis and periosteal new bone formation are to be identified. Finally, if radiographs are attempted

NOTES



Fig. 1. A caudomedial-craniolateral oblique projection is taken from 45° medial to the dorsoplantar plane. Notice that the short axis of the plate is positioned perpendicular to the long axis of the tibia, the top portion of the tibia is being radiographed, and the radiographer is standing behind the horse with the cassette positioned along the cranial aspect of the tibia. When radiographs are collected, the radiographic machine is positioned from a caudal perspective and the shorter plate perspective is rotated so that it is parallel to the long axis of the tibia. When the plate can be positioned closer to the tibia, the shorter plate-to-object distance allows for a more diagnostic image to be taken with the use of lower exposure settings.

within days after the history of high-speed exercise and lameness in the training Thoroughbred, they are likely to be inconclusive.

The objective of this presentation is to provide the ambulatory veterinarian with a simple technique to collect excellent-quality radiographs of the tibia to facilitate making the initial diagnosis. Another objective is to outline an appropriate schedule for taking follow-up radiographs after diagnosis so that return to performance recommendations can be based on findings of serial radiographic examinations tailored specifically to patient needs and not the customary recommendations of 6 months of convalescence after injury.

2. Materials and Methods

History and Physical Examination

Indications for radiographing the tibia are young Thoroughbred horses (usually less than 2 years of age) that have hind limb lameness on average of grade 3/5 within 4 days of any speed work (recent breezing or racing). Typically, these horses[3] have not raced or are lightly raced before a diagnosis of tibial stress fracture. Many horses have a history of more severe lameness soon after exercise that improves over a 24-hour period with stall rest and non-steroidal anti-inflammatory drug treatment.

Horses rarely respond to firm palpation and percussion of the mid tibia, and lameness is exacerbated by a spavin test in up to 76% of cases evaluated in one study.[3] Another indication for tibial radiographs is lameness that persists after a diagnostic low, four-point nerve block. Nerve blocks performed in more proximal locations on the hind limb are difficult to perform reliably.

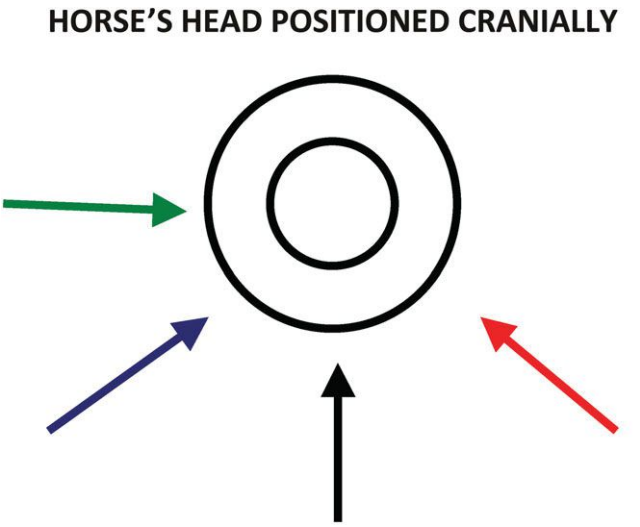


Fig. 2. Drawing taken from a dorsal perspective above the horse. The circle represents the tibia and the arrows represent the caudal location of the radiographer standing behind the horse with the horse's head positioned cranially toward the top of the image. Our four standard projections are Cd45M–CaLO projection (red line), Cd10Pr–CrDiO projection (black line), Cd45L–CrMO (blue line), and an LM (green line). Most radiographic lesions are identified by the projection outlined by the red arrow (Cd45M–CaLO projection).

Radiographing the Tibia

When obtaining radiographs of the tibia, we make three changes to what is conventionally described in the literature: (1) the perpendicular orientation of the cassette; (2) radiographing the tibia in halves; and (3) positioning the radiographer caudally behind the horse. Each of these is explained in detail.

Perpendicular Orientation of Cassette

Most digital radiography plates are rectangular in shape, with a shorter and longer axis. With these rectangular shapes, it is easier to rotate the plate so that the short axis of the plate is oriented up and down the limb. In other words, the long axis of the cassette is perpendicular to the long axis of the limb.



Fig. 3. These six projections are normal radiographs of the tibia. The top row is the medial and latter obliques, respectively; the middle row is the top and bottom of the lateral-medial projection; and the bottom row is the top and bottom of the caudal-cranial projection.

Radiograph the Tibia in Halves

We position the cassette and take radiographs of the tibia from two different perspectives: from the proximal aspect and from the distal aspect. This allows the entire tibia to be radiographed, given its length, and allows for higher technique settings on larger horses that have more soft tissue associated with the proximal aspect of the tibia. When this technique is used , approximately two thirds of the tibia fits on a standard-sized cassette; therefore, images of the middle can be evaluated in the top and bottom projections.

Position of the Radiographer and the Cassette

The radiographic machine and radiographer are positioned behind the horse. Given the concave anatomy of the musculature on the caudal aspect of the thigh, the dorsal position of the cassette allows it to

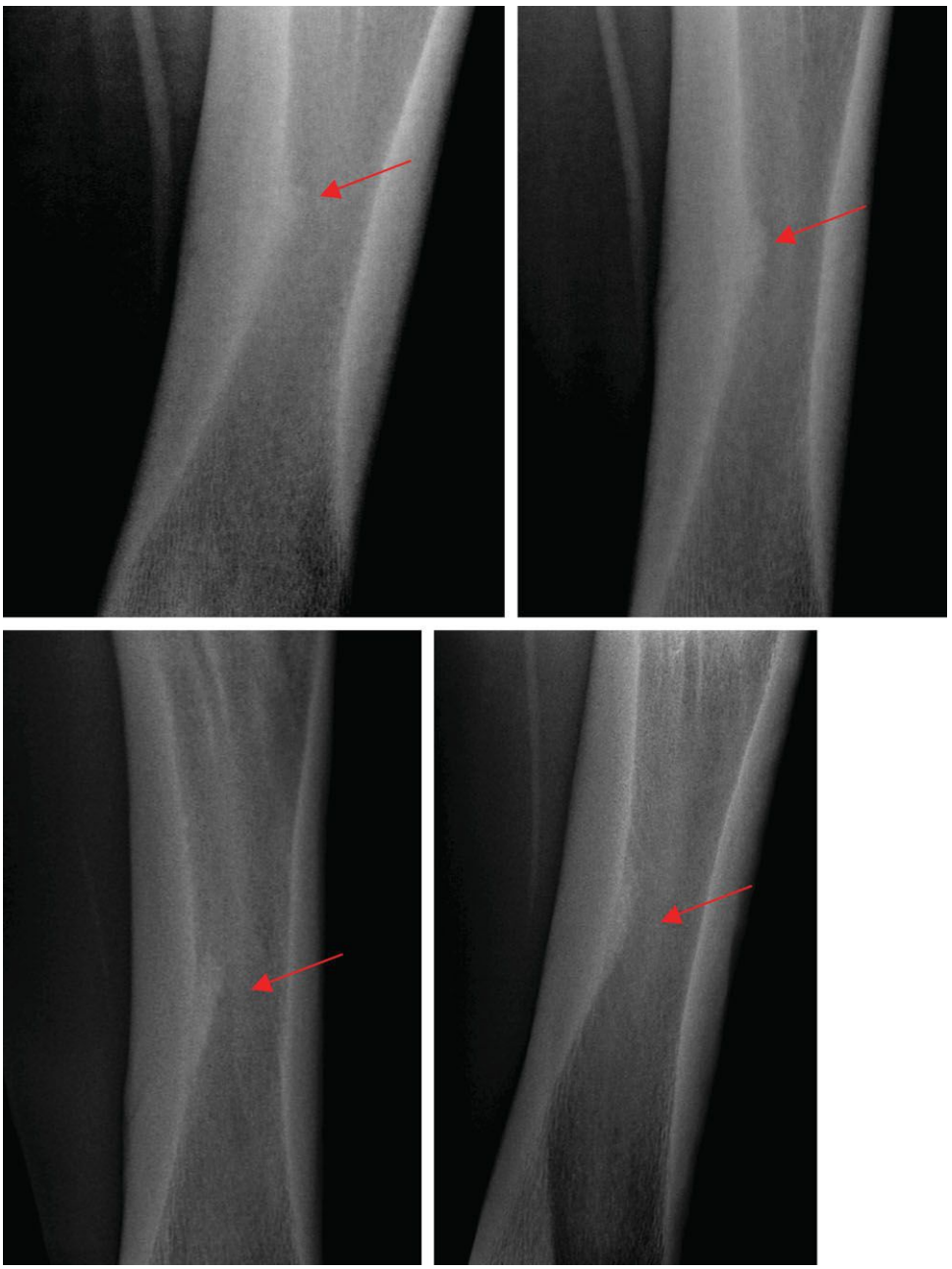


Fig. 4. The four images are examples of the Ca45M–CaLO (red line in Fig. 2) projection. The red arrow identifies the endosteal reaction created from a stress fracture in the racing Thoroughbred.

be positioned closer to the tibia in this location. In addition, this shorter plate-to-object distance allows for a more diagnostic image and lower technique settings (Fig. 1). The insertion of the patellar ligaments on the proximal aspect of the tibia is a useful landmark that allows for more exact positioning of the plate with respect to the tibia during the initial examination and during follow-up examinations.

Standard Projections

The operator stands behind the horse (Figs. 1 and 2) to collect our four standard projections.

(1) A caudomedial-craniolateral oblique projection made at 45° medial to the dorsoplantar plane (Cd45M–CaLO; Fig. 2, red line);

(2) A caudoproximal-craniodistal oblique projection made at 10° proximal to the supporting surface and parallel to the dorsoplantar plane (Cd10Pr–CrDiO; Fig. 2, black line);

(3) A caudolateral-craniomedial oblique projection made at 45° lateral to the dorsoplantar plane (Cd45L–CrMO; Fig. 2, blue line);

(4) A lateromedial (LM) projection (Fig. 2, green line).

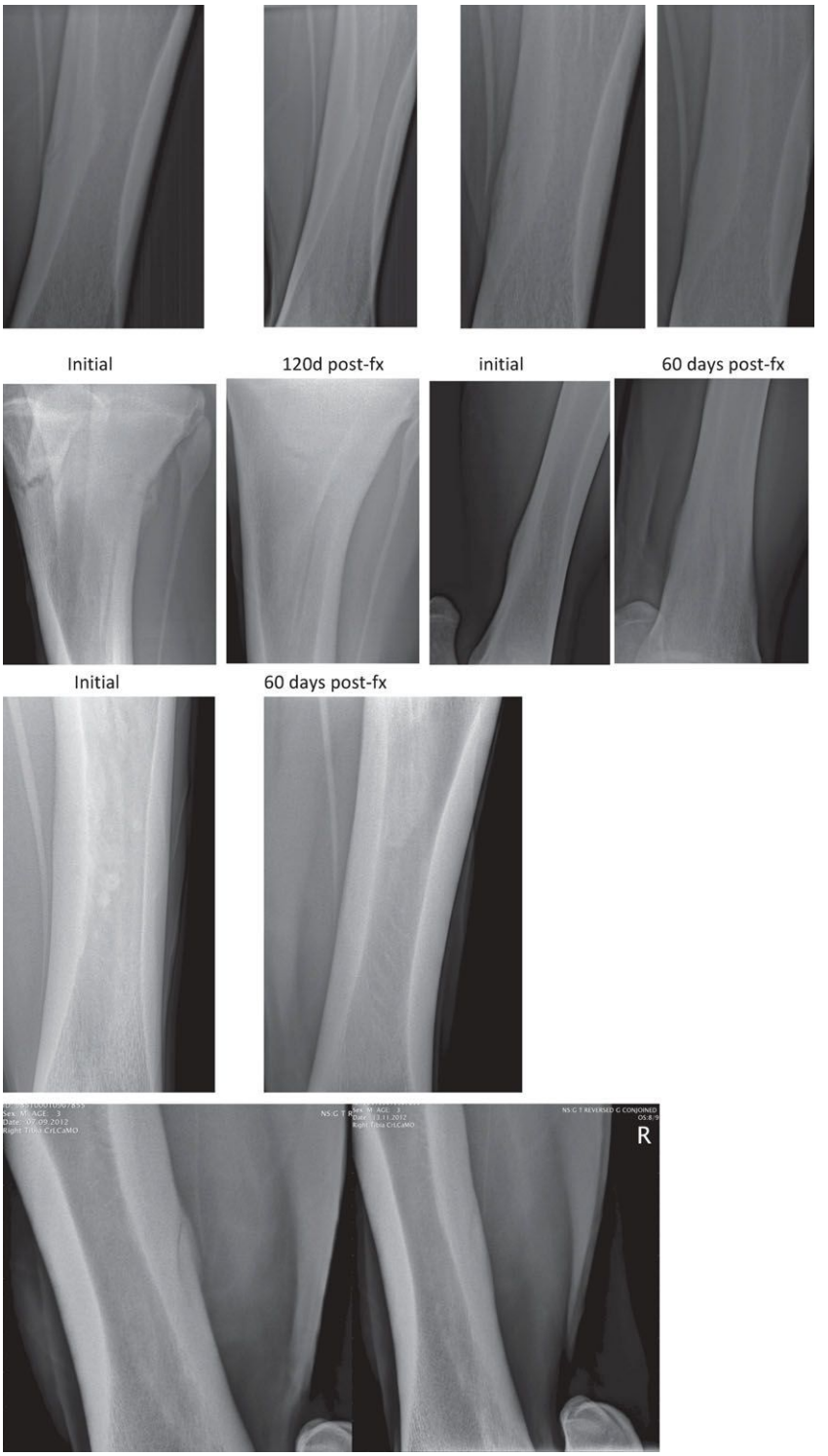


Fig. 5. Example of a horse with a cortical fracture and the appearance of the fracture taken 30 days later.

The four standard projections taken from proximal and distal perspectives typically create a minimum of eight radiographic images. Most of the pathology that we find (especially in the 2-year-old Thoroughbred racehorse) is in the mid diaphysis on the Cd45M–CrLO projection (Fig. 2, red line); therefore, we usually recommend taking a few additional views, concentrating on the center of the tibia. The end result is up to 10 images per tibia. Because the occurrence of bilateral tibial stress fracture can be high,[1,3] it can amount to up to 20 radiographs per patient.

3. Results

With hind limb lameness, the tibia is radiographed on day 1 of lameness if diagnostic blocks do not localize the lameness to the lower limb. Cases that are radiographically negative on day 1 are radiographed again on day 10. The author (PW) has radiographed approximately 100 tibias in 90 horses since January 1, 2008. Most of these radiographs were taken on 2-year-old Thoroughbreds preparing for an in-training sale. It is the presenting author's estimation that when this combination of tibial radiographs is evaluated, approximately 50% of the 90 horses radiographed have evidence of either a fracture line or endosteal reaction consistent with a tibial stress fracture. If significant changes are identified, the radiographs taken earlier in time are often negative. In the presenting author's experience, radiographs taken on day 1 of lameness are helpful in approximately 20% of cases and radiographs taken 10 days later are helpful in the remaining 80% of cases. It is the presenting author's opinion that follow-up radiographs should be repeated for all cases that are negative initially.

Radiographic Diagnosis

Initial radiographic changes include an endosteal reaction that can progress to a periosteal reaction and a cortical fracture line originating from the outer cortex. Typically, bone develops on the endosteum and results in a sclerotic region that forms secondary to fatigue fracture.

Radiographic evidence of endosteal, focal periosteal, or an intracortical fracture line is considered diagnostic of a stress fracture, although it is recognized that some horses will only develop endosteal reaction and never develop the periosteal reaction and/or a cortical fracture line.

Schedule of Initial Radiographs and Follow-Up Radiographs

Stress fractures can be difficult to detect early in the acute stages until the mineral content of the fracture line decreases by 30% of the total mineral content of the bone—a phenomenon called radiographic rarefaction.[1] Thus, a fracture line that was not radiographically apparent on the initial radiographs may be detected on films obtained 5 to 10 days later.

Horses with non-displaced fractures and physeal fractures are radiographed immediately after injury, and the diagnosis is often readily evident. In training, Thoroughbreds with the appropriate history should also be radiographed immediately after injury, although these initial radiographs can often be negative. These same horses should be re-radiographed in 10 days' time and should be on restricted exercise schedules until these radiographs are repeated. Some fractures are never visible radiographically and may be preceded by the development of endosteal sclerosis.

Once the diagnosis of non-displaced or physeal fracture is made, follow-up radiographs should be taken every 30 days, and the results of these examinations should guide future exercise schedules. Non-displaced fractures can take up to 6 months to heal, but light exercise can begin at 3 months in advance of radiographic evidence of fracture healing. Physeal fractures can be returned to exercise in 4 to 6 weeks.

Once a diagnosis of tibial stress fracture is made, radiographs should be taken every 30 days and once before the horse does any high-intensity exercise (racing or breezing). Current recommendations are to stall-rest the horse until it is sound at the jog 5 to 7 days, followed by 60 days of paddock exercise or light galloping.[2–8]

Some suggest that training can be resumed 4 to 6 weeks after diagnosis, although one author comments that training initiated sooner than 16 weeks after injury predisposes the horse to the reoccurrence of stress-related bone injury.[8]

4. Discussion

Tibial stress fractures are one of the most common causes for hind limb lameness in the young Thoroughbred. Until digital radiography, the only way to confirm a tibial stress fracture was with a bone scan. Today, many of the digital radiography units on the market will produce very high-quality images of the tibia. In the presenting author's experience with young Thoroughbred racehorses and 2-year-olds in training, approximately 90% of tibial stress fractures occur in the dorsal lateral aspect of the mid-diaphysis, and this observation is supported in the literature.[3–8]

Historically, radiography of the tibia was not practiced in the field because of the limitations of standard radiography: inexperience with radiographic technique, large cassette sizes, and the number of exposures needed and time delays in determining whether quality images were collected. Digital radiography has made high-quality, motion-free radiographs of the tibia a reality, so that endosteal sclerosis and periosteal new bone formation is significantly easier to identify. In one study, 45 of 58 (76%) tibial stress fractures were identified radiographically,[5] and in another study, 33% of horses with tibial stress fractures were identified on the basis of radiographs alone.[3] Most studies agree that stress fractures and non-displaced fractures of the tibia are significantly more common in proximal and mid-diaphyseal locations.

The three techniques used in this report allow the ambulatory veterinarian with a simple means to collect excellent-quality radiographs of the tibia to make the initial diagnosis. It also offers a schedule for serial follow-up radiographs after diagnosis so that return to performance recommendations can be based on patient needs (also see Figs. 3–6).

5. Conclusions

Hind limb lameness that originates in the tibia is common in the young Thoroughbred racehorse.

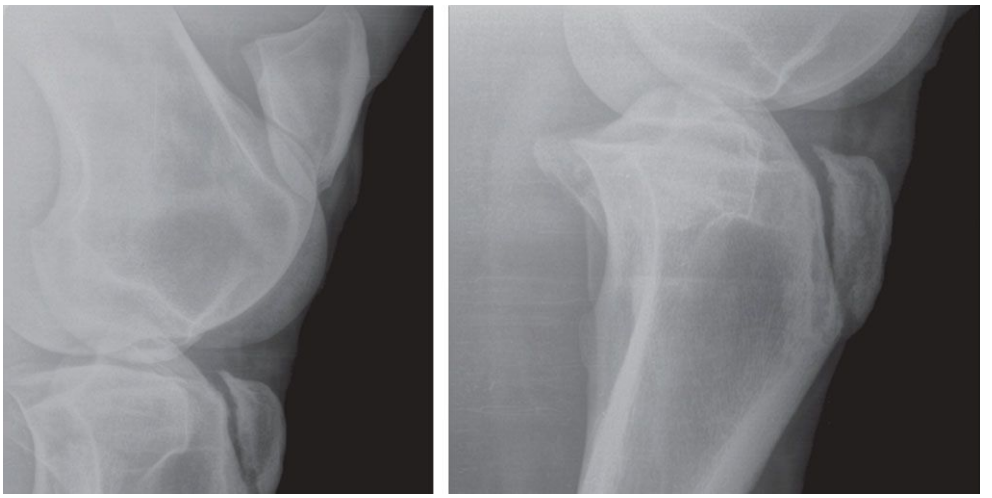


Fig. 6. Example of a tibial crest fracture of the tibia.

Aside from hind limb lameness, the absence of physical examination findings and clinical signs make fractures of the tibia a diagnostic challenge.

Taking quality radiographs is crucial when making an initial diagnosis of non-displaced fractures and stress fractures of the tibia. When obtaining quality radiographs of the tibia, three technical details will improve image quality and reproducibility: (1) turn the cassette so that the long axis is perpendicular to the tibia; (2) radiograph the tibia in proximal and distal halves; and (3) position the radiographer behind the horse and the cassette on the cranial aspect of the tibia.

Although four standard projections are taken, lesions are most often identified in the mid-diaphysis (70%), which suggests that the caudomedial-craniolateral oblique projection (Ca45M–CaLO; Fig. 2, red line) is the most useful projection. Typically, the tibia is radiographed on day 1 of lameness if diagnostic blocks do not localize the lameness to the lower limb. Significant pathology is identified in some of the cases, and a course of action is outlined on the basis of these radiographs. If significant changes are not identified, then radiographs are repeated in 10 days. In the presenting author's opinion, the value of the technique is not to make an early diagnosis but to make a diagnosis in the field without a bone scan, and, if successful, to provide a radiographic lesion that can be followed over time with recovery recommendations that are individualized.

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Effect of Insertional Suspensory Branch Desmopathy on Racing Performance in Juvenile Thoroughbred Racehorses

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Juvenile insertional suspensory branch injury causes decreased racing performance. The severity of suspensory branch injury is important when providing prognostic advice. Authors' address: Florida Equine Veterinary Associates, 10195 N Hwy 27, Ocala, FL 34482; e-mail: infieldems@hotmail.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The objective of this report was to investigate the effects of juvenile insertional suspensory branch injury (JISBI) on future racing performance, to allow clinicians to provide evidence-based prognostic advice to their clients.

2. Materials and Methods

Medical records of horses that presented with JISBI in one suspensory ligament branch were reviewed. Fifty-eight horses met the inclusion criteria. Horses were assigned a severity grade on the basis of an ordinal system. Maternal siblings were used as matched controls in a nested case-control study. Race records were evaluated for their 2- and 3-year-old racing seasons.

3. Results

The prevalence of JISBI was 9.5% for this population. Sixty-six percent of JISBI cases started a race compared with 89% of cohorts ($P < 0.001$). Total average earnings per start (EPS) were decreased ($P < .005$), as were 2-year-old ($P < 0.001$) and 3-year-old EPS ($P < 0.01$) when compared with matched controls. Mild cases performed similarly to controls by their 3-year-old season, whereas severe cases had reduced ability by all measured parameters. Although the wastage was higher in cases with JISBI, individual cases that made it to a race had EPS similar to that in their matched control. Cases with concurrent sesamoiditis had more severe grades of JISBI and decreased racing performance.

Research Abstract

NOTES

Preliminary Investigation of the Treatment of Subchondral Cystic Lesions in the Equine Medial Femoral Condyle With a Transcondylar Bone Screw

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Stifle subchondral cystic lesions can be successfully treated by placement of a transcondylar screw. Authors' addresses: Department of Veterinary Clinical Sciences, The Ohio State University, 601 Vernon L. Tharp Drive, Columbus, OH 43210 (Santschi, Williams, Bertone); Equine Medical Associates, 996 Nandino Blvd., Lexington, KY 40511 (Morgan); Woodford The Equine Hospital, 3550 Lexington Road, Versailles, KY 40383 (Johnson); Manor Equine Hospital, 15801 Old York Road, Monkton, MD 21111 (Juzwiak); e-mail: santschi.5@osu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Subchondral cystic lesions (SCL) in the medial femoral condyle (MFC) of horses can cause lameness. Our objective was to use an MFC transcondylar screw to promote cyst healing and reduce lameness.

2. Materials and Methods

A 4.5-mm lag screw was placed across the MFC SCL in horses with hind limb lameness caused by SCL. Postoperative radiography and lameness examination were performed at 30- to 60-day intervals for 120 days, and SCL healing and lameness were graded. Treatment was considered successful if lameness was eliminated and ≥50% bone healing occurred in the SCL by 120 days after surgery.

3. Results

Twenty horses had 27 limbs treated. Nine horses had adjunctive biologic agents placed into the cyst. In all horses, lameness was reduced by one to two grades by 60 days after surgery, and in 15 horses it was abolished by 60 to 120 days. At 120 days, the mean amount SCL healing was 70%. Of the 15 successes, all are in work (mean follow-up of 12 months) without stifle lameness. Successful treatment occurred in 78% of horses treated with biologic therapies and in 73% without biologic therapies. Three of the five failures had additional injuries to cartilage, meniscus, or tibia, one apparent at surgery and two after surgery.

4. Discussion

Transcondylar screw placement promotes SCL healing and lameness reduction in approximately 75% of all treated stifles. The simplicity of the technique and lack of specialized equipment required should make it an attractive option to equine surgeons.

Research Abstract

NOTES

Response of Bone Necessitated by High-Speed Exercise

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1. Introduction

Bone was once thought of as inert endoskeleton, but accumulated knowledge assuaged that belief. Bone is a very dynamic tissue, highly sophisticated in its adaptability to increase and decrease in the demands of exercise. It will enlarge to withstand additional load and is capable of reducing its mass when loads regress. As such, bone is the one tissue that is capable of regenerating itself and tuning its anatomy to its needs. The neonate is born with a highly adaptable skeletal model. The phenotype is genetically predisposed but refinement to the functional skeleton is dictated by the biomechanical loads that the skeleton experiences.[1] Unlike organ systems such as the lungs, in which training causes no change in organ size, bones can enlarge cortices to double or triple thickness and tune the enlargement to best neutralize the demands placed on it.

In addition to its role as the biomechanical support for the body, the bone serves as the depot for the body's reserves of calcium,[2] which must be controlled within very narrow limits in the circulation because of its role in muscle contractility and muscle tone.[2,3] The medullary cavities of bone are also the reservoir for mesenchymal and hematopoietic cells that supply the precursor cells for tissue healing and circulating blood.

The heavily training horse is required to carry skeletal adaptation to an extreme. Race training literally molds the juvenile skeleton into a racehorse skeleton; a horse is not born with a racehorse skeleton.[4] The skeleton is also tuned for horses of other uses, but the racehorse demands the most of the bone because of the repetitive cyclic load the bone must endure during exercise.

It is advantageous for the horse to carry as little skeleton as possible because skeletal load is deadweight during high-speed exercise. The skeleton's function during racing and training is to carry the weight of the horse and the rider. Redundant bone strength adds extra pounds and demands extra work to carry those pounds during competition, reducing the horse's competitive advantage. Therefore, it is ideal for the racehorse to carry the minimum skeleton necessary for traversing the distance of the race but to carry sufficient skeleton to complete the race without permanent damage.[5] To accomplish this, there is great rationale in work-specific adaptation of the skeleton to meet the demands it experiences, but to maintain a minimum of redundancy.

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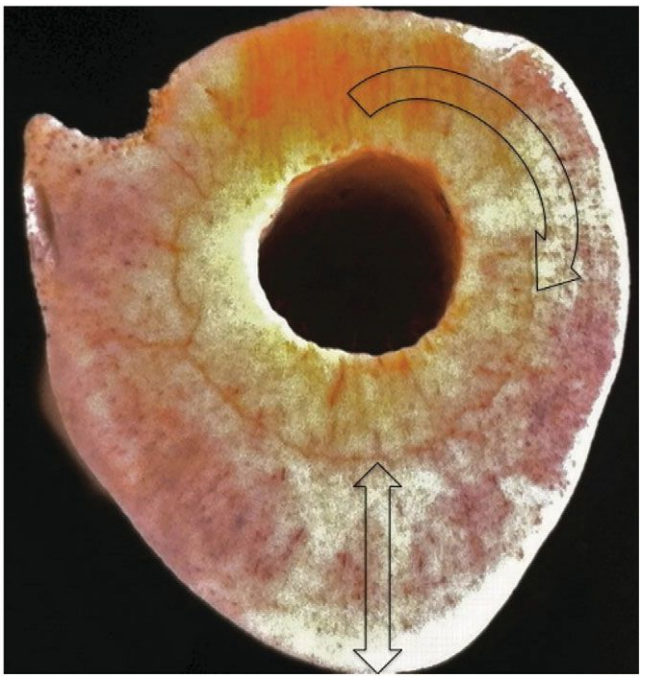


Fig. 1. Transverse cut thorough the right hind metatarsus of a 3-year-old Thoroughbred shows the original yearling shin marked by the curved arrow. The newly formed bone dorsal to the original metatarsus marked by the open arrow has been formed in response to training. Note the clearly demarcated cement line where growth stopped and bone made in response to training was re-initiated. Ideally, training would begin before growth stopped to minimize the demarcation between the bone of two different ages because this leaves an area of differing maturity, density, and stiffness of bone until the cement line is remodeled.

The extreme number of cyclic loads that must be withstood by the racehorse requires localized adaptation between bones and within the same bone to withstand these loads.[5,6] The most extreme examples of this occur in the dorsal cortex of the metacarpus/metatarsus and the posterior cortex of the tibia.[6] These cortical bone sites literally double or triple their cortical thickness on the heavily loaded cortex to enable the bone to withstand the cyclic loads that are produced by high-speed racing and training, but the trans cortex remains virtually unchanged (Fig. 1).

There are basically two cell populations that increase or decrease the size of the skeleton and remodel the skeleton to better withstand exercise: the osteocyte and the osteoclast populations.[7,8] The osteocyte, which is the mature osteoblast encased in the bone matrix, is the permanent cell of bone. "Bone-lining cells" are the osteoblast source cells and are present within the medullary cavity and on the periosteal surface of bone. Growing bones have high populations of bone-lining cells until the end of growth, when the cells are no longer needed. These quiescent cells are able to quickly respond to increasing demand by becoming osteoblasts and initiating bone formation. When not used, these cell populations atrophy.[9] Therefore, it is ideal to initiate training at the end of growth before the cell populations and supporting blood supply atrophy after growth, so they can be converted to the tasks of bone modeling and adaptation of the skeleton to training with as little interruption in the bone formation process as possible. This is desirable because of the economy of converting the blood supply and cell populations from one job to the next without significant "retooling," but it is also advantageous to have a homogeneous bone structure without the distinct interfaces (cement lines) of markedly different ages of bone that are created when bone formation stops for a time, then is reinitiated due to changing stimuli (Fig. 1). Definitive "cement lines" are sources of stress concentration until the normal remodeling process eliminates them and homogenizes the bone cortex.[10]

Bone-lining cells become osteoblasts under the biomechanical stimulus that exercise creates. Osteoblasts do not make bone directly; they make the proteinaceous precursor of bone called osteoid, which contains the collagen matrix component of bone. The osteoblast then excretes the mineral component, which forms the crystalline mineral hydroxyapatite, $Ca_{10}(PO_4)_6(OH)_2$, within the osteoid matrix to form bone. The osteoblast eventually surrounds itself and becomes entrapped by the bone it produces, becoming an osteocyte.[11] Though an osteocyte is trapped within the bone, it is far from isolated from the other osteocytes and the external environment because osteocytes maintain communication with other cells and the supporting blood supply by means of multiple cytoplasmic dendrites that lie within the numerous bone canaliculi.[9] The dendrites interconnect with other osteocytes, with the vessels of the Haversian canal circulatory system of bone, and the periosteal surface of the bone.

Osteocytes are capable of adding to or subtracting from bone mass by adding bone or reabsorbing bone from the walls of their lacunae and canalicular spaces.[3] Therefore, they can increase or decrease the density of the bone per unit of volume of bone tissue. Bone formation and dissolution must go on at low levels continuously to make calcium available to the body as needed to maintain the circulating calcium levels, should the ingested calcium be insufficient to supply these needs. It can be accelerated or decelerated as needed with physiologic demands. Calcium ingested in the diet and calcium excreted in the urine are the macro-sources of calcium acquisition and excretion, but the stability of the narrow physiologic range of serum calcium requires the stability of the bone's supply of calcium to fine tune systemic calcium levels. The ions in flux during the process of increasing or decreasing bone density as bone is added or subtracted from the intra-osseous canalicular spaces produce the body's readily available calcium ions.[9]

Adding hydroxyapatite to the surfaces of the canaliculi and lacunae increases the density of bone per unit volume. This process can adjust the mass of mineralized bone per given volume, altering the strength of the bone. Bone generally increases in density with time. This is done principally by controlling the pH of the space between the cellular cytoplasm and the bone surface and the flux of hydroxyapatite into or out of the bone.[3,9] Inflammation in an area of bone can demineralize bone by the same effect of decreasing the pH in the area, causing the loss of hydroxyapatite crystals, and therefore causing the loss of bone mass per unit volume. This has implications for the understanding and treatment of inflammation in bone. The lifespan of an uninjured osteocyte is estimated at 25 years in the human[11]; we assume that this is similar in the horse and that the undisturbed osteocyte may outlive the horse itself.

The destructive arm of the bone remodeling system is the osteoclast. This is a multinucleated giant cell whose sole responsibility is bone reabsorption.[12] Osteoclasts are manufactured on demand by the fusion of monocytes into a multinucleated giant cell with the capability of attaching itself to a bone surface and rapidly removing bone by localized acid digestion and dissemination of the resultant byproducts into the circulation and local environment. Osteoclasts work by attaching themselves to and sealing off an area of bone and demineralizing it with acid and then digesting the remaining collagen. They can do this very rapidly should the demand arise. The demand is created by physical damage, marked need for Ca^{2+} ion, or the need for bone remodeling. Osteoclasts and osteoblasts normally work in concert under the influence of the osteocytes to add or remove bone as the biomechanical load indicates the need. Osteocytes direct bone remodeling in response to loading, or the lack of loading, of the bone. The net result will be bone formation or bone elimination as dictated by the biomechanical input to the osteocyte, which mediates the response on behalf of the body as a whole. Osteoclasts are totally silenced in times of high mechanical demand to preserve bone for the short term.[12]

Bone senses load by determining tension and compression. This is done to some degree by the deformation of the hydroxyapatite crystals, which create positive and negative charges on the surface of the crystals. This is a physical property of all crystals and was thought to stimulate bone formation. More recent consensus indicates that the cytoplasmic extensions of the osteocytes are sensitive to stretch and relaxation and the osteocyte dictates the addition of bone to neutralize the deformation.[2,9,11] Compression deformation always dictates bone formation, whereas tension tends to create remodeling or sometimes bone loss. The compressed surface of the bone remodels the bone internally by adding to the bone density and models the external surface of the bone by adding bone mass to the most heavily loaded areas.[13] This shapes the bone to best resist the stress of training. The dorsal surface of the cannon bones and the caudal surface of the tibia are very active examples of this bone modeling to neutralize ongoing bone deformation.[4,6] Over time, the modeling/remodeling process alters the bone to the shape and strength that best prevents deformation on loading. At this point, the load is balanced on the long axis of the bone unless the workload changes. The modeling process then slows or ceases as long as the exercise load is not altered, but the remodeling must continually occur to repair "wear and tear."[10,14] Biologic systems never become "elastic" to the point that deformation does no damage. Biologic systems are always "plastic," meaning that all loads do some degree of damage, which must be healed on an ongoing basis.[14]

If damage is occurring too rapidly for the repair capabilities then the damage begins to accumulate, forming micro-fractures, then stress fractures, and eventually gross fractures and structural damage to the bone if unimpeded.[10] Damage also accumulates if the modeling process to neutralize the loads of increasing training is overwhelmed by the damage of progressive exercise in the naive skeleton. The horse is particularly vulnerable to this damage because the heart and lungs of the horse are so capable that they tend to train faster than the skeleton. This makes the skeleton the "rate limiting system" in almost all training horses.[15,16]

Osteoblasts and osteoclasts cooperate in the principal remodeling mechanism of bone called the bone-remodeling unit (BRU), also sometimes called the bone metabolic unit (BMU). The BRU is a phalanx of osteoclasts, which cut a well-defined tunnel through and across existing bone, removing old bone in need of remodeling or strengthening. The osteoblasts then follow the osteoclasts in close concert, forming new bone (osteoid, which is then ossified to bone), creating new osteocytes during the bone remodeling, rejuvenating the bone into a newer form of itself. The BRU can only operate within mineralized tissue and cannot invade other tissue forms such as fibrous tissue, cross-gaps, or cross-unstable fracture planes. Gaps must be filled with mineralized tissue first, and the BRU can then remodel and refine the tissue to bone. The BRU is the principal tool the bone uses to heal fractures in the stable fracture healing situation.

The number of BRU functional at any point in time is dictated by the local osteocytes, which sense the biomechanical conditions and local needs with their canaliculi and dictate when remodeling is needed.[2,11] "Exercise debt" in the form of microdamage is built up during exercise and then repaired between periods of damage. Injury and death of an osteocyte is the strongest stimuli for BRU activation and the remodeling that will replace the bone that has lost its resident osteocytes with

new bone containing living osteocytes. This raises the question of whether the mechanism of stimulating remodeling is actually the loss of the inhibition of the BRU that a living osteocyte may produce.

Training is the next most powerful stimulus for bone modeling and remodeling. Exercise trumps all hormonal and metabolic stimuli for bone remodeling, and its effect on bone lasts for years.[6,17] The micro-damage, stimulated by exercise, causes overcompensation by the bone through the modeling and remodeling process, strengthening the bone mass and changing its shape to prevent the micro-damage from occurring in subsequent exercise sessions. If the exercise is then increased, the bone is overloaded again and over-repairs again, further strengthening and modeling the bone to neutralize the load. This stepwise stimulus of hypertrophy by "overload" then "over-repair" is the basis for training in all tissues.

The bone's ability to respond to training is, however, not an unlimited resource. There is a maximum rate of response that the bone can generate. It takes time for the osteocytes to increase the density of the existing bone and for the osteoblasts to change the size of the bone. The "art" of training is to use the hypertrophy response to produce a continually stronger athlete, without progressing too rapidly for the bone to adapt, causing injury.

The question is often posed "Why is the horse so prone to injury and lameness?" The answer is not so much that the bone is weak but that the other systems are so strong. The heart and circulatory system are so highly developed that they can adapt rapidly to nearly any level of sequentially increasing exercise that they encounter. Bone is more limited.[18]

Overload in excess of repair results in structural damage, injury, and eventually, pain.[10] Pain is demonstrated as lameness. Lameness is the shifting of weight from one limb to another to avoid loading of the painful limb. This makes the horse asymmetric and then often precipitates a secondary additional lameness. Few horses performing at high levels are compromised by a single minor lameness. It is often the second or the third painful area that creates the decline in form. A horse will protect and demonstrate lameness on the most painful limb until that pain is relieved by treatment or diagnostic anesthesia. The horse will then protect the next most painful site. Progressive elimination of one lameness at a time until the horse is sound is the best way to determine all of the sites of pain. It is often not the most painful lameness that is the original and therefore most important lameness site. Treatment of the secondary lameness alone is prone to failure because the inciting lameness is still present and the secondary lameness will re-occur. The key or underlying lameness must be identified and treated as well if the horse is to regain soundness. This is the "art" of lameness treatment that must be combined with the "science" of understanding the mechanism of pain in bone and how the primary problem can be treated without endangering the horse or its performance capabilities over the long term. Lameness is a sign of a problem, not the problem itself. This concept is difficult for some owners and trainers to grasp.

Pain in bone and the resultant lameness is a sign of damage and results from instability, hypertension, or invasion of sensitive structures such as the periosteum, ligaments, or joints by the structural damage. Pain results in lameness, therefore lameness must be properly regarded as a sign of a problem, not the problem itself, to correctly interpret and treat it. Veterinarians should be well equipped to help the client with the understanding and interpreting of this normal response to training.

Trainers often regard lameness as "the problem" rather than a sign of a problem. This understanding is difficult for some and should be the purview of the veterinarian to understand the cause and treat the cause rather than only the symptom of the problem. Treatment of the symptom rather than the cause is often only temporarily effective, often damaging in the long term, and can be dangerous if pain indicating progressive structural damage is eliminated without regard to its cause.

What causes lameness in the racehorse? The most common causes are cyclic loading, load accumulation, plastic deformation, and structural damage to the bone.[10,19] Joint inflammation and overload of tendons and ligaments is frequently a secondary sequela.[20]

Bone is very good at adapting to gradual changes in loading levels or loading types, but this adaptation is work-specific and dictated by the biomechanical load the bone receives. Julius Wolff described this a century ago, and the adaptation of bone to specific needs has come to be known as "Wolff's Law," which states "bone is laid down where strength is needed and removed where strength is unnecessary."[13] Few animals carry this to the extreme of the training horse. The overload/over-repair cycle literally molds the racehorse skeleton (Fig. 1).

The bone can take two actions when faced with the increased need for strength dictated by exercise. It can change the material make-up of the existing bone volume, or it can change the size and shape of the bone to better resist the loads. Changing the material make-up occurs by adding bone mass per unit bone; filling in trabecular, canalicular, and lacunar space with more osteoid and hydroxyapatite to increase density increases the amount of osseous mass in the same volume of bone. Bone can also change its shape to neutralize the applied stress by adding bone as occurs in the dorsal cortex of the metacarpus/metatarsus and the caudal tibia.[18] With the use of the same amount of material, changing the shape of a material from a rod to the cylinder doubles the strength of the structure in resistance to

bending; therefore bone enlarges its cylindrical shape whenever it has the opportunity.[14]

The adaptation of bone to training is different than most tissues because it trains to the level of work rather than the volume.[17] Bone requires only a limited number of loads in a specific exercise period to stimulate its response. Experimentally, this has been determined to be approximately 36 similar cyclic loads.[17] Two thousand similar loads were no better than the first 36 loads at producing hypertrophy of the bone. In fact, in an exercise period of more than 36 similar cycles, the excess cycles become destructive, adding excess damage that can then begin to accumulate overwhelming the bone's ability to repair the damage.[10,17] The end result of this can be destruction of the bone rather than hypertrophy and eventually injury in the form of a stress fracture.[10] Soft tissues such as the heart and muscles require a volume of work to train. The "interval training" of these tissues to increase the volume of work at a higher level revolutionized the training of people, but, when tried in the Thoroughbred racehorse, the skeleton could not withstand the volume of work. Endurance horses and Standardbred racehorses are able to somewhat more successfully apply the interval training principles, but it results in too much skeletal damage in the Thoroughbred to be successfully applied.[4]

Because damage and response to training is work-specific, the damage to a racehorse's bone can be mitigated somewhat by varying the gait and the training surface. Mixing more trotting and the use of different training surfaces varies the load the bone encounters and therefore varies the type of damage done by the training. This allows the volume of work to be increased without as much cumulative damage to the bone. Monotonous large volumes of similar training, such as lots of repetitive galloping over the same surface, becomes damaging to bone rather than stimulating of bone adaptation.[21]

As the level of exercise increases, the tolerable volume decreases. Epidemiologic data shows "a horse that had accumulated a total of 35 furlongs of race and timed-work distance in 2 months, compared with a horse with 25 furlongs accumulated, had an estimated 3.9-fold increase in risk for racing-related fatal skeletal injury (95% confidence interval = 2.1, 7.1)."[19] Mindlessly ignoring the ability of the horse's bone to respond to the amount of exercise it is performing will eventually result in damage accumulation and injury.[10,22]

The diaphysis of the metacarpus provides a dramatic example of the adaptation to exercise in a more macro-example of the adaptation that is occurring in the entire skeleton and how it can be disrupted if the adaptation does not occur fast enough or completely enough to maintain soundness.[10,21] Exercise produces new bone at sites of compression but not at sites of tension strain during exercise. The best evidence indicates that this is an active "biologic" process mediated by the osteocytes in the loaded bone that monitor the loading by means of their large dendritic networks within the canaliculi of the cortical bone.[3,9,11] Compression loading results in new bone production that models the bone to best resist the compression (Fig. 1). Bone is produced in the areas of the most compression.[4] This new bone then reduces the compression differential until the load is balanced on the long axis of the bone and the bone receives neutral forces during loading. Overload of compressed bone results in the classic stress fracture configuration that occurs in all materials.[10] The shear stress of compression results in an oblique fracture at 45 ° to the long axis of the bone (Fig. 2). It propagates into the bone until a lamellar plane that is of different stiffness is encountered, and the fracture then propagates along that plane in the long axis of the bone. If no lamellar plane is encountered, the stress fracture may propagate through the cortex into the medullary cavity (Fig. 3). In the young training horse, radiographically evident multiple stress fractures can sometimes be identified in horses that have pain and lameness originating from the metacarpus (Fig. 4). When training is proceeding faster than the bone can ossify and strengthen, the newly formed bone is



Fig. 2. Metacarpal (left) and tibial (right) stress fractures show the typical 45° configuration of a stress fracture.



Fig. 3. Stress fracture propagated from the dorsal cortex into the medullary cavity. The horse was removed from training and stall-rested. The periosteal callus covers the exit of the fracture on the dorsal cortex, but the deep remodeling of the fracture is incomplete. Surgical treatment is often elected because of the prolonged remodeling time necessary for stress fracture healing.

overloaded before it can become strong enough to neutralize the damage (Fig. 4). Stress fractures are part of the modeling process, though they do not become radiographically apparent in most horses. Multiple stress fractures are easier for the horse to overcome than is a single fracture that begins to predominate. In the presence of multiple fractures, the stress of loading is divided among many fractures. If one fracture begins to predominate, it becomes a single stress concentrator, the stress is focused at this one site, and the rate of damage increases.[10] When one fracture begins to predominate, it is difficult for the horse to remodel the bone fully enough to eliminate the stress concentration; therefore, surgery to accelerate the remodeling and promote healing is beneficial.[23]

Because the stiffness of the older bone of the yearling metacarpus is greater than the newly formed



Fig. 4. Radiograph of the metacarpus shows multiple stress fractures developing in the newly formed bone because the training is proceeding faster than the newly formed bone can be formed and strengthened in response to training.

bone (because the osteocytes have fully mineralized the bone), it is more susceptible to stress fractures than is the newly formed bone that is less stiff[14,18] (Fig. 5). The stiffer the material, the less the elasticity, and the sooner the plastic limit where cumulative damage is done is reached. This biomechanical loading will often fracture the older, stiffer bone first. If bone of multiple ages is present within the same bone, the bone that is the most vulnerable will fracture. This is the bone that is the weakest combination of stiffness and strength (Fig. 6). As long as the fracture does not penetrate the surface of the newly formed shin, there is no pinpoint tenderness on the surface of the shin, though the horse is lame and the surface of the metacarpus is often diffusely tender to deep palpation. The newly formed bone re-enforces the metacarpus until exercise no longer causes deformation and stress fracture, but if exercise continues at a level higher than the rate of repair, it overwhelms the new bone formation and the new bone may be overloaded by the rate of damage caused by the exercise. As a stress fracture deep in the bone progresses and becomes more plastic, it may fracture through the newly formed bone to the surface of the metacarpus and cause progressively increasing lameness, focal tenderness on the surface of the metacarpus, and eventually, an unstable fracture if exercise is not slowed (Fig. 5). When the periosteal surface is reached by the stress fracture, pinpoint tenderness along with increased lameness is seen because the highly innervated periosteum is violated. If the newly formed bone is sufficient to stabilize the stress fracture the metacarpus is then strengthened and the pain subsides.

If the process has not yet developed a dominant fracture, modifying training to reduce the stress can aid the bone in neutralizing the overloading force by allowing it to get ahead of the damage caused by exercise.[21] The most damaging force to bone is monotonous, repetitive training: after a finite number of cycles necessary to stimulate strengthening of the bone, the remaining cycles become trauma, contributing to the stress fracture creation.[17] Modification of training reduces the trauma.

On occasion, acute severe lameness will occur after exercise in the absence of palpable tenderness on the surface of the bone. When lameness is localized to the metacarpus and radiographs are taken, the interface of the yearling shin and the bone formed since training was initiated is visible on the radiographs (Figs. 7 and 8). This appears to be a separation of the bone at the cement line interface of the two densities of bone caused by a differential stiffness, as can be seen on anatomic specimens (Fig. 1). This process can occasionally also be seen in the tibia, but it is harder to define radiographically because of the much larger soft tissue coverage of the tibia (Fig. 8). If the horse is given a break from training and the radiographically visible interface disappears, the lameness subsides as well.



Fig. 5. Radiograph of a dorsal metacarpal stress fracture was initiated in the yearling shin because it was stiffer than the newly formed shin and more susceptible to cyclic stress. Open arrow marks the demarcation between the yearling shin and the bone formed in response to training. Once the fracture became unstable, the newly formed bone fatigued and broke as well. As the fracture penetrated the dorsal cortex and the periosteum, the lameness increased.

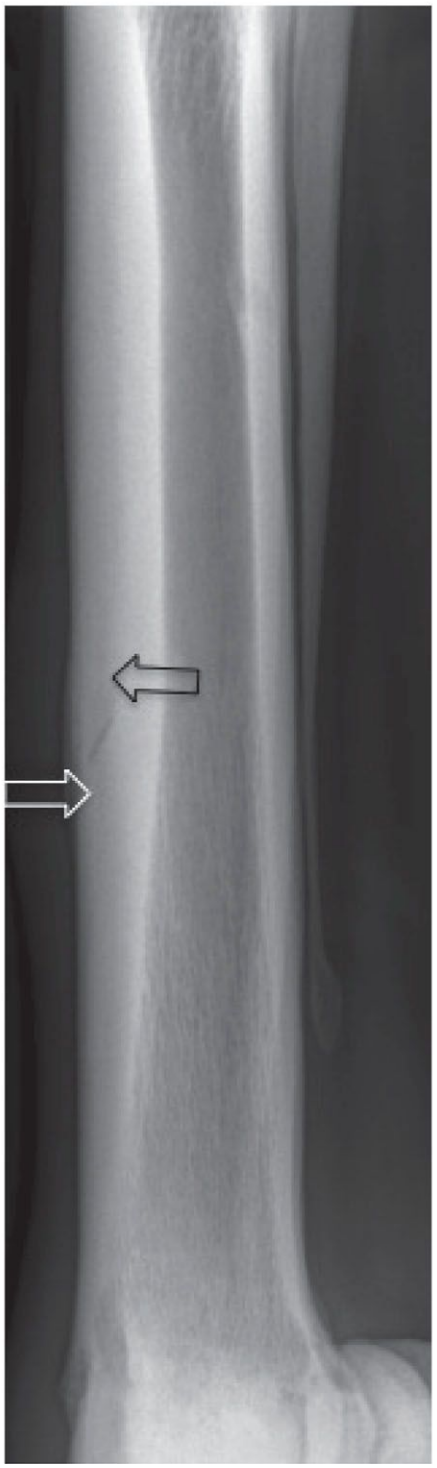


Fig. 6. Radiograph of the dorsal metacarpus shows a stress fracture in the middle of three layers of bone. The horse had a period of rest, and the lameness re-occurred during the second training episode. Black arrow marks the demarcation between the yearling shin and the bone formed after a period of training; white arrow marks the new bone formed after the period of rest. It is likely that the stress fracture in the middle layer of bone was incompletely healed during the period of rest.



Fig. 7. Left figure (A), with the small arrows, shows a milder case of separation of the yearling shin and the newly formed bone that was formed in response to training. Right figure (B), with the large arrows, shows a more severe separation of the yearling shin and the newly formed shin with more periosteal response than in the left figure.

There is some individual variation in the ability to respond to training, as with most biologic systems. Some individuals can mount the response at a rate that virtually precluded their bone being overloaded and some individuals cannot respond fast enough for even the most moderate levels of training. The art of understanding this biologic variation is another quality needed to properly dose training.

Whereas the addition of bone to the surface of the bone to add strength and improve resistance to overload is a workable option for long bones, it is not an option for joint surfaces such as the distal cannon bone.[20,24] Because the anatomy must be preserved in the joint, the bone does not have the option to simply hypertrophy the size of the distal metacarpal or metatarsal surface because it would distort the joint surface. Therefore, in its adaptation to training, the only option available for the distal metacar-



Fig. 8. Lateral radiographs of the tibia show separation of the yearling cortex and the new bone formed in training, similar to that in the radiographs of the metacarpal III's (McIII's) in Fig. 7.

pus and metatarsus is to alter the material. The bone must do this by largely infilling the trabeculation in the subchondral surface to produce "cortical-like" bone because of the needed strength.[24,25] This has the unwanted side effect of eliminating the ability of the trabecular subchondral bone to absorb load through minute distortion and energy absorption. The eventual end result is increased bone and cartilage loading between the relatively unforgiving distal metacarpus or metatarsus and the opposing phalanx and sesamoid bone.[26] The transient result is subchondral bruising of the subchondral articular surface with frequent progression to other disease conditions such as condylar fracture, palmar articular degeneration, performance limiting lameness, or alteration of the shape of the articular surface and damage to the articular cartilage, predisposing to degenerative arthritis, or fracture of the opposing sesamoid bone.[27] This is especially true if the performance-limiting pain is mitigated to some degree with intra-articular medication and training is carried on despite the lameness.

This injury to the bottom of the cannon bone has received much attention in the literature, recently. It has been given many names such as bone bruising, mal-adaptation, and repetitive cyclic stress, all of which indicate damage to the bone in excess of the bones ability to repair that damage.[27] Initially this was thought to be a disease only of the racehorse, but, more recently, the same syndrome has been implicated in reining horses, cutting horses, eventing horses, and jumping horses.[a] It is less common in these uses but it makes sense that any horse subjected to similar exercise levels would sustain the same pathophysiologic response, which, if insufficient to compensate for the cumulative damage, would then result in pathology that creates performance limiting lameness and clinical disease. This pathology is a very common cause of a horse's sudden dislike of training or refusal to perform routine tasks that it once enjoyed. In the racehorse, this is often seen as a sudden refusal to enter the starting gate when ordinarily the horse is unfazed by the loading process. It is also manifest by the lack of desire to train or go to the racetrack when the horse's normal response has been enjoyment of training, as would be expected. We once termed this behavior "track sour," though we now realize because of better diagnostic methods and better understanding of the pathologic process that this is simply an avoidance of the pain that the racing and training induces in the bruised distal cannon bone, if the training is proceeding faster than the bone can respond. Many clinical descriptions of this syndrome, its diagnosis, and its treatment exist in the literature.[20] The most successful approach to the treatment of this disease in the author's hands and in the literature is the use of free choice exercise to allow the horse to heal the injury before reinitiating training.[27]

The premise for this treatment is that the horse is normally a grazing animal. As such, the evolution of the horse's distal-limb circulatory system evolved with the horse spending much of its time grazing in a field. Horse grazing involves a few bites of grass followed by a few steps across the field and then a few more bites of grass followed by a few more steps, for hours at a time. Contrary to this evolution, we have altered the training horse's daily routine to involve a short time, perhaps an hour, of high intensity exercise followed by many hours of stall confinement. This creates the trauma of exercise but

eliminates the natural stimulation of circulation that would best support repair. We see the result of this perturbation of the horse's natural routine as the accumulation of edema within the distal limb ("stocking up") of the horse when they are stall-confined for long periods of time after intense exercise. We circumvent the edema with the application of "stall bandages" to reduce the edema or "stocking up." The elimination of the edema eliminates the clinical symptom but does not resolve the primary condition of accumulated damage to the bones of the distal limb of the horse. Returning the horse to the natural grazing environment for at least 8 hours of every 24 for a 2- to 3-month period of time resolved the clinical lameness seen with the repetitive cyclic loading and allowed a return to racing in 95% of the horses treated.[27]

Another man-made predisposition to subchondral bone injury in the distal metacarpus is the addition of the excessive "toe grab" in the Thoroughbred racehorse.[28] The toe grab adds a braking affect to the impact of the horse's foot in the traditional dirt-racing surface. Experiments with instrumented shoes have shown that the forces that these horses receive at the impact of the foot with a toe grab into the traditional racing surface exceed the load received with weight-bearing.[29] The toe grab increases the resistance to slide of the foot in the forward direction and causes the toe to turn down into the loose cushion of the classic dirt-racing surface. Lowering the toe creates a functional elevation of the heel of the foot, which then translates into a reciprocal increase in extension within the fetlock. Hyperextension of the fetlock joint increases the load on the suspensory apparatus and the palmar/plantar articular surface of the cannon bone at its interface with the sesamoid bone. This increased load increases the damage to the subchondral bone, cyclically producing damage in excess of repair, bruising, and structural damage to the articular surface of the fetlock joint in this location.[20,30] Because the articular surface has a limited means to adapt (because it cannot change its shape), this leads to the myriad of clinical lameness encountered as a result of this process if training continues, proceeding faster than the horse can adapt. The same forces will be in play on other surfaces such as turf or artificial racing surfaces, but experimental studies have shown that the magnitude is greatest on the traditional dirt racing surface.[30] This syndrome at least partly explains the high incidence of injury to the third metacarpus of the racehorse fetlock joint when compared with other articulations of the distal limb.

Recent questions have surfaced as to whether we could have inadvertently altered the genetic material and the bone's ability to adapt by selective breeding, increasing the predisposition of the horse to injury. Progressive decrease in the number of races per year completed by the North American Thoroughbred seems to raise the question, but closer examination indicates the economics of racing are probably a bigger driver of the decreases in starts than the question of soundness.[b] Publication of trainers' statistics concerning the percentage of horses that finish in the top three places and the continual growth of the expense concerned with training without a similar growth in purses encourages more selective entry of horses in races. This increases the amount of training, and the participation in a race occurs only when the connections feel the horse is at peak form. Emphasis on the horse's value as breeding stock also promotes the level of performance rather than the number of races competed. These economic pressures continually reduce the emphasis on career longevity and reward early retirement with lucrative breeding careers. One would suspect that the emphasis on early career "brilliance" and the de-emphasis of longevity would eventually take a toll on the ability to the racehorse to produce durable skeletons and long-lived careers.

In summary, bone is not a purely structural inert material; it is a dynamic support tissue that adapts to the work it is asked to do. Most clinical diseases are the result of training and damage in excess of the horse's ability to respond to the exercise stimulus. Understanding of the horse's bone response to exercise and modulation and adaptation of exercise programs to individual horses and to their response to the stimulus greatly aids in the avoidance of injury in the equine athlete.

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Pathologic Changes and Diagnostic Workup of Palmar/Plantar Metacarpal/Metatarsal Condylar Disease in the Thoroughbred Racehorse

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1. Introduction

The palmar aspect of the third metacarpal condyles (PMC) and the plantar aspect of the third metatarsal condyles (PMT) are common sites of injury in the Thoroughbred racehorse. Several disease processes can occur at these sites including condylar fracture, traumatic arthrosis, and osteoarthritis. The PMC and PMT articulate with the proximal sesamoid bones at maximal extension, causing high pinpoint loading at that site. The dorsal aspect of the condyles also articulate with the proximal aspect of the first phalanx, probably adding to the compressive stress across the condylar surface. The purpose of this presentation is to review the pathologic processes that occur in the PMC and PMT, the clinical techniques used to diagnose such diseases, and the various therapies used for their treatment.

2. Normal Adaptation and Pathologic Processes

The PMC and PMT undergo normal adaptive bone remodeling and modeling to withstand the intense loading of training and racing. At maximal extension, it appears that the bone in the PMC and PMT becomes thickened in the subchondral and trabecular areas between the articulations of the proximal sesamoid bone and proximal first phalanx. However, the quality of bone produced and its integration with parent bone can be influenced by the intensity of training.

Most racehorses do not have PMC or PMT disease, but, probably because of normally adaptive bone, there are indications that some diseases of the PMC and PMT could begin to develop before training. Lucencies and defects within the condyles and parasagittal groove have been seen in samples from young Thoroughbred horses before training.[1,2] Areas of thin and fragmented calcified cartilage with subchondral bone resorption are often present, with areas of hypermineralized matrix protruding into articular cartilage, indicative of pathologic changes during development.[3] The hypermineralized tissue within the calcified cartilage layer may create a density gradient predisposing the area to fracture.[4] Degenerative changes in articular cartilage matrix overlying these areas of subchondral bone changes are also seen.[5]

In horses that have trained and raced, the pathologic changes often progress. Abnormal bone ma-

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trix and reduction in osteocyte volume are often present in these horses.[6] Significant bone modeling is commonly seen and is the primary form of bone adaptation, with little remodeling, except in areas of microdamage.[7,8] The intense bone modeling also appears to cause a density gradient, which may create a strain environment conducive to fracture.[9–14] The articulation between the palmar/plantar aspect of the third metacarpal/metatarsal bones and the proximal sesamoid bones undergoes high stress. The proximal sesamoid bones articulate with both the condylar surface and the abaxial surface of the transverse ridge at this site. Consequently, in the young horse, there is good trabecular pattern in both the condylar surface and the abaxial surface of the transverse ridge; however, in the parasagittal groove, a highly remodeled/modeled trabecular pattern is usually apparent. This is a site of relatively low subchondral bone density even in a young, trained horse. It has been shown in young Thoroughbred horses that this is a site of poor articular cartilage integrity and strength and a site of significant subchondral bone density gradient in some horses.[2,15] Microdamage is often present in fractured joints and at sites predisposed to fracture.[16,17] The severity of subchondral bone changes appears to be associated with overlying articular cartilage changes.[18]

Condylar fractures typically occur within the parasagittal groove, originating at the palmar/plantar aspect of the third metacarpal/metatarsal condyles. This correlates well with the pathologic changes mentioned above. However, condylar fractures can commonly occur more abaxially on the condylar surface.[19–21] Therefore, the study of pathologic changes leading to fracture must include changes on the condylar surface. In addition, there may be associated changes on the dorsal aspect of the condyle that are worth investigating.

Factors that can affect the incidence of fracture include the shape of the bone,[14] bone quality, the intensity of the remodeling/modeling cascade (often times affected by training intensity), bone quality, and the shear stresses at the interface between old bone and new remodeled/modeled bone. One must consider that conformation may be involved as a factor in these cases.

Significant articular cartilage erosion and subchondral bone exposure can occur at the PMC and PMT. This has been referred to by several names, including palmar arthrosis, overload arthosis, and palmar/plantar osteochondral disease. Postmortem specimens of this disease often demonstrate significant subchondral bone remodeling/modeling, which can often lead to significant sclerosis at that site. In some cases, the bone mineralization front can enter into and through the non-calcified articular cartilage, leading to erosion.[22–24] However, even in a horse training without this problem, significant bone density can be apparent. Some horses with pain in this area often can have edema at the site without any structural damage at that point.[25] However, structural damage can occur with progression of this disease. This commonly manifests as subchondral bone microdamage and consequently, necrosis.[22] The necrosis can continue to occur while the articular cartilage is intact. This cascade often leads to subchondral bone modeling, which advances through the calcified cartilage and into the articular cartilage. Once the articular cartilage either collapses into a cyst-like structure or subchondral bone completely models through the cartilage layer, then the horse can become refractory to treatment.

Osteoarthritis (OA) can result from any of the above pathologic processes.[26] In some cases, the origin of OA is idiopathic and the horse typically shows significant erosions, wear lines, osteophytes, and joint collapse, which are typical of OA.

3. Diagnostic Techniques

Depending on the type of injury in the joint, the horse can either show overt signs of clinical disease within the fetlock joint or in more subtle cases the joint may have no outward signs of disease. Horses that show no outward signs of disease other than a history of training sore can be a diagnostic challenge. For disease processes that do not involve the actual synovial environment, there may be no synovial effusion and the horse may be lame and may or may not be positive to digital flexion. Often, these horses will not improve with intra-articular analgesia of the joint; however, a low four-point nerve block often alleviates the lameness. More specifically, diagnostic analgesia of the lateral and medial palmar metacarpal nerves may alleviate the lameness in the forelimb, and a block of the lateral plantar metatarsal nerve may alleviate the lameness in the hind limbs.[27] A full series of radiographs should be performed, and, especially in the Thoroughbred racehorse, radiographic imaging of the palmar/plantar aspect of the joint should also be performed. This entails a 35° dorsodistal-palmaroproximal oblique projection and a flexed 30° plantarodistal-dorsoproximal oblique projection.[27] Downward oblique images (a dorsoproximolateral-palmarodistomedial [plantarodistomedial] oblique projection and a dorsoproximomedial-palmarodistolateral [plantarodistolateral] oblique projection) may also be performed to highlight the palmar or plantar aspect of the third metacarpus/metatarsus.[27]

Nuclear scintigraphy often will demonstrate radioisotope uptake in the palmar/plantar aspect of the joints. However, one must remember that in the young Thoroughbred racehorse, there may be significant increase in radioisotope in this area caused by training. However, for horses that have disease at that site, the degree of radioisotope uptake can be significant.[27] Trope et al[28] found that increased radiopharmaceutical uptake in the PMC and PMT was the most common abnormality in

Thoroughbred racehorses but was not sensitive for screening fracture risk.

Magnetic resonance imaging to characterize disease in the PMC and PMT area is now the gold standard. Powell et al[25] have demonstrated all of the disease processes characterized in this report. This includes demonstrating subchondral bone edema, sclerosis, necrosis, and fracture. Although a high-field MRI is typically preferred, general anesthesia is needed. However, Powell et al[25] have demonstrated the use of low field standing MRI to demonstrate these lesions. In a postmortem study, Tranquille et al[29] found MRI to be sensitive for detecting small incomplete lateral condylar fractures.

Computed tomography has also been used to characterize diseases in the PMC and PMT. Incomplete fractures can be detected as well as subchondral sclerosis and necrosis.[30,31]

Treatment of diseases in the PMC and PMT varies, depending on the disease process present. For condylar fractures, an incomplete or a complete fracture that involves the joint should be surgically reduced through the use of the lag screw technique to give the best prognosis. Arthroscopic visualization of the joint surface is suggested because the prognosis is often determined by articular cartilage damage in the joint. In general, retrospective studies of condylar fracture repair demonstrate that horses with incomplete fractures carry a better prognosis than those with complete fractures and that prognosis diminishes with increasing pathologic change within the joint.[32–34]

Palmar arthrosis, depending on its severity, often will respond to rest in the early stages; however, with more advanced disease the pain becomes more difficult to control.[27] Intra-articular medication is thought to work in some cases; however, in those horses, a point is reached of which that is no longer effective. Systemic non-steroidal anti-inflammatory drugs can also be used to help control pain in this area.

For osteoarthritis, medical therapy is used to help control pain and disease progression in these areas. Intra-articular stem cell therapy may have some benefit; however; this is still unproven in the metacarpophalangeal/metatarsophalangeal joints.

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Diagnostic Workup of Upper-Limb Stress Fractures and Proximal Sesamoid Bone Stress Remodeling

Susan M. Stover, DVM, PhD, Diplomate ACVS

Stress fractures and stress remodeling occur in specific locations within the scapula, humerus, and proximal sesamoid bones of racehorses. Knowledge of locations and of racehorse characteristics that place racehorses at high risk for scapular and humeral stress fractures and remodeling injuries enhances veterinarians' ability to detect scapular and humeral stress lesions. Detection of stress remodeling in proximal sesamoid bones is more challenging because of the small size and location of the lesions. Author's address: JD Wheat Veterinary Orthopedic Research Laboratory, Department of VM:APC, School of Veterinary Medicine, University of California at Davis, One Shields Avenue, Davis, CA 95616; e-mail: smstover@ucdavis.edu. © 2013 AAEP.

1. Introduction

As elite athletes, racehorses are susceptible to overuse injuries from repetitive motions incurred during intense training and competition. Thoroughbred and Quarter Horse racehorses competing over flat racecourses have respective sets of occupational skeletal injuries related to sites of concentrated high stresses incurred during task-specific training and racing activities. These injuries are typically referred to as stress fractures or fatigue fractures when the affected site is associated with the cortex of a long bone and as stress remodeling when the affected site is in a trabecular or subchondral location. The etiopathogenesis and biological repair mechanisms of these repetitive, overuse injuries are similar, irrespective of location. Fundamentally, injured bone tissue is capable of healing and regeneration. However, continued training and racing of horses with stress fractures or sites of active stress remodeling can promote severe or catastrophic fractures or irreversible osteoarthrosis when the underlying bone tissue is weakened and can no longer support the overlying articular cartilage.[1,2] Thus, it is important to identify stress fractures and sites of stress remodeling in racehorses so that affected racehorses can be appropriately rehabilitated to allow healing and return to athletic performance.

The purpose of this presentation is to enhance awareness of preferential sites and features of stress fractures in the shoulder and arm of the forelimb and of stress remodeling in the proximal sesamoid bones so that lesions in these regions are more likely to be detected and appropriately managed to achieve resolution in racehorses.

2. Unsoundness Associated With a Stress Fracture or Stress Remodeling

Lameness varies markedly in severity between affected horses, from unapparent or only a change in demeanor to non–weight-bearing.[3,4] Lameness

NOTES

is most commonly noted immediately after a training or racing event[3,4] and may appear to resolve within days of onset. Stress fractures and stress remodeling commonly affect bilateral limbs because the repetitive stresses associated with training and racing are incurred by both forelimbs or hind limbs.[5,6] Consequently, affected horses may not demonstrate a distinct unilateral limb lameness. Instead, bilateral limb lameness may manifest as an unwillingness to perform at an expected level, a change in attitude, or unusual resistive behavior (for example, unwillingness to enter the starting gate). Poorly performing horses should be given a thorough physical examination to rule out nonmusculoskeletal causes and elucidate skeletal injuries. Knowledge of the sites predisposed to stress fracture enhances ability to detect these injuries in racehorses.

3. Scapular Stress Fractures

Complete scapular fractures occur through the site of a pre-existing stress fracture.[2,5] Complete scapular fractures are the cause of 2% and 4% to 8% of fatal musculoskeletal injuries in Thoroughbred and Quarter Horse racehorses, respectively.[2,7–9] Approximately 1 in 2500 Thoroughbred starters and 1 in 1000 Quarter Horse starters incurs a complete scapular fracture during a race.[10] Although the overall prevalence and incidence of scapular stress fractures in racehorses are unknown, scapular lesions comprised 2% of positive findings for forelimb scintigraphic examinations in Thoroughbred racehorses, with some horses affected bilaterally.[11]

Scapular stress fractures occur along the typical site and configuration of complete scapular fractures in racehorses (Fig. 1). Complete fractures occur transversely or obliquely at the distal end of the spine of the scapula, dividing the scapula into a large proximal component and a smaller distal component.[5,7,12] Although comminution, distal propagation into the glenoid, and proximal propagation of incomplete fracture lines are common, these are secondary to the transverse/oblique fracture component. Stress fractures occur predominantly at the distal end of the spine of the scapula in racehorses that died because of a complete scapular fracture,[5,7] but stress fractures have also been reported to occur in the infraspinous fossa and supraspinous fossa, sites along the course of the transverse/oblique component of the complete fracture.[4,5,13]

Horses are at highest risk for complete scapular fracture early in their career (2 years of age) or as 5-year-old or older horses, although racehorses of all ages can be affected.[4,10,13] They occur more commonly in males than females. The right forelimb is affected over twice as frequently as the left forelimb, and bilateral complete fractures affect approximately 9% of horses that died because of a complete scapular fracture.[10]

Complete scapular fractures occur during racing or during training. Approximately a quarter of affected Quarter Horses and a third of Thoroughbreds had never raced. Only half of unraced horses had completed an official timed work. Horses that incurred complete scapular fracture had fewer high-speed official timed works and races, lower accumulated high-speed distance, and fewer days in active training than age matched control horses.[14] Consequently, racehorses that are in early high-speed training but behind that of their training cohort, Quarter Horses that had a prolonged lay-up, and Thoroughbreds in high-speed training for a longer duration than that of their training cohort should be examined for signs of scapular stress remodeling.[14]

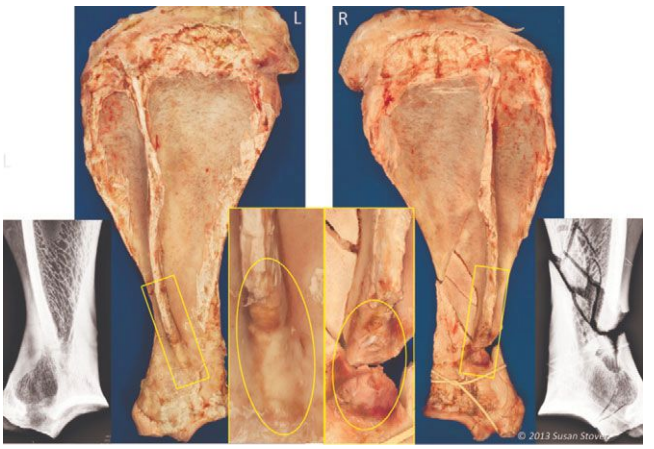


Fig. 1. Bilateral scapulae from a racehorse that had a unilateral (right) complete fracture of the right scapula. Periosteal callus associated with underlying bone resorption is present bilaterally, at the distal end of the neck of the scapulae. Periosteal callus attempts to buttress the spines, which are affected with an underlying intracortical weakness created by intracortical porosities and low bone mineral density. The focal region of osteoporosis creates a stress riser that facilitates bone fracture at otherwise physiologic loads. Stress remodeling occurs bilaterally, but the weakest scapula fails catastrophically. In some affected racehorses, both scapula catastrophically fracture.

Clinical Presentation and Detection

Horses affected with a scapular stress fracture are usually presented because of acute onset of lameness after a race or high-speed work.[4,13] Lameness is generally rated 2 to 3 of 5 (American Association of Equine Practitioners lameness scale). Importantly, the lameness may appear to resolve quickly. As expected, the lameness does not improve with diagnostic and regional nerve blocks administered at the level of the carpus and distally, although diagnostic blocks should be done with caution because of the risk of catastrophic fracture.

Physical examination is useful in detecting some affected horses. Some horses have responded positively to forelimb abduction and to palpation of the scapular spine (Fig. 2).[4,13] Ultrasound examination can be useful for detecting irregular periosteal modeling/remodeling, a stair-step in the spine or



Fig. 2. The distal end of the spine of the scapula (right hand) can be palpated between the supraspinatus muscle (cranially) and the infraspinatus muscle (caudally) proximal to the groove between the cranial and caudal parts of the greater tuberosity of the humerus (left thumb).

cortical surface, or hematoma,[4,13] but these findings may not be present in horses with acute sub-periosteal bone trauma (Fig. 3). Radiography has not been useful for detection of scapular stress fractures, largely because of limitations related to extensive overlying soft tissues and bony structures of the thorax and neck. Scintigraphy remains the gold standard for detection of a focus of high metabolic activity at sites consistent with scapular stress fracture and is useful in acute and chronic stages of disease (Fig. 4).[4,11,13]

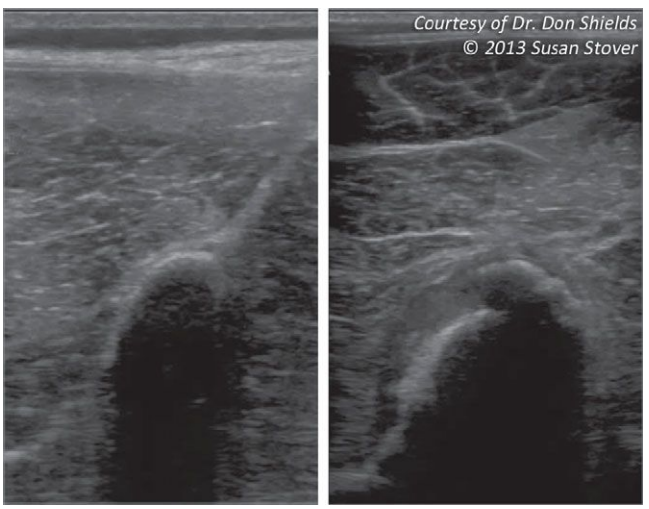


Fig. 3. Transverse ultrasound images of the distal end of the spine of the left (asymptomatic) and right (affected) scapulae of a racehorse with a stress fracture of the right scapula. The spine of the left scapula is narrow and symmetric and has a normal distinct, smooth surface contour. The spine of the right scapula is markedly thickened and has an abnormal irregular surface contour that is consistent with production of periosteal callus.

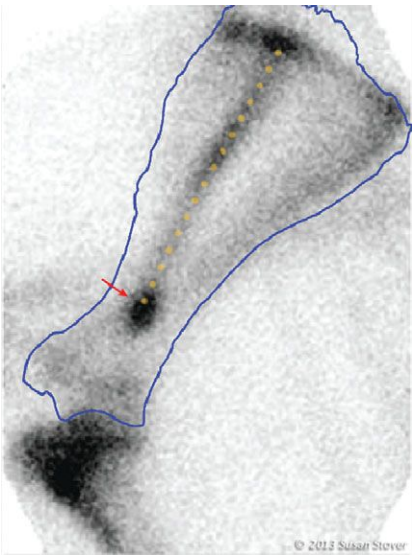


Fig. 4. Bone scan of the left scapula of a horse with a stress fracture at the distal end of the spine of the scapula.

Rehabilitation

Conservative therapy of horses with scapular stress fracture has allowed return of racehorses to successful race performance, even after recurrence of a scapular stress fracture.[4,13] Two months (60 days) of stall confinement, followed by 30 to 60 days of turnout in a small paddock, has been recommended before re-introduction to race training.[4] Response to treatment and length of rehabilitation is guided by follow-up scintigraphic examination. Rehabilitated horses have raced from 6 to 17 months (median, 9 months) after diagnosis of a scapular stress fracture.[13]

4. Humeral Stress Fractures

Humeral injuries comprised 6% of lesions for Thoroughbred racehorses examined scintigraphically for a musculoskeletal problem[15] and 10% of positive findings for forelimb scintigraphic examinations in Thoroughbred racehorses,[11] with 15% to 29% of horses affected bilaterally.[11,15] More recently, 20% of racehorses that had a bone scan that included the humerus had a humeral lesion.[16]

Three-year-old racehorses are most commonly affected with humeral stress fracture; however, only 32% to 51% of horses had some race experience before diagnosis.[3,15,17] Collectively, several studies support a left limb predilection for stress fractures in live racehorses[3,15,16] (although this predilection can vary with racetrack surface material[16]) and catastrophic fractures in deceased racehorses[16]; but both forelimbs can be affected and a large proportion of affected racehorses have bilateral lesions.[16]



Fig. 5. The proximal aspect of the left humeri from two different horses demonstrates a complete fracture coursing through an acute stress fracture (left) and a chronic stress fracture (right). The humerus on the left has periosteal callus that is a thin (<2 mm thick) layer of highly vascular (red), rough-surfaced, woven bone. These features are characteristic of an acute periosteal reaction. The humerus on the right has a larger diameter periosteal callus with two features. A thick (~8 mm in height), dense, central callus is indicative of bone that was formed and remodeled to yield more compact dense bone, which is more typical of lamellar bone. Surrounding the central callus is a thin layer of rough surfaced woven bone. Together, these two features indicate a recent exacerbation of a more chronic lesion.

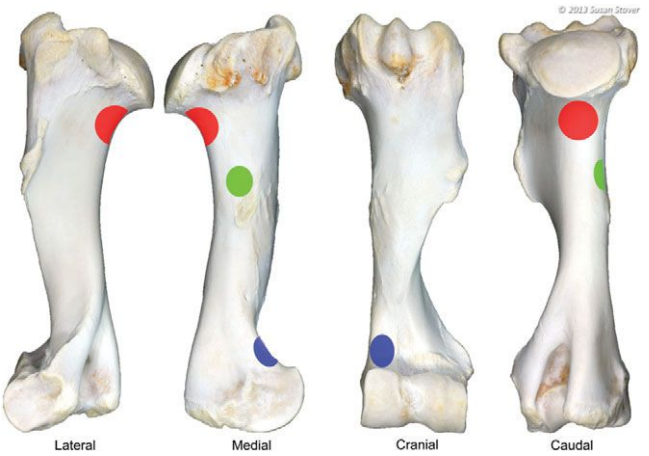


Fig. 6. Lateral, medial, cranial, and caudal views of the left humerus illustrate the most common locations of stress fractures in the humerus: caudoproximal at the neck of the humerus (red), craniodistomedial (blue), and medial (green).

Complete humeral fractures were the cause of 3% to 9% and 0% to 13% of fatal musculoskeletal injuries in Thoroughbred and Quarter Horse racehorses, respectively.[2,7–9,18] Complete humeral fractures are known to be associated with a pre-existing stress fracture (Fig. 5).[6,7] Humeral stress fractures occur at several typical sites on the humerus in racehorses.[3,6,15,16,19] Because stress fractures at caudoproximal (37% of stress fractures), medial diaphyseal (5%), and craniodistal (31%) sites occur along the typical long oblique catastrophic complete humeral fracture that occurs in racehorses (Fig. 6),[6,7,16,20] the consequences of continuing to train and race on stress fractures at these sites may be more severe than stress remodeling at other sites. Most complete fractures (69–85%) from convenience samples of completely fractured humeri from racehorses extended through a caudoproximal region of stress remodeling.[6,16]

Complete humeral fractures occur more commonly during training (89–91%) and often at a slow gallop but also occur during racing.[6,17] However, 49% to 63% of affected horses had not raced before diagnosis of a humeral stress fracture.[6,15] Risk for complete humeral fracture is highest after an increase in exercise after a 2-month or longer layup.[17]

Clinical Presentation and Detection

Horses affected with a humeral stress fracture are usually presented because of acute onset of lameness after a race or high-speed work but may have a chronic insidious lameness duration of weeks to months.[3,15] Lameness can range from 1 to 5 of 5 (American Association of Equine Practitioners lameness scale), with most horses having grade 2 to 4 lameness and some horses initially non–weight-bearing.[3,15] Importantly, the lameness may appear to improve quickly, even within 24 hours.[15] As expected, the lameness does not improve with diagnostic and regional nerve blocks administered at the level of the carpus and distally,[15] although diagnostic blocks should be done with caution because of the risk of catastrophic fracture. Lameness was exacerbated in about half of affected horses after manipulation of the shoulder and elbow joints during flexion, adduction, and abduction of the upper portion of the limb.[3,15] Horses with severe lameness typically had a shortened cranial phase of the stride at the walk and trot.[15]

Radiographs have been useful for detection of humeral stress fractures but cannot be relied on for diagnosis of acute stress fractures when bone changes are insufficient to alter the radiographic appearance of affected humeri.[3,15] Radiographic evidence of a stress fracture was observed in 56% to 92% of affected horses, 69% to 80% of caudoproximal stress fractures, and 71% to 100% of craniodistal stress fractures (Fig. 7).[3,15] In some cases, radiographic detection may have been enhanced because radiography was preceded by scintigraphic diagnosis. It is notable that radiographic findings can be negative when lameness is first noticed but positive 10 days to 12 months later.[3]

The author is unaware of any reports that demonstrated the diagnosis of humeral stress fractures through the use of ultrasound examination. However, ultrasound detection of periosteal new bone may be achievable with knowledge of high-risk horses and sites of predisposition.

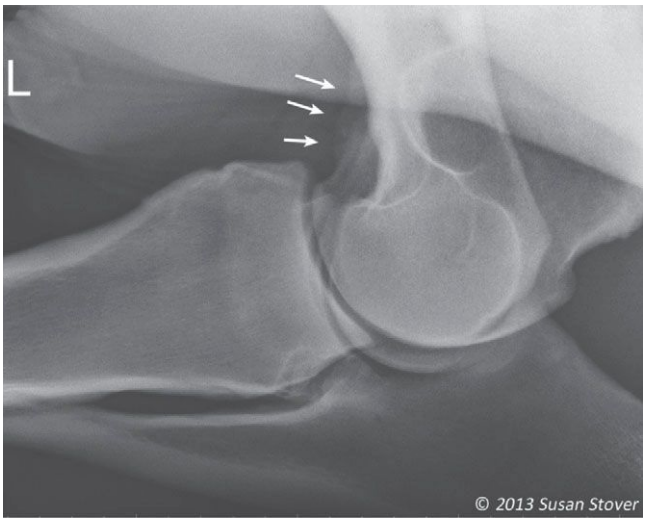


Fig. 7. Lateromedial radiograph of the left elbow of a racehorse demonstrating periosteal callus (arrows), consistent with stress fracture of the craniodistomedial aspect of the humerus.

Scintigraphy remains the gold standard for detection of a focus of high metabolic activity at sites consistent with humeral stress fracture and is useful in acute and chronic stages of disease (Fig. 8).[4,11,13] In two separate studies, none of the horses diagnosed with a humeral stress fracture developed a catastrophic complete humeral fracture in the affected humerus.[15,16] In contrast, none of the 32 racehorses that developed catastrophic fracture had a known record of a scintigraphic examination.[16]

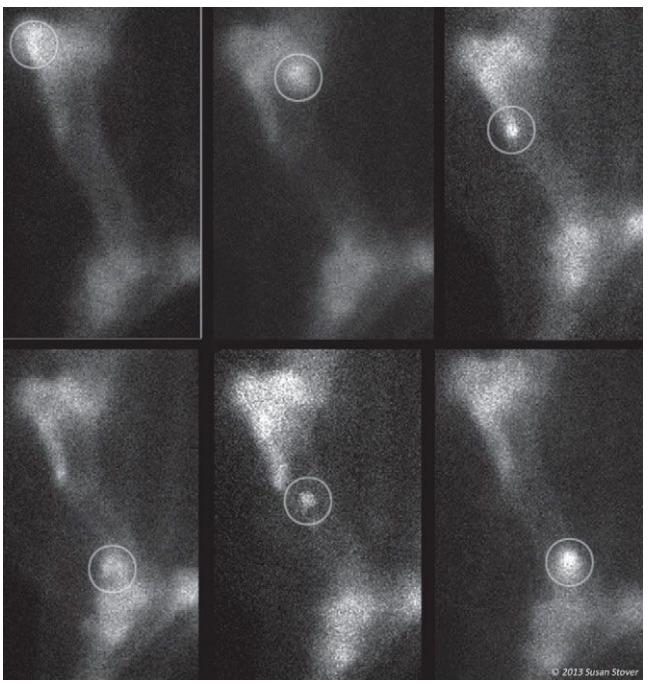


Fig. 8. Lateral view bone scans of humeri demonstrating craniproximal (upper left), caudoproximal (upper center), deltoid tuberosity (upper right), craniodistal (lower left), middle diaphyseal (lower center), and caudodistal (lower right) lesions.

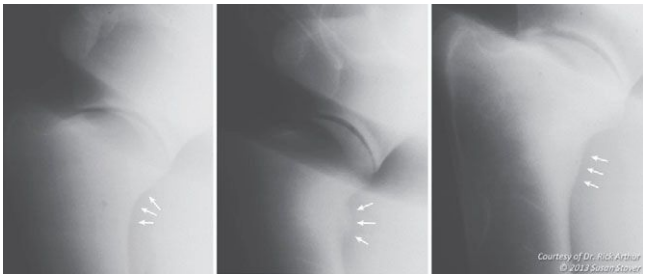


Fig. 9. Lateromedial radiographs of the proximal aspect of the humerus from the same horse at the initiation of clinical signs (left) 1 month later (center) and 3 months later (right). The contour of the humeral neck changes from the normal concave contour to convex with apposition of periosteal callus to blunted with remodeling and flattening of the callus to result in bone adaptation by residual expansion (thickening) of the cortex.

Rehabilitation

Conservative therapy of horses with humeral stress fracture has allowed resolution of lesions and adaptation of the humerus to the stresses of racing by expansion of the affected cortex (Fig. 9). Racehorses return to race performance, even on the occasion when a humeral stress fracture recurs and is managed in the same or another location.[3,15] Exercise restriction in the form of 4 weeks of stall rest with limited hand-walking if not lame at the walk; when not lame at the trot, 4 weeks of turnout in a small area, then 4 to 8 weeks of turnout in pasture; and resumption of training.[15] These periods would be extended if lameness persisted at any stage. When possible, response to treatment and length of rehabilitation is guided by follow-up scintigraphic examination. Median time to return to racing after stress fracture diagnosis was 7.5 months (range, 5–22 months) for the 77% of horses that raced after fracture.[15] Median number of races after fracture was 8.5 races (range, 1–33 races).[15] No significant difference was found for mean earnings per start between before and after fracture.[15] However, humeral stress fractures can recur at the same or distant sites in the same or contralateral limbs.[3,15]

5. Proximal Sesamoid Bone Stress Remodeling

Proximal sesamoid bone fracture (with or without associated metacarpal bone fracture) is the most frequent catastrophic (fatal) musculoskeletal injury in racehorses, accounting for 45% to 50% of Thoroughbred injuries and 37% to 40% of Quarter Horse injuries.[1,2,8,18] Although small displaced abaxial, apical, and basilar fragments are common in racehorses, these findings do not lead to catastrophic fractures of the proximal sesamoid bones. Complete transverse or oblique fractures of the proximal sesamoid bones disrupt transmission of force from the suspensory ligament to the distal sesamoidean

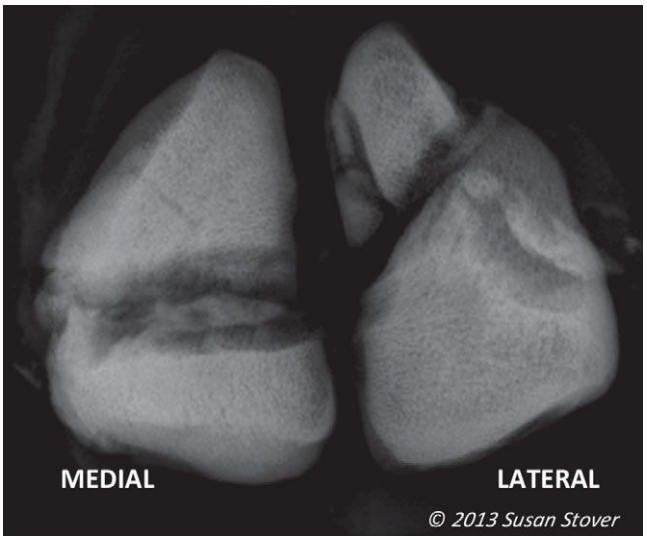


Fig. 10. Dorsopalmar radiograph of proximal sesamoid bone specimens illustrates a common fracture configuration. The medial proximal sesamoid bone incurs a relatively simple transverse fracture that splits a larger proximal fragment from the base. The lateral proximal sesamoid bone fracture varies more but commonly has an oblique fracture that separates the apex from the body.

ligaments and are the most common cause of fetlock injuries that are catastrophic because they disrupt the integrity of the suspensory apparatus.[21]

The most common configuration for catastrophic fetlock injury is biaxial proximal sesamoid bone fracture.[2,9,21] Complete, transverse, articular fracture that separates the body from the base of the medial proximal sesamoid bone is accompanied by complete, oblique or transverse, articular fracture of the lateral proximal sesamoid bone (Fig. 10).[21] Fractures often have some comminution and disruption of adjacent soft tissues. Although other proximal sesamoid bone fracture configurations also disrupt the suspensory apparatus, the focus of this report will be on medial proximal sesamoid bone fracture because of lesions that probably predispose to fracture and initiation of suspensory apparatus failure.[22,23]

Two lesions probably predispose to medial proximal sesamoid bone fracture.[22,23] Both lesions are foci of bone resorption that occur in response to the local accumulation of microdamage acquired from the repetitive load cycles associated with training and racing. The first lesion that was recognized occurs on the palmar margin of the proximal sesamoid bone (Fig. 11).[22] The second lesion is more common and occurs consistently within a subchondral, abaxial location within the medial proximal sesamoid bone (Fig. 12).[23] Both lesions are large enough to create a stress riser sufficiently large to initiate bone fracture but small enough, and, in difficult locations, to detect with the use of radiography.

Clinical Presentation and Detection

The clinical signs that distinguish horses with impending catastrophic fracture of the proximal sesamoid bones from horses that can race successfully without injury is challenging. However, several pieces of evidence demonstrate that some features of horse signalment and history and some clinical signs are associated with increased risk for catastrophic fracture.

Racehorses that have catastrophic failure of the fetlock suspensory apparatus, including horses with proximal sesamoid bone fractures, are typically older horses (≥5 years old), more likely to be male than female, have had a long racing career (≥20

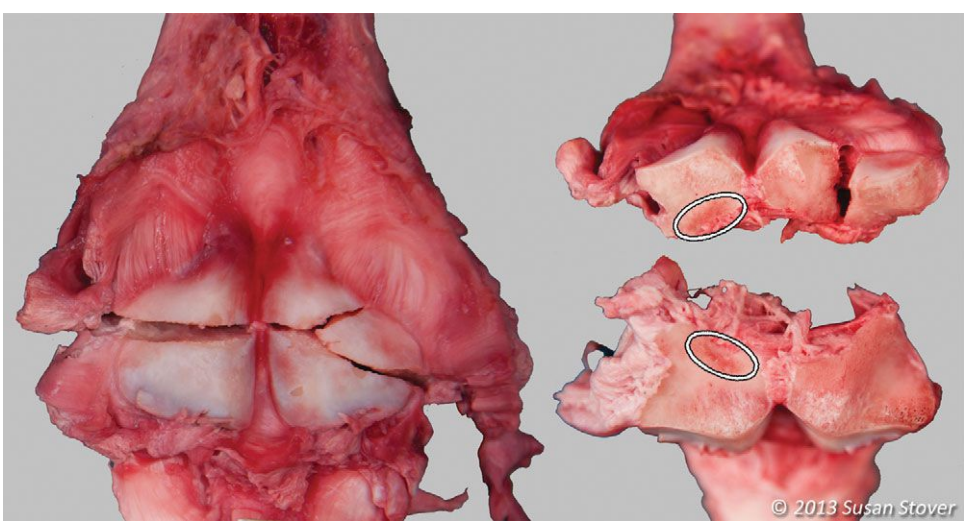


Fig. 11. Dorsal (articular) surface of the proximal sesamoid bones from the fractured leg of a racehorse with fractures of the medial and lateral proximal sesamoid bones. The fracture surfaces of the medial proximal sesamoid bone have been opened to visualize the stress-remodeling lesion (ellipses) on the palmar aspect of the bone.

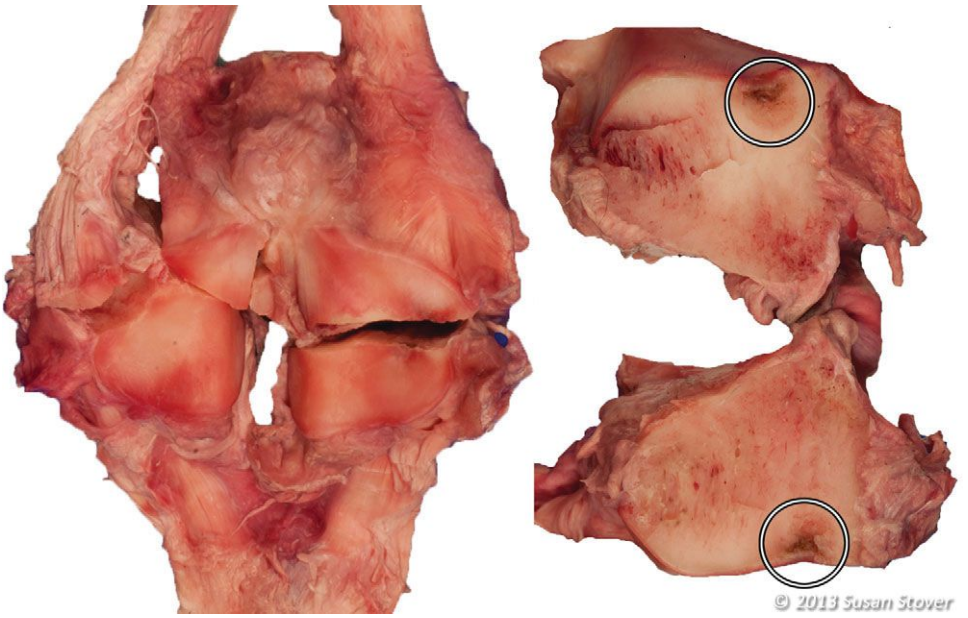


Fig. 12. Dorsal (articular) surface of the proximal sesamoid bones from the fractured leg of a racehorse with fractures of the medial and lateral proximal sesamoid bones. The fracture surfaces of the medial proximal sesamoid bone have been opened to visualize the stress-remodeling lesion (circles) in an abaxial subchondral location.

races), and have experienced prolonged high exercise intensity.[24,25] Furthermore, horses that had a catastrophic fetlock injury were 3 times more likely to have had a palpable abnormality of the suspensory ligament; they were 2 times more likely to have had an abnormality of the fetlock joint on pre-race inspection; and the pre-race veterinary inspector was 8 to 14 times more likely to have considered the horse to be at higher risk.[9,24] The usefulness of these findings is tempered by the low specificity of the findings; that is, a much larger number of horses had a similar abnormality but did not receive a subsequent injury in the associated race.[26] However, when the long-term performance of horses is considered, 45% of horses with signs of mild suspensory apparatus injury had a training remission caused by a severe musculoskeletal injury within 3 months compared with 14% of horses without the same signs.[27] Thus, horses with mild injuries to the suspensory apparatus are much more likely to drop out of the racing pool.

The palmar and subchondral locations of the foci of stress remodeling create challenges in lesion detection. Synovial effusion and synovial fluid abnormalities are unlikely to result directly from stress-remodeling lesions in the proximal sesamoid bones because neither lesion is intra-articular or intrathecal. However, synovial effusion of the fetlock joint may be present because other degenerative lesions (eg, proximodorsal proximal phalangeal osteochondral fragments, score lines, palmar osteochondral disease) usually accompany proximal sesamoid bone lesions. Similarly, intra-synovial anesthesia may not result in improvements in lameness unless lameness is caused by other attendant intra-synovial degenerative changes. However, regional anesthesia that desensitizes the fetlock region is likely to improve any observed lameness or result in enhancing lameness in the contralateral limb because stress remodeling is often bilateral.

Radiographs have not been useful for detection of lesions that might predispose to catastrophic fracture of the proximal sesamoid bone.[21] Paradoxically, among all proximal sesamoid bones from horses that had a catastrophic proximal sesamoid bone fracture, the likelihood of bone fracture was reduced by half when the bone had radiographic evidence of osteophytes and to less than a quarter when the bone had prominent vascular channels.[21] The two sites of stress remodeling are in difficult locations to visualize radiographically because of overlying bone tissue and contours (Fig. 13). Whereas the abaxial site appears to be associated with abaxial vascular channels, the subchondral site appears to be associated with basilar vascular channels.

Radiographs are useful for detecting other lesions of the fetlock that usually (but not always) accompany proximal sesamoid bone disease. Horses that sustained catastrophic fracture of the proximal sesamoid bones often have degenerative lesions of the metacarpal condyle (palmar osteochondral disease), osteochondral degeneration or fragmentation of the dorsoproximal margin of the proximal phalanx, and other articular cartilage degenerative changes. However, it is not known how often these changes are present without proximal sesamoid bone disease.

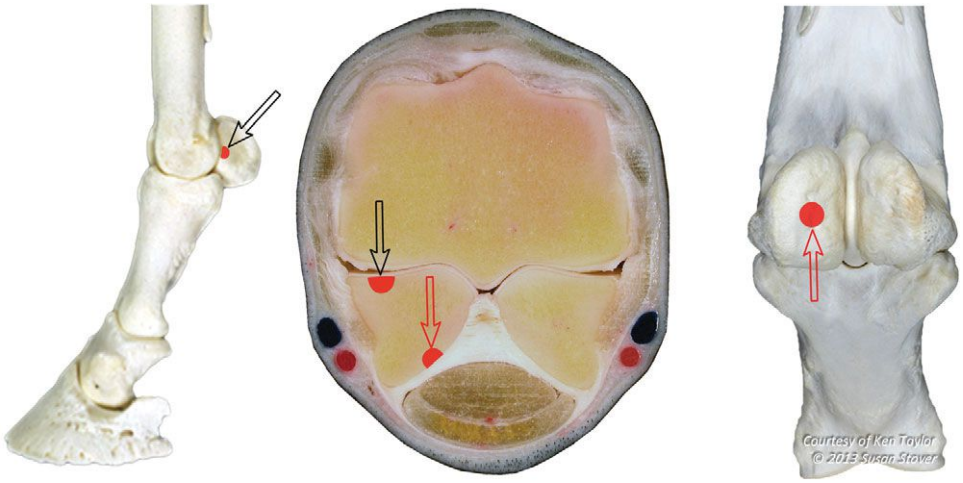


Fig. 13. Palmar (red arrows) and subchondral abaxial (black arrows) sites of stress remodeling (red filled regions) in the medial proximal sesamoid bone, superimposed on medial (left), transverse section (middle), and palmar (right) views of the fetlock region.

Ultrasound examination might be useful for detecting abnormal bone contours on the palmar aspect of the proximal sesamoid bone. Unfortunately, this region of the bone is normally irregular in contour; therefore, distinction of contours caused by lesions from normal contours could be difficult. The author is unaware of any reports that demonstrated the diagnosis of proximal sesamoid bone stress remodeling through the use of ultrasound examination.

Magnetic resonance imaging has potential for detection of stress-remodeling lesions. Technology and image acquisition and analysis software enhancements for standing magnetic resonance imaging machines may make detection of stress-remodeling lesions in the proximal sesamoid bone feasible in horses without the risks associated with general anesthesia.

Clinical computed tomography currently requires general anesthesia, and, in our experience, the small foci of stress remodeling in the proximal sesamoid bones makes lesion detection challenging. Enhancements in computed tomography technology could increase chances of lesion detection.

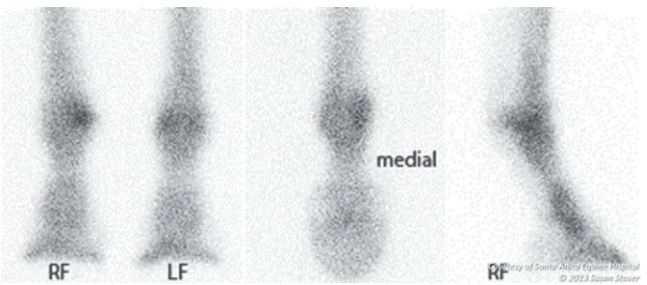


Fig. 14. Bilateral distal forelimb bone scan illustrating increased radiopharmaceutical uptake in a region consistent with a lesion in the medial proximal sesamoid bone.

Scintigraphy remains the gold standard for detection of a focus of high metabolic activity at sites consistent with stress remodeling (Fig. 14). The challenge is differentiating changes associated with adaptive remodeling of the proximal sesamoid bone in response to the increased loads associated with training and racing[22,28] from pathologic or maladaptive remodeling. Furthermore, it can be difficult to differentiate increased activity in the palmar aspect of the metacarpal condyle from increased activity in the proximal sesamoid bone, particularly because palmar osteochondral disease often accompanies subchondral proximal sesamoid bone lesions.[23] Because flexion of the fetlock moves the proximal sesamoid bones proximal to the metacarpal condyle, a lateral image of the flexed fetlock may allow differentiation of proximal sesamoid activity from metacarpal condylar activity.

Rehabilitation

Bone lesions are capable of healing. Affected horses need time without intensive training and racing to allow resolution of bone lesions. Clinically, the difficulty is detecting lesions so that healing progression can be monitored to guide rehabilitation of affected horses.

Acknowledgment

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Managing Joint Disease in the Racehorse in the Face of Stricter Drug Restrictions

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1. Introduction

Intra-articular use of corticosteroids has become a recent focus (or re-focus) of attention in the Thoroughbred racing industry. The clinical use and scientific basis of intra-articular corticosteroid administration including catastrophic injury, articular cartilage degradation, and the development of osteoarthritis (OA) as well as the timing of injection relative to racing have been reviewed.[1] Most recently, there has been very specific examination by the Racing, Medication, and Testing Consortium (RMTC). At the time of that press release, the RMTC had approved (1) minimal withdrawal time recommendations for corticosteroids on the basis of both recently completed work funded in part by the RMTC and conducted at the University of Pennsylvania, University of California-Davis, Kenneth L. Maddy Laboratory, and HFL Laboratory-Kentucky and other corticosteroid research conducted both in the United States and abroad and (2) recommendations were developed during an RMTC-hosted Corticosteroid Experts Conference in Anaheim, California, on November 30, 2012. This meeting brought together qualified individuals with professional expertise in key areas with the goal of providing a comprehensive plan for regulating corticosteroid use in horse racing to protect equine health and welfare. Participants included analytical chemists, veterinary pharmacologists, veterinary surgeons, racing regulatory veterinarians, and practicing racetrack veterinarians. Among the recommendations was a prohibition on intra-articular use of corticosteroids within 7 days of a race, taking into consideration the concerns expressed by many participants about the proximity of intra-articular injections to race day. The experts also recommended a 72-hour withdrawal time for dexamethasone, a commonly used short-acting corticosteroid that can be administered intravenously, intramuscularly and orally. Other short-acting corticosteroids would have similar restrictions. It was also noted that the recommendations would fundamentally change the use of corticosteroids in veterinary practice in racing in the United States and therefore recommended a grace period to allow veterinarians time to adjust their veterinary practices and to allow trainers time to adjust their training practices to comply with the new regulations. Further details regarding these recommendations are discussed below (Table 1).

The purpose of this review is to offer possible modifications and alternatives with the intra-articular and systemic therapy for traumatic joint injury

NOTES

Table 1. Corticosteroid Threshold Chart

Medication	Threshold	Minimum Withdrawal Time*	Route of Administration†	Experimental Administration Dosage‡	Withdrawal Time for 95/95 Tolerance Interval at Experimental Dose and Route of Administration
Betamethasone	10 pg/mL of plasma or serum	7 Days	Intra-articular	Total of 9 mg of betamethasone (as a mixture of betamethasone sodium phosphate and betamethasone acetate) in one joint	7 Days
Dexamethasone	5 pg/mL of plasma or serum	3 Days	Intravenous, intramuscular, and oral	0.05 mg/kg of dexamethasone (as dexamethasone sodium phosphate)	3 Days
Isofluprodone	TBD	TBD	Intra-articular	20 mg total dose of isoflupredone (as isoflupredone acetate, Predef 2X) in one joint	Pending
Methylprednisolone	100 pg/mL of plasma or serum	7 Days	Intra-articular	Total of 100 mg of methylprednisolone (as methylprednisolone acetate) in one joint§	21 Days
Prednisolone	TBD	TBD	Intravenous and oral		Pending
Triamcinolone acetonide	100 pg/mL of plasma or serum	7 days	Intra-articular	Total of 9 mg triamcinolone acetonide in one joint	7 Days

*The RMTC has recommended a 7-day regulatory withdrawal time for all intra-articular use of corticosteroids, recognizing that the clearance times for some will be longer than 7 days. Furthermore, this regulation is not in conflict with the withdrawal time for dexamethasone listed here; the use of dexamethasone intra-articularly is inappropriate because it would be considered off-label and a violation of the provisions of the Animal Medicinal Drug Use and Clarification Act (AMDUCA).

†Route of administration: Administration of these corticosteroids by other routes of administration may affect the length of time it takes before the medication is below the regulatory threshold (eg, betamethasone injected intramuscularly takes approximately 33 days for its plasma concentration to fall below the regulatory threshold). The withdrawal times for betamethasone, methylprednisolone, and triamcinolone acetonide apply to the intra-articular route of administration only and should not be interpreted as guidance for withdrawal of these substances after other routes of administration.

‡Administration dosage and related 95/95 withdrawal times: This is provided as guidance and does not constitute a guarantee. It does not account for repeat dosage, multiple articular site dosage, the effects of combining other medications with these corticosteroids, or different dosages from those listed here. A risk assessment must be done by the veterinarian and trainer to determine protocol that complies with the prohibition against administration of an intra-articular dose within 7 days of racing.

§Please note that for this dose of methylprednisolone acetate, a 21-day period from administration to racing is recommended to ensure that the medication is below the regulatory threshold. The minimum withdrawal time in this case anticipates the possibility of lower doses being used and assumes that these lower doses may decrease the length of time required for the concentration of the medication to fall below the regulatory threshold.

and OA in the horse. Other countries have traveled down this pathway some time ago. More clear restrictions on the use of medication should be considered in the positive light that at the same time, there has been validation of non-corticosteroid treatments even in the acute stage of joint inflammation and newer biological therapies have emerged that have no side effects and offer positive advances.

2. Intra-Articular Corticosteroids: What Has Changed?

As discussed above, the RMTC issued a 7-day limitation for long-acting corticosteroids so that they would fall below their respective threshold levels regarding a series of experimental studies with intra-articular therapy.

After review of the data and addition of a tolerance interval for safety, the RMTC was able to calculate a recommended withdrawal time with the experimental dose of 100 mg of intra-articular methylprednisolone acetate (MPA) in one articular space, on the basis of a 95/95 tolerance interval, requiring approximately 16.6 days for the concentration of MPA in plasma or serum to fall below the 100 pg/mL threshold. This was rounded out to 21 days. In February 2012, Mid-Atlantic regulators, horsemen, and industry representatives met to discuss the implementation of numerous medication guidelines,

including the corticosteroid recommendations, on a regional uniformed basis. Numerous regulators and horsemen expressed concerns regarding the disparity between the 7-day ban on intra-articular use of MPA and the 21-day recommendation for the experimental dose to comply with the proposed regulatory threshold. There was a feeling from veterinarians as well that compliance with the recommendations would be difficult and could place horsemen and veterinarians attempting to comply with the recommendations in good faith in "harm's way" and that the better approach may be to ban the drug and recommended use of alternative therapies. To continue the principal goal of the RMTC in having regulatory thresholds adopted and uniformity attained, a decision was made to remove methylprednisolone from the list of approved therapeutics.

3. Working Within the 7-Day Rule

As discussed in an earlier review,[1] controlled studies have been performed in the horse to clarify therapeutic response as well as deleterious effects for betamethasone esters,[a] MPA,[b] and triamcinolone acetonide[c] (TA). In a survey of AAEP members, a lower number of respondents were using MPA in high-motion joints (25.7%), with most (77.3%) opting for TA instead.[2] A higher percentage (72.7%) were still using MPA in low-motion joints. In contrast to the published works documenting the negative effects of MPA, most respondents indicated that scientific papers and data determined their choice of corticosteroid use. The use of Depo-Medrol[b] has decreased since demonstration of negative effects on the cartilage, but there is still considerable use. It is expected that the pragmatics of prolonged withdrawal time will further decrease the use of the drug. In addition to the options of betamethasone esters and TA as well as isoflupredone acetate,[d] there are other medication options that are also presented below. A window into the future can easily be examined by looking at racetrack practitioners' use in other countries. In the author's native country of New Zealand, there is currently a 48-day withholding period for Depo-Medrol; personal communications indicate that there is no use of that drug in racehorses and that it is not missed.[e] Racetrack practice has emerged with considerable TA use (withdrawal period is 4 to 5 days) and an increasing use of interleukin-1 receptor antagonist protein therapy in carpal, fetlock, and distal tarsal joints.

4. Hyaluronan

Combination therapy of corticosteroids and hyaluronan (HA) is quite common and can be defended scientifically. In an experimental study of equine osteoarthritis induced by osteochondral fragmentation in the middle carpal joint, eight horses received 20 mg of HA[f] intra-articularly 14, 21, and 28 days after induction of OA.[3] No adverse treatment-related events were detected. No changes in clinical signs were seen with HA compared with that in control horses, but, histologically at day 70, there was significantly less fibrillation with HA treatment compared with that in controls. The place for HA in combination therapy is therefore not as an acute anti-inflammatory but as a long-term disease-modifying osteoarthritis drug (DMOAD).

5. Polysulfated Glycosaminoglycan

Work examining the effect of intra-articular polysulfated glycosaminoglycan (PSGAG)[g] suggests that this product has significant effects on acute synovitis and can be used in lieu of intra-articular corticosteroids.[4] In a controlled study with the use of the equine osteochondral fragment–OA model, eight horses received PSGAG (250 mg) and amikacin sulfate (125 mg) intra-articularly at 14, 21, and 28 days after induction of OA, and eight control horses received 2 mL of saline (0.9% NaCl) solution and amikacin sulfate (125 mg) intra-articularly on study days 14, 21, and 28. The degree of synovial membrane vascularity and subintimal fibrosis was significantly reduced with PSGAG treatment, and there was a trend for reduced fibrillation at day 70. This indicated significant symptom-modifying OA effects as well as potential DMOAD effects. In other unpublished work, however, it was shown that the combination of triamcinolone acetonide and PSGAG was inferior to either triamcinolone acetonide alone or PSGAG alone and so current indications are that PSGAG, if used intra-articularly, should be used alone and not in combination with corticosteroids.

6. Extracorporeal Shock Wave Therapy

Evaluation of extracorporeal shock wave therapy (ESWT) in the osteochondral fragment model of OA also showed symptom-modifying effects in the carpal joint.[5] Extracorporeal shock wave therapy has also been used to treat horses with OA.[6,7] Horses were randomly allocated to receive local application of ESWT at days 14 and 28, a positive control therapy (intramuscular PSGAG), or sham ESWT (placebo). The degree of lameness in horses treated with ESWT improved significantly compared with the degree of lameness in placebo-treated or PSGAG-treated horses. No disease-modifying effects were evident in the results for synovial fluid, synovial membrane, or cartilage from the ESWT-treated or PSGAG-treated horses. There were no significant effects of ESWT on any bone variable, but serum osteocalcin concentration was significantly greater than in horses that received ESWT compared with placebo-treated control horses. The increase of serum biomarkers was indicative of bone remodeling, but there were no negative effects revealed. It could be concluded that ESWT is a viable non-pharmaceutical treatment for acute inflammation in equine joints.

7. Underwater Treadmilling

Aquatic therapy has become increasingly popular in its use for rehabilitation of equine musculoskeletal injuries. The mechanism of action of aquatic therapy and its potential use in the clinical management of equine OA has been recently reviewed.[8] Recent human research has increased our understanding of neuromuscular responses to joint pain.[9] Joint mechanoreceptors are characterized as sensory receptors within periarticular tissues that respond to changes in joint position and motion and are also important in regulating neuromuscular control associated with joint stability.[10] Pain, inflammation, and joint effusion alter the normal sensory input from articular mechanoreceptors, which may cause motor neuron excitability and reduced muscle activation.[11] Experimentally induced knee effusion produced significant quadriceps muscle inhibition.[12–14] Joint instability alters the distribution of weight-bearing forces across articular surfaces and induces an increase in the recruitment of adjacent muscles to help aid in joint stability.[15] The resulting functional imbalance and paired agonist-antagonist muscle groups contributed to increased joint instability and altered limb biomechanics, which leads to further progression of OA and chronic maladaptive compensatory mechanisms.[16] There is an increasing perception among veterinarians that joint injuries may recur or be exacerbated as the result of muscle weakness, reduced joint range of motion, and poor proprioception, as exemplified by immobilization of the equine metacarpophalangeal joint. The entire musculoskeletal system must be rehabilitated to return the horse to optimal performance.[8] Recently, the use of underwater treadmilling has been assessed in the equine OA model, and both decrease in osteoarthritis as well as improved proprioception were demonstrated.[h]

This is a clear demonstration that underwater treadmilling can be used as an adjunctive device for the treatment of traumatic joint inflammation, and there is no indication that it could not be used with a racehorse in full training.

8. Autologous Conditioned Serum (IRAP or IRAP II)

Autologous conditioned serum (ACS) was initially developed for the treatment of human OA as a product called Orthokine[i] and was initially tested in horses in Europe and shown to be particularly beneficial in OA (of the distal interphalangeal joint) not responding to triamcinolone and HA.[j] Orthokine was subsequently distributed in the United States as IRAP[k] and in an experimental study[17] was shown to provide benefit in osteochondral fragment–induced equine OA. Horses received 6 mL of ACS at 14, 21, 28, and 38 days after treatment (control horses received 6 mL of saline intra-articularly at the same time). Horses treated with ACS were observed to have significantly reduced lameness in the OA limbs even 5 weeks after the last treatment compared with placebo-treated horses. There was also a significant reduction in synovial membrane hyperplasia in treated compared with placebo OA joints at day 70 and a trend for improvement ($P < 0.10$) in cartilage gross score and cartilage histochemistry noted in ACS-treated OA joints compared with placebo-treated OA joints. The hypothesis that the main effect of this new therapy was significant increases in interleukin (IL)-1 receptor antagonist protein (IL-1ra) was confirmed, with increases seen at days 35 and 71.

A newer product, IRAP II,[l] with a modified technique including a newly designed device with dual ports, has since been produced, and a comparative study was performed on the cytokine profiles of IRAP and IRAP II with the use of equine blood.[18]

Specifically, the level of IL-1ra in IRAP II was significantly increased compared with IRAP, and the ratio of IL-1ra to IL-1β also significantly increased in IRAP II compared with IRAP. On the other hand, there was a significantly increased level of tumor necrosis factor-α in IRAP compared with IRAP II but no significant difference in IL-1β levels. Production of insulin-like growth factor (IGF)-1 and transforming growth factor-β was both significantly increased over serum alone, and no significant difference between the two products was seen. On the basis of the extrapolation of doses used in this study, the recommendation for IRAP after arthroscopic surgery is 6 mL in knees, fetlocks, distal interphalangeal joints, and tendon sheaths but 10 to 12 mL in femoropatellar or femorotibial joints. Treatment in the latter joints would therefore require two preparations (two preparations of IRAP or IRAP II to give three injections at this dose rate).

The use of the IRAP products has considerably increased in racing jurisdictions outside the United States. They represent a specific biological therapy without negative side effects and have been well accepted by racing jurisdictions other than in Scandinavia, where there have been attempts to ban their use. There is no scientific validity to excluding such products from use in the competing athlete. In the 2009 joint therapy usage survey, sport horse practitioners were significantly more likely than racehorse or show horse veterinarians to use IRAP products ($P = 0.0035$ and $P = 0.04$, respectively), but this was in the United States. The author suggests that the use of IRAP in racehorses is used more often by racetrack practitioners outside of the United States.

9. Platelet-Rich Plasma

Platelet-rich plasma (PRP) has been advocated as a way to introduce increased concentrations of growth factors and other bioactive molecules to injured tissues in an attempt to optimize the local environment. There are various definitions of PRP, but the consensus now is that the product should have an increase in platelet content over the level in blood. The initial enthusiasm for PRP was based on growth factors within the α-granules including transform-

ing growth factor-β, platelet-derived growth factor-1, and IGF-2, fibroblast growth factor, epidermal growth factor, as well as endothelial growth factor. There are a number of other bioactive factors in PRP contained in dense granules of platelets,[19] and there is an emerging paradigm that more than just platelets are playing a role in PRP.[20]

The use of PRP to treat joint disease is currently increasing in the horse. Up until this time, the author has tended to recommend IRAP rather than PRP for treatment of joints. However, good clinical results with the use of PRP to treat OA in people have been reported,[21] and recent in vitro work showed beneficial effects on cartilage metabolism.[22] More recently, a comparison between HA and PRP intra-articularly in the treatment of OA in the knee showed that local injection of a low-platelet-count PRP product[m] had a significant effect shortly after the final infiltration and a continuously improved sustained effect up to 24 weeks Western Ontario and McMaster Universities Arthritis Index (WOMAC scores 65.1 and 36.5 in the HA and PRP groups, respectively, $P < 0.001$), in which the clinical outcomes were better as compared with HA-treated horses.[23] Also in the HA group, the worst results were obtained for grade III gonarthrosis, whereas the clinical results obtained in the PRP group did not show a statistically significant difference with regard to the grade of gonarthrosis. The mean WOMAC scores for grade III gonarthrosis were 74.85 in the HA group and 41.20 in the PRP group ($P < 0.001$).

Along with this study, recent work has suggested that whereas higher numbers of platelets are seen in some PRP products, this brings higher white cell count levels and higher catabolic cytokine levels.[24] In another study looking at the anabolic and catabolic activities of cartilage and meniscal explants in vitro and the effect of a single-spin PRP[m] compared with a double-spin product,[n] ADAMTS-4 gene expression was lowest for the single-spin PRP (ADAMTS-4 or aggrecanase 1 is considered a major factor in articular cartilage degradation). Also, radiolabel incorporation with 35-sulfate (an index of glycosaminoglycan synthesis) and 3H-proline incorporation (as an indication of collagen synthesis) were significantly enhanced with the single-spin system. Therefore, there is growing scientific support for the possible use of PRP after surgery, but we still need good clinical evidence in the horse.

At this stage, the use of PRP as a treatment for acute synovitis and capsulitis or early OA cannot be recommended.

10. Mesenchymal Stem Cells

The use of mesenchymal stem cells (MSCs) or mesenchymal stromal cells has become popular as an adjunctive therapy in equine orthopedics. Most work has concentrated on the multipotent cells present in adult bone marrow that can replicate an undifferentiated cell and have the potential to differentiate to lineages of mesenchymal tissue including bone, cartilage, muscle, ligament, tendon, adipose, and stroma.[25] Multiple different pathways of multipotent MSCs and the proteins involved in their transcriptional control have been described in a review of MSC therapy in equine musculoskeletal disease.[26] The clinical use of MSCs in horses, justification for their use, and issues surrounding their use have been reviewed.[27] Early work with labeled MSCs has shown that they have an affinity for damaged joint tissue, and more recent work has confirmed their ability to localize and participate in the repair of damaged joint structures including cruciate ligaments, menisci, and cartilage lesions.[28]

Most in vivo studies performed in animals other than horses are focused on meniscal repair. A particularly significant study involved direct intra-articular injection with beneficial effects to meniscus and secondary OA.[29] This led to the initiation of a clinical study with intra-articular bone marrow–derived mesenchymal stem cells (BMSCs) plus HA therapy in clinical cases of femorotibial joint trauma. Meniscal injury (and other soft-tissue injury) with secondary OA is a common problem in our population of horses.[30] It has also recently been demonstrated that experimental equine meniscal lacerations can be healed with equine BMSCs in fibrin glue and show increased vascularization, decreased thickness in repair, and increased total bonding.[31] We also examined the use of BMSCs in the equine osteochondral fragment model.[32] In a comparative study, it was shown that there was significant improvement in synovial fluid prostaglandin E (PGE)2 levels with BMSC treatment and nominal improvement in symptom- and disease-modifying effects. On the other hand, there was an interesting negative response with adipose-derived stromal vascular fraction cells in that there was an increase in synovial fluid tumor necrosis factor-α levels and no significant change in PGE2 levels. The anti-inflammatory effect was another good example of trophic effects with MSCs. The term "trophic effects" applies to the observation that in addition to adult marrow-derived MSCs being capable of dividing and their progeny further differentiating into one of several mesenchymal phenotypes such as osteoblasts and chondrocytes, these MSCs also secrete a variety of cytokines and growth factors that have both paracrine and autocrine activities.[33] These secreted bioactive factors suppress the local immune response, inhibit fibrosis (scar formation) and apoptosis, enhance angiogenesis, and stimulate mitosis and differentiation of tissue-intrinsic reparative or stem cells.

The trophic effects of BMSCs have been further demonstrated by a recent study in the horse in which an intra-articular injection of 20 million BMSCs in 20 mg of HA[f] was compared with HA alone in the repair of full-thickness microfractured defects on the medial femoral condyle.[34] There was enhancement of the firmness of the repair tissue at 6 and 12

months as well as a significant increase in aggrecan content in the repair tissue.

As a potential alternative for the use of acute traumatic synovitis and capsulitis, MSCs would not appear to be an option. There are good indications that MSCs have long-term effects for soft tissue inflammation within the joint as well as articular cartilage repair. The amount of DMOAD effects is less certain; their use is important in long-term regenerative therapies but not in the acute situation.

11. Systemic (Parenteral) Medications

Polysulfated Glycosaminoglycan

Polysulfated glycosaminoglycan administered intramuscularly is still very popular, but evidence is limited to anecdotal opinion. Intramuscular injections of 500 mg after surgery were commonly recommended by the author until a controlled study in experimentally induced equine OA showed no significant difference in the administration of intramuscular PSGAG (every 4 days for 28 days) as a positive control treatment in an evaluation of ESWT on experimentally induced osteoarthritis in the middle carpal joints of horses.[5] Consequently, it is thought that the use of intramuscular PSGAG is very limited as a treatment. Any prophylactic value is still unproven.

Pentosan Polysulfate

Intramuscularly administered sodium pentosan polysulfate (NaPPS) has been commonly used outside the United States for the treatment of osteoarthritis as well as after surgery. Recently, NaPPS at a dose of 3 mg/kg intramuscularly at 15, 22, 29, and 36 days after induction of experimental carpal osteoarthritis caused a significant reduction in articular cartilage fibrillation and an increase in chondroitin sulfate 846 epitope (a synthetic biomarker) in the synovial fluid of both osteoarthritic and non-osteoarthritic joints. This indicated that NaPPS has some beneficial disease-modifying effects.[35] The product is available in a 12-mL vial[o] (which is the approximate dose for a 1000-lb horse), but caution must be exercised in the United States because the product is not licensed and is only available as an intra-articular lavage.

Intravenous HA

The use of intravenous HA (40 mg IV) after surgery is used by some clinicians, and there is scientific evidence that would support its use. With a study in the equine osteochondral fragment–OA model, three treatments of 40 mg were given intravenously 13, 20, and 27 days after osteochondral fragment creation. There was a significant decrease in lameness, synovial fluid protein and PGE2 levels, synovial membrane vascularity, and synovial membrane cellular infiltration at day 72 compared with the control group given 4 mL of saline intravenously at those same times.[36]

Oral HA

A number of oral nutraceuticals are used after arthroscopic surgery. All these products are unlicensed, and, with the exception of oral HA,[p] no scientific evidence has been produced to support their use. However, one oral HA product[p] has been shown to benefit postoperative effusion after arthroscopic surgery for tarsocrural osteochondritis dissecans (OCD).[37] Oral HA (100 mg) was given daily for 30 days after surgery in 24 yearlings (with 27 joints operated), and another 24 yearlings (30 joints operated) were treated with placebo daily for 30 days. An examiner blinded to the treatment groups scored the effusion at 30 days on a scale of 0 to 5. The mean 30-day effusion score in the treated group was 0.67 as compared with 2.05 in the placebo group ($P < 0.0001$).

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[b]Depo-Medrol, Pharmacia and Upjohn Co, Kalamazoo, MI 49001.
[c]Vetalog, Bristol Myers Squibb for Fort Dodge, Fort Dodge, IA 50501.
[d]Predef 2X, Pharmacia and Upjohn Co, Kalamazoo, MI 49001.
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[f]Hyvisc, Boehringer Ingelheim Corp, Ridgefield, CT 06877.
[g]Adequan IA, Luitpold Pharmaceuticals Inc, Animal Health Division, Shirley, NY 11967.
[h]*The Role of Aquatic Therapy in Equine Osteoarthritis.* M. King, PhD Thesis, 2012.
[i]Orthokine, Orthogen Lab Services GmbH, Düsseldorf, Germany.
[j]Weinberger, personal communication, 2003.
[k]IRAP, Arthrex (now Dechra Veterinary Products), Overland Park, KS 66204.
[l]IRAP II, Arthrex Vet Systems, Naples, FL 34108.
[m]Arthrex ACP System, Arthrex Vet Systems, Naples, FL 34108.
[n]Harvest Technologies SmartPReP 2, Harvest Technologies Corp, Plymouth, MA 02360.
[o]PentAussie, Matrix Animal Health LLC, Summerville, SC 29484.
[p]Conquer, Kinetic Technologies, Lexington, KY 40509.

The Thoroughbred Racehorse Foot: Evaluation and Management of Common Problems

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1. Introduction

The importance of foot shape and balance in high-performance racehorses is paramount to maintaining soundness and optimal performance. Functionally adapted for speed and efficient use of energy, the Thoroughbred foot is light and lacks the mass for protection commonly seen in other heavier-boned breeds. The relatively thin walls and soles of the Thoroughbred foot make it more susceptible to injury and hoof capsule distortion. Hoof capsule distortion refers to hoof abnormalities such as flares, cracks, under-run, collapsed, and sheared heels—all of which result from long-term abnormal weight distribution on the foot. Distortions affect function and have been correlated to musculoskeletal injuries and lameness.[1–3]

The racehorse practitioner is often presented with an acute or chronic foot problem to manage. Having knowledge of the etiology of the more common foot problem will help formulate a successful treatment plan to heal the acute condition, return the horse back to soundness, and prevent reoccurrence. Without the proper knowledge of the entire problem, most foot problems are quickly fixed and patched up, and the underlying hoof capsule distortion is never effectively addressed. Therefore, it is likely to reoccur.

Balance is the term used when describing the shape, angle, and spatial arrangement of anatomical structures of the foot. Learning how to evaluate a foot and detect imbalance or overloaded regions of a foot are important for formulating a treatment plan. Although some properties of balance will vary with limb conformation and are subjective, it is generally accepted[4,5] that balanced Thoroughbred feet possess the following characteristics:

- Even distal interphalangeal (DIP) joint space on anterior-posterior standing radiograph
- Straight hoof pastern axis
- Center of the DIP joint or widest part of foot should be located in the center of the weight-bearing surface of the shoe in the sagittal plane
- Palmar angle of pedal bone should be between 2° to 5°
- Heel position should be located at the widest part of the frog
- Heel angle should be within 5° of toe angle
- Solar surface of foot perpendicular to long axis of pastern bone in the sagittal plane
- Even hoof wall growth from all regions of the coronary band

NOTES



Fig. 1. Common racehorse foot with negative palmar angle and thin sole depth at heel.



Fig. 2. Properly glued shoe; note that there is very little glue on the sole, and the frog sulci are not obstructed.

The typical Thoroughbred conformation of a longer, more sloping pastern places more force on the heel region.[6] Repetitive speed training in racehorses decreases hoof angle over time,[7] and, as hoof angle decreases, more stress is placed on the heel region.[6] Therefore, low hoof angles and increased stress on the heel region can become a self-perpetuating cycle if proper intervention is not implemented to reverse the downward trend of the foot (Fig. 1).

The heel region is usually the first part of the foot to display a distortion because it is generally made up of softer, more compliant structures than is the toe. Hoof capsule distortions occur slowly over time and are the result of long-term abnormal weight-bearing.

The horse's foot is capable of handling huge impact forces without structurally collapsing. This is because when a horse is traveling, the moving foot fills with blood during the swing phase, probably from centrifugal force and creating turgor pressure. This fluid in closed spaces may help support the architecture of the foot during ground impact, thus allowing the foot to withstand high impact forces.[8] Most hoof capsule deformities (under-run, collapsed heels) slowly develop over time. The author believes that most of these distortions occur while the foot is semi-static (while the horse is just standing around). Racehorses spend 22+ hours a day standing in a straw-bedded stall. It is during this period that the foot is mostly dependent on the architecture of the foot tissues for support. Long-term, low-magnitude loading creates distortion rather than short-term, high-magnitude force. Horses standing in a stall with little arch/sole support slowly fatigue the integrity of the capsule and propagate distortions. The arch of the sole slowly flattens, the heels become under-run, and perhaps a heel bulb becomes sheared or shunted proximally. The insidious nature of a hoof capsule distortion slowly compromises the foot, rendering it more susceptible to an acute injury.

2. Heel Pain in the Front Limb

In the author's experience, the most common site of heel pain in the racehorse is pain in the medial heel region of the front feet. Pain in this region can originate from a variety of sources: bruising of the sensitive sole, osteitis of the wing of the coffin bone (pedal osteitis), submural pain from sensitive laminae/wall separations, sheared or shunted heels, and quarter/heel cracks. Pain in the medial heel is so prevalent that rarely does the author fail to find sensitivity over the medial heel/bar region on a routine hoof tester exam of a racehorse. The etiology of heel pain in the racehorse is multifactorial; conformation, farriery, track conditions, and management all play major roles. Even though common causes of heel pain can be seen simultaneously as a syndrome, this paper will discuss them separately.

3. Heel Bruise/Stone Bruise

A common reason for a racehorse to be scratched from a race or given time off is for a stone bruise or bruised foot. The author finds this cause of lameness has always been surprising. Racehorses generally are housed, hot-walked, trained, and raced on fairly good footing, with little chance for a stone or hard terrain to directly bruise the foot. Therefore, the term "stone bruise," in most cases, is a misnomer. Bruising of the foot can be caused in three different ways: (1) sole pressure from the shoe or glue. This can be from a shoe that was fitted too small or too tight, creating sole pressure, or from a shoe that has been left on too long, allowing the heels to overgrow or expand over the shoe creating sole pressure. (2) Gluing shoes with acrylic has become common practice in racehorses with brittle, poor-quality walls. Care must be taken in using very little glue and not allowing the glue to set up on the pliable solar surface. The acrylic, once set up, can become very firm, creating sole pressure or pressure on the soft solar heel bulbs. If the glue is allowed too high up the wall near the heel bulb, then, during speed training when the heel bulb compresses, the top edge of the glue can pinch the heel bulbs, creating soreness (Fig. 2).

The third cause of a heel bruise is an excessive heel check on the shoe. That is, the branches of the



Fig. 3. A, Properly fitted race plate, with heel checks and unobstructed frog sulci. B, Race plate fitted too tightly in the heels, with no heel checks. The frog sulci are obstructed and likely to trap dirt, causing bruising.

shoe wrap around too tightly, covering up the medial and lateral sulci of the frog and don't allow clearance of the footing through the sulci. The branch of the shoe will allow footing to ball up and become tightly compacted as it is forced into the sulci at high speeds during the sliding phase of impact. Rapid deceleration of the foot will drive the track substrate into the sulci like a wedge, creating bruising. This is

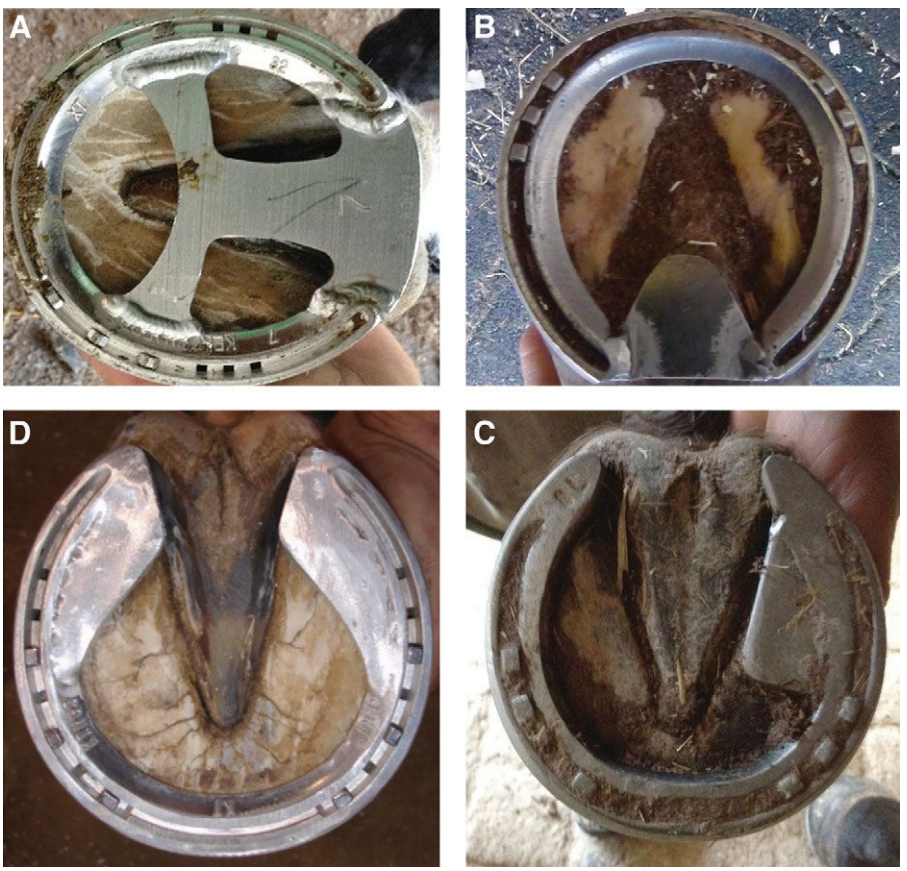


Fig. 4. A, Shoe with a stabilizer pad welded in for support and protection. B, Heartbar welded into a steel training plate. C, Onion heel shoes, used to protect the bars. D, Unilateral onion heel, used to protect the wing of the pedal bone and bar.

probably the most common cause of medial heel bruising (Fig. 3). Repetitive bruising of the sole or an acute severe bruise can cause inflammation of the bone (pedal osteitis). Pedal osteitis is most successfully diagnosed by nuclear scintigraphy or magnetic resonance imaging, which can show active inflammation and edema. Less accurate diagnostics are radiographs that may show the chronic change of demineralization and loss of the normal, smooth contour of the solar border of the pedal bone (Fig. 4). This, along with positive response to hoof testers and diagnostic analgesia, is probably sufficient to make a diagnosis in a young horse; however, it is questionable how accurate the radiographic changes are without the aid of diagnostics that show the physiology of acute inflammation.[9]

Pedal osteitis can be seen more commonly in the heel region of a low-heeled foot, but it can also be seen in the toe region, particularly in upright or club-footed horses. Severe trauma to the margins of the pedal bone can cause marginal rim fractures. These cases are typically severely acutely lame and then improve over a few days with stall rest. These horses require at least 60 to 90 days of rest to heal before being gradually reintroduced back into training. Shoeing is for protection, shock absorption, and ease of the breakover, and to decrease tension on anterior laminae against the bone fragments in the toe region. A wide web shoe, or a shoe and pad, with a rolled/rockered toe are indicated. For pedal osteitis of the palmar/plantar process, a shoe with protection of the heel quarters is indicated. Shoe with pad, stabilizer plate, onion heel shoe, or bar shoe are indicated. Once the inflammation has resolved, most cases will require special shoeing when they go back into training because reoccurrence is very common. Training in an onion heel shoe, bar shoe, or spider plate is usually effective.

4. Shunted Heel/Sheared Heel

By far the most frustrating hoof problem in the racehorse is the reoccurring quarter crack. Often the author hears about a quarter crack hampering the ability of a Triple Crown or other high-profile race contender. Quarter cracks are almost always preceded by a shunted or sheared heel hoof capsule distortion (Fig. 5). The quarter or heel crack is an acute episode that results from the more insidious, slowly developing condition of the sheared/shunted heel. A shunted or sheared heel is when one heel bulb is displaced proximally. This condition is most commonly the result of long-term overloading of the medial heel quarter. This condition has been described in horses with a base narrow, outward rotational limb deformity; creating the ground reaction force to shift medially during the landing/support phase. These cases typically strike on the lateral aspect of the foot and then load medially, However, in the author's experience, sheared heels are seen more commonly in racehorses without outward rotation of the limb. Horses with severe outward rotation usually interfere, which commonly affects performance. Sheared heels in upper-level racehorses are more commonly seen in horses that are slightly carpal valgus and have an inward rota-



Fig. 5. Sheared medial heel.



Fig. 6. Outward rotation of the front limbs. Note the white hairs on the inside of the right front fetlock from interference.

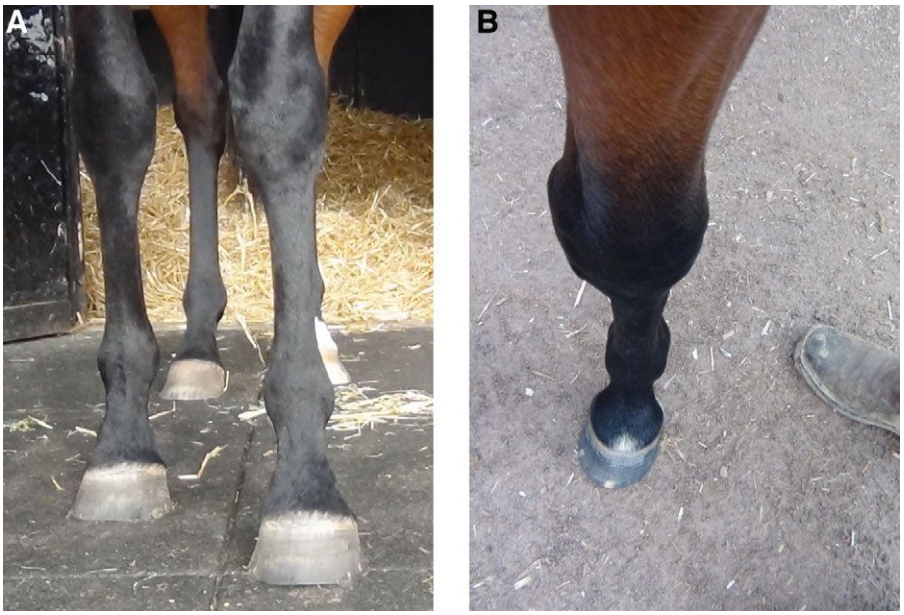


Fig. 7. A, Front view of the left front limb with slight carpal valgus and inward rotation of the pastern. B, Left front limb with slight carpal valgus and inward rotation of the distal cannon bone and pastern.

tion of the distal limb (emanating from the distal metacarpus or pastern). Carpal valgus conformation has been shown to shift the center of pressure medially,[10] and the inward rotation of the distal limb turns the hoof like a dial, putting the medial heel more in line with the center of force, or directly beneath the cannon bone (Fig. 6). This combination of conformation faults is very common in high-level, successful racehorses. However, it puts increased compressive forces on the medial heel, shunting or displacing it proximally. The increased compression on this region of the foot slows wall and sole growth medially, causing the foot to easily grow out of balance between shoeing cycles. It is very common for these feet to be high on the lateral side and low medially when viewed from the solar surface (Fig. 7). Radiographs taken before trimming and shoeing typically show the coffin bone low medially, especially when evaluated several weeks after shoeing and trimming.

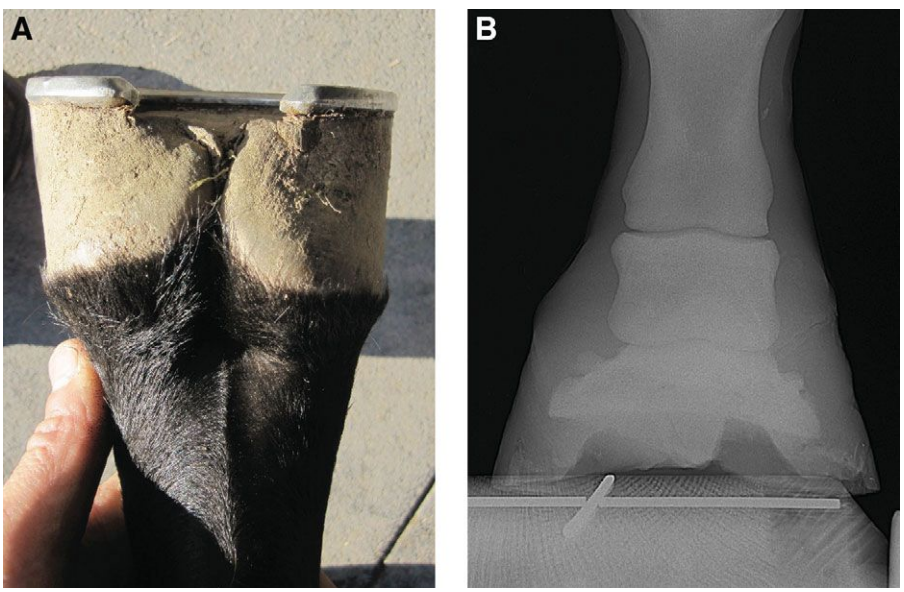


Fig. 8. A, Medial sheared heel that is also out of balance and high laterally. B, Radiograph of a medial sheared heel case that is high laterally.



Fig. 9. A, Case with slight carpal varus and significant inward rotation of the pastern on the right front. B, Same case, viewed from the front; note the offset appearance of the hoof and pastern. C, Radiograph of the same right front foot.

Since 2010, the author has examined 72 sheared heels on Thoroughbred racehorses and recorded the conformation of the limb, with photographs taken directly dorsal to the carpus of each leg affected. Of the 72 sheared heels on the front feet, 70 sheared heels were medial and two were lateral. Both cases with laterally sheared heels were fetlock varus. Of the 70 medially sheared heels, 60 had a combination of carpal valgus and inward rotation of the distal limb, eight had outward rotation of the limb, one had fetlock valgus, and one had normal conformation (no major conformation fault). Therefore, conformation is very likely to be involved in the development of a sheared heel.

In the hind end, it is more common to see sheared heels and subsequent quarter crack in the lateral heel. This is more common in horses that are base narrow behind and/or fetlock varus in the hind end. It appears that sheared heels and quarter cracks are most common in the better horses, as higher speed increases ground reaction forces on the foot (the better horses strike the ground harder).[11]

Management of the sheared heel is key in maintaining soundness and preventing the occurrence of quarter cracks. Patching the quarter crack has been described in detail elsewhere and is not covered in this report. Keeping the foot trimmed and balanced is fundamental. Trimming and shoeing on a shorter 3-week schedule may be necessary to prevent severe imbalances. The sole surface of the foot is typically divided into quadrants or quarters (medial/lateral toe quarters, medial/lateral heel quarters). If the toe (quarters) are left long, more force is placed on the heel quarters. If the lateral toe and heel quarters are left high, the medial half of the foot is loaded. Although studies show that artificially elevating the lateral aspect of the hoof shifted the center of pressure laterally, it is unknown if this holds true in cases that naturally grow faster laterally and grow out of balance. In practice, the author sees more signs of compression and damage on the medial side as the lateral wall grows higher and the foot becomes more imbalanced. Therefore, to unload the medial quarter, the author trims the toes as short as possible and lowers the lateral heel so that the solar surface of the foot is perpendicular to the long axis of the pastern. It is important to fit with a shoe that is centered on the coffin joint, so that the widest part of the foot is in the center of the shoe and the heel of the shoe is at least at the widest part of the frog. Too much length of the toe combined with a medial-lateral imbalance can overload the medial quarter. Because the sheared heel results from overloading and displacement of the hoof



Fig. 10. Foot trimmed and shod perpendicular to the long axis of the pastern.



Fig. 11. A, The two-part elastomer is mixed and applied to the sole surface of the foot. B, It is then wrapped onto the foot, and the horse stands on a flat surface until elastomer sets up. C and D, The orthotic is removed and trimmed up so that it can easily be inserted and removed daily.

wall/heel bulb, methods to unload and allow the wall to drop back down are necessary for successful management. This can be performed most effectively with the use of a heartbar, bar shoe, or stabilizer plate, which transfers weight onto the base of the frog (Fig. 8). The wall beneath the sheared heel can then be floated off the shoe so that the displaced wall can drop down. Many trainers do not like the added weight and decreased traction created with a bar shoe; therefore this shoeing prescription sometimes can have poor compliance. One option is to train in the bar shoe and switch to a normal race plate on race day or shortly before. Another option used in the author's practice with significant success is the use of temporary orthotics. This is when a two-part elastomer dental impression putty[a] is used to make a custom orthotic or arch support for each foot. The removable orthotics are placed into the feet and wrapped in place with Vetwrap[b] (Fig. 9). The orthotics can be removed for training and placed back in the foot when the horse is in the stall. The orthotics provide arch support and help unload the perimeter wall to strengthen the arch and allow the shunted wall to drop. Bar shoes or temporary orthotics, combined with shortened balanced trimming intervals, have significantly decreased the occurrence of quarter cracks and improved the sheared heels in the author's practice. Although not always feasible, letting these cases go barefoot when possible is very effective in improving the sheared heel. Often the medial heel sole depth is so thin that these cases need shoes to resume training.

5. Pedal Bone Fractures

Secondary to weak heel structures and high loads placed on the heels is trauma to the coffin bone, including fracture of the wing. Wing fractures are the most common pedal bone fracture the author sees in the Thoroughbred racehorse. It has been reported that wing fractures are more common laterally in the left front and medially in the right front.[2,6] However, in the author's experience, lateral wing fractures are more commonly seen in the right and left front feet. This could be because most horses land lateral heel first at higher speeds, and fractures of the wing may occur during initial impact. Clinical signs are acute severe lameness. Radiographs taken usually show the fracture line immediately. Occasionally, the fracture line is not evident until there is demineralization and lysis at

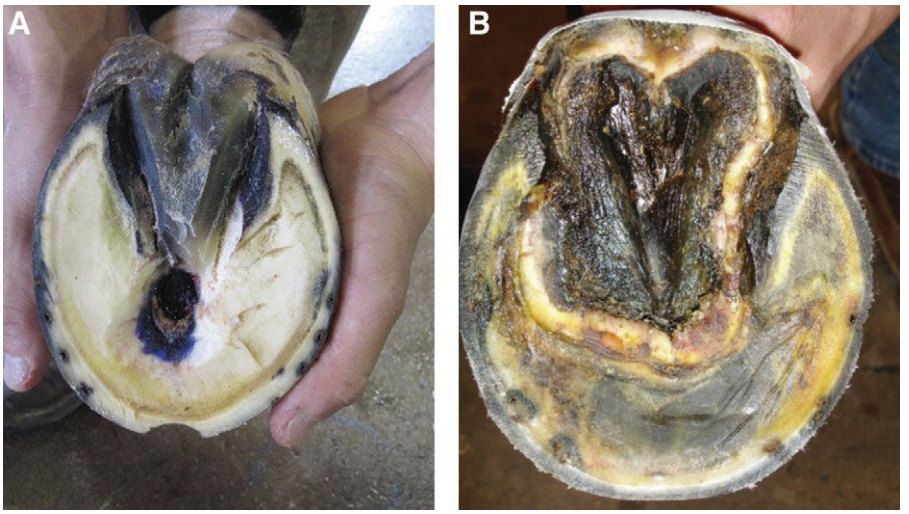


Fig. 12. A, Chemical burn to the sole region. This burn extended down to the solar surface of the pedal bone and required surgical debridement to heal. B, Chemical burn to the frog, which involved the digital cushion, deep digital flexor tendon, and navicular bone. This horse was euthanized.

the margins of the fracture. If a diagnosis is not evident on initial radiographs and a fracture is suspected, then follow-up radiographs are usually repeated in 3 to 5 days. Most wing fractures require 6 to 8 months to heal. Treatment consists of bar shoe with sole support or a wall cast. Most cases are stall rested for 2 to 3 months and hand-walked until the fracture has healed. Some cases will not heal completely and may only heal with a fibrous union, in which a radiolucent line persists. Adequate stabilization with shoeing and stall rest will probably result in radiographic healing.

6. Subchondral Bone Trauma/Contusions to the Pedal Bone

Cases with contusion to the pedal bone or subchondral bone present similar to a fractured pedal bone. However, follow-up radiographs fail to reveal a diagnosis. Cases are usually diagnosed on magnetic resonance imaging or nuclear scintigraphy. Treatment is shoeing to absorb concussion and protect the solar surface and stall rest, combined with aspirin and isoxsuprine until digital pulses have returned to normal and the horse walks soundly. Some cases may resolve in days and some severe cases can take months.

7. Chemical Burns

Treating infections or exposed sensitive tissue in the foot is not in the scope of this lecture, but mismanagement and topical medication on the sensitive tissue of the foot is so prevalent on the racetrack that it should be mentioned here.

It is very common to see cases that have had sensitive tissue exposed by a subsolar abscess/infection or a quarter crack that has been treated at the racetrack with caustic materials in an effort to "harden" and heal over the defect. Most of the compounds are formulated for treating thrush and contain iodine and formalin and usually contain purple dyes to indicate the presence of medication. These compounds are made to treat superficial infections of the frog and have no place in treatment of exposed sensitive tissue or coria. Many quarter cracks are first treated with these substances to "dry them out" before patching. The iodines and formalin damage the sensitive submural tissues or corium/lamellar, creating scar tissue. This weakened, damaged tissue in the bed of the crack is probably part of the reason for the high prevalence of crack reoccurrence. Similarly, the author has seen numerous cases of exposed corium of the sole chemically burned (Fig. 10). Severe cases can involve the solar surface of the coffin bone and the deep digital flexor tendon, creating career-ending or life-threatening damage (Figs. 11 and 12). Client education in proper use and misuse of topical agents is imperative to prevent misuse of these compounds.[12–26]

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[a]Elastomer dental impression putty, NANRIC, Lawrenceburg, KY 40342.
[b]Vetwrap, 3M Animal Care Products, St Paul, MN 55144-1000.

How to Choose Your 'First Bag' of Intravenous Fluids

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1. Introduction

There are many intravenous (IV) fluid choices available to equine practitioners. Deciding which of these fluids to carry in inventory and in the truck will depend in part on the types of emergency cases most commonly seen in practice that require IV fluid intervention. Having information on hand regarding IV fluids most commonly chosen in different emergency settings will aid practitioners in making economical and practical decisions regarding which IV fluids to stock and carry. "First bag" choices for IV fluids may need to be made with limited clinical pathology data available but are generally aimed at restoring various fluid compartments within the body (Fig. 1). Follow-up, longer-term IV therapy is generally more directed and is based, at least in part, on recognized electrolyte and acid-base abnormalities. This presentation is aimed at aiding the practitioner in deciding what to administer initially, the first bag.

2. Materials and Methods

A brief e-mail survey was sent to veterinarians with specialty board certification in Large Animal Internal Medicine (via the ACVIM-LA list) and/or Emergency and Critical Care (by direct e-mail request). The survey presented nine distinct clinical situations in which intravenous fluid administration might rationally be part of the initial therapy:

- Ten-minute-old foal delivered from a prolonged dystocia (~120 minutes) with an APGAR score of 6
- Obtunded, hypothermic minimally responsive 24-hour-old foal
- Six-day-old foal with diarrhea
- Three-day postpartum mare presenting as mild to moderate colic with foal at side and in shock, heart rate 72; mucous membranes (mm) pale; CRT, 3 seconds; slow jugular fill; extremities cold
- Two-year-old Thoroughbred filly with a head injury from falling backward on its poll, no epistaxis
- Four-year-old Quarter Horse with frequent pipestream diarrhea, depressed, approximately 8% dehydration, has been on antibiotics, mucous membranes injected, heart rate 80
- Ten-year-old Arabian mare, badly tied up after an apparently energetic trail ride, not making urine
- Twenty-two–year-old Morgan horse with mild colic and palpable pelvic flexure impaction
- Eight-year-old Thoroughbred broodmare 338

NOTES

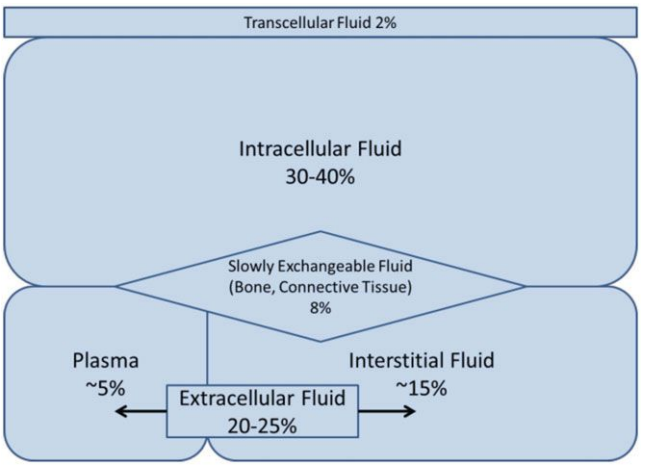


Fig. 1. Distribution of total body water as a percentage of body weight throughout the various compartments of the body in a juvenile patient. In adults, total body water is lower and interstitial percentage is lower. In true neonates, total body water is greater and extracellular percentage is greater.

days in foal, presenting with severe colic and abdominal distention

The survey purposefully did not provide information regarding clinical pathology or clinical chemistry results, nor were blood gas results included to more accurately represent an ambulatory situation in which such testing and results might not be immediately available. Detailed physical examination findings were also not included because this information is not initially available when choosing which fluids to carry to any call. The survey asked a single question: "What do you use and/or add to your 'first bag'?"

3. Results

Veterinarians from the United States, England, Europe, and Australia, representing both specialties, replied. Approximately 20 equine-oriented veterinary specialists responded. The following is a summary of responses by case situation supplied:

Ten-Minute-Old Foal Delivered From a Prolonged Dystocia (~120 Minutes) With an APGAR Score of 6

Two veterinarians would not necessarily administer IV fluids at that point in the described case but would continue to observe. Most responding veterinarians described the use of some type of isotonic polyionic crystalloid replacement fluid with a normal strong ion difference, although one veterinarian listed 0.18% saline with 4% dextrose as the first choice. One veterinarian would use either 0.9% saline or polyionic crystalloid replacement fluid. Fluids specifically mentioned included lactated Ringer's solution[a], Hartmann solution[b], Plasmalyte[c], Plasmalyte 148[d], and Vetivex 18[e] (0.18% saline with 4% dextrose, a maintenance rather than replacement fluid) (Table 1). If the amount of fluid to be administered was mentioned, it was between 500 mL and 1 L. Supplements added included (in order of frequency mentioned): dextrose (1% to 5%), thiamine (1 g/L), vitamin C, dimethylsulfoxide (DMSO) (1% to 2%), and 50% $MgSO_4$ (25 mL).

Obtunded, Hypothermic, Minimally Responsive 24-Hour-Old Foal

All veterinarians chose to administer isotonic polyionic crystalloid replacement fluids at bolus rates. One chose to administer 200 mL of hypertonic saline before other fluids. Dextrose/glucose supplementation was suggested in 60% of responses and was supplied as a piggyback constant rate infusion (CRI) 5% dextrose solutions or as a 1% additive to the first crystalloid bag followed by a CRI at 4 mg/kg per minute. One veterinarian suggested hetastarch administration and another suggested calcium, if ionized calcium was low on blood gas. Thiamine was supplemented in the first bag by several veterinarians.

Table 1. Composition of Commonly Used Intravenous Fluids

	Na	Cl	K	Ca	Mg	Lactate	Acetone	Gluconate	Dextrose	Osm	
	mEq/L	mEq/L	mEq/L	mEq/L	mEq/L	mmol/L	mmol/L	mmol/L	%	mOsm	pH
5% Dextrose in water	–	–	–	–	–	–	–	–	5	252	4.0
10% Dextrose in water	–	–	–	–	–	–	–	–	10	505	4.0
0.9% NaCl	154	154	–	–	–	–	–	–	–	308	5.0
Lactated Ringer's solution†	130	109	4	3	–	28	–	–	–	273	6.5
PlasmaLyte A†	140	98	5	–	3	–	27	23	–	294	7.4
PlasmaLyte 148†	140	98	5	–	3	–	27	23	–	294	5.5
Hartmann solution†	131	111	5	2	–	28	–	–	–	279	6.5
Vetivex 18*	31	31	–	–	–	–	–	–	4	264	–
7.5% Hypertonic NaCl	1283	1283	–	–	–	–	–	–	–	2567	5.0–5.7
Hetastarch: COP 31 mm Hg (6% in 0.9% NaCl)	154	154	–	–	–	–	–	–	–	310	5.5

*Maintenance solution for use after volume replacement complete.
†Isotonic polyionic fluid with a relatively normal strong ion difference.

Six-Day-Old Foal With Diarrhea

Saline, either hypertonic (500 mL) or isotonic, was more frequently chosen (30% of respondents) for use in this case than for the first case. Hetastarch was also mentioned by one respondent, whereas the majority chose isotonic polyionic crystalloid replacement fluids. Many suggested KCl addition to fluids at 20 mEq/L. Overall, this case prompted the most requests for blood gas or electrolyte results before making fluid choices. Calcium gluconate was suggested if low, whereas one respondent suggested bicarbonate if low on blood gas.

Three-Day Postpartum Mare Presenting With Mild to Moderate Colic With Foal at Side and in Shock (Heart Rate, 72; mm Pale; CRT, 3 Seconds; Slow Jugular Fill; Extremities Cold)

Seventy percent of responding veterinarians would initially administer 2 L of hypertonic saline to this horse, followed by isotonic polyionic crystalloid replacement fluids at two times maintenance. Additives were uncommon in responses to this question but when mentioned included aminocaproic acid (70 mg/kg over 20-minute loading dose, then 15 mg/kg per hour in 0.9% saline as a CRI), oxytocin (50 mg), calcium (120 mL 23% calcium borogluconate in 5 L), and hetastarch.

Two-Year-Old Thoroughbred Filly With a Head Injury From Falling Backward on Its Poll, No Epistaxis

One veterinarian would not administer fluids until neurologic status fully assessed, whereas 60% of respondents chose to administer 1 to 2 L of hypertonic saline as their first bag. Saline (0.9%) was administered by 20%, whereas isotonic polyionic crystalloid replacement fluids were mentioned either as first-line treatment or as follow-up to hypertonic saline. Additives included thiamine and $MgSO_4$ added to follow-up fluids at 20 mL 50% $MgSO_4$/L for a maximum of 50 mg/kg.

Four-Year-Old Quarter Horse With Frequent Pipestream Diarrhea, Depressed, Approximately 8% Dehydration, Has Been on Antibiotics, Mucous Membranes Injected (Heart Rate, 80)

Forty percent of respondents would initially treat this case with hypertonic saline (1 to 2 L) followed by isotonic polyionic crystalloid replacement fluids. The remainder would administer only isotonic polyionic crystalloid replacement fluids at bolus rates, whereas a small percentage would use 0.9% saline. Additive or additional infusions during initial treatment included polymyxin B (1.5–3 million units), calcium (as 23% calcium gluconate), and hetastarch; follow-up fluid additives were potassium chloride (20 mEq/L) and calcium gluconate.

Ten-Year-Old Arabian Mare, Badly Tied Up After an Apparently Energetic Trail Ride, Not Making Urine

Initial fluid choices in order of frequency chosen included isotonic polyionic crystalloid replacement fluids as a bolus (generally 20 L), 0.9% saline, and hypertonic saline followed by isotonic polyionic crystalloid replacement fluids. Enteral water administration was mentioned. Calcium gluconate was the only mentioned additive.

Twenty-Two–Year-Old Morgan Horse With Mild Colic and Palpable Pelvic Flexure Impaction

Respondents overwhelmingly chose enteral water with or without electrolytes and $MgSO_4$ as their first bag. Mineral oil administration by nasogastric tube was mentioned. Many stated that if the impaction was severe, they would add isotonic polyionic crystalloid replacement fluids at twice maintenance with KCl (20 mEq/L) as the most commonly mentioned additive.

Eight-Year-Old Thoroughbred Broodmare 338 Days in Foal Presenting With Severe Colic and Abdominal Distension

Both hypertonic saline and isotonic polyionic crystalloid replacement fluids were commonly the first fluids administered, with isotonic polyionic crystalloid replacement fluids also required as follow-up to hypertonic saline. Hetastarch was chosen to be given simultaneously with isotonic polyionic crystalloid replacement fluids in one instance. One respondent chose 0.9% saline. Calcium was mentioned once as an additive. Many respondents were very busy trocharizing the colon/cecum and preparing for surgery while the fluids were being administered.

4. Discussion

Intravenous fluids are administered to veterinary patients for several purposes, including replacement of lost vascular volume (bleeding, sensible and insensible fluid losses, etc); replacement of total body water deficits (dehydration); replacement of electrolyte losses or correction of electrolyte aberrations (ie, hyperatremia or hyponatremia); correction of metabolic acid-base abnormalities (hyperlactemia, metabolic acidosis); provision of parenteral nutrition (glucose, proteins as amino acids, lipids, vitamins, trace minerals); correction of low oncotic states (hypoproteinemia); and treatment for specific medical conditions. Determining which IV fluids to administer for which purposes and which medical conditions has been the subject of many reviews and discussions, but, ultimately, any equine practice must choose IV fluids to keep readily available for administration in cases requiring immediate intervention. This brief survey was conducted to provide information to equine veterinary practices regarding which fluids might be suggested for initial use by specialists in both internal medicine and emergency and critical care on the basis of their experience in treating emergent cases. From the responses submitted, it was clearly frustrating for many specialists not having hematology results, physical examination findings, and history; however, this created more realistic ambulatory situa-

tions requiring the specialists to use their best guess of the underlying problem(s).

The next sections grouped each clinical situation assessed by the specialists into four groups: gastrointestinal disturbances, critically ill neonatal foals, central nervous system (CNS) trauma, and renal injury. The physiology will be reviewed and the choices of the specialists discussed in each of the categories.

Gastrointestinal Disturbances

Four of the nine clinical situations were designed as examples of gastrointestinal disturbances ranging from mild to severe, with one additional case presenting as either a gastrointestinal disturbance or blood loss case. In the most mild example, the 22-year-old Morgan horse with mild colic and palpable pelvic flexure impaction, most veterinarians did not immediately administer intravenous fluid support but rather preferred to administer enteral fluids (frequently with mineral oil) with or without electrolytes. Intravenous fluids, administered as isotonic polyionic crystalloids, were only suggested if the impaction was severe and then only as two times maintenance in addition to enteral fluids.

The 6-day-old foal with diarrhea prompted many requests for additional information because the severity and potential importance of fluid loss associated with diarrhea in these cases can be quite variable. Whereas the majority of respondents chose isotonic polyionic crystalloids, many chose to administer saline, either as 0.9% (normal saline) or as 7.5% (hypertonic saline). It is not unusual for foals of this age to have unpredictable but severe electrolyte imbalances—hyponatremia and hypochloremia, for example—with severe diarrhea and dehydration, prompting some to reach for saline as their first bag.[1–4] For others, the advantage of isotonic polyionic crystalloid fluids being closer to normal plasma values in their makeup (Table 1) may have been of greater benefit, allowing for more gradual correction of any unrecognized electrolyte imbalances. Too-rapid correction of either hypoatremia or hypernatremia has been reported as deleterious in many species.[5] In these cases, fluid resuscitation is aimed first at volume replacement and then at electrolyte correction, once data become available; this approach is reflected in the majority choice of isotonic polyionic crystalloid fluids.

The 4-year-old Quarter horse with frequent pipestream diarrhea, depression, approximately 8% dehydration, injected mucous membranes, and a heart rate of 80 beats/minute that had been on antibiotics represents a severely dehydrated horse with (probable and somewhat predictable) electrolyte abnormalities (again, hyponatremia and hypochloremia) and endotoxemia (implied).[6,7] Respondents were somewhat equally divided between administration of hypertonic saline (1 to 2 L) followed immediately by isotonic polyionic crystalloid fluids or immediate bolus administration of isotonic polyionic crystalloid fluids. The goal of immediate therapy in these cases is adequate restoration of vascular volume to ensure tissue perfusion and either approach is probably acceptable. Correction of electrolyte imbalance can take place later in the clinical course, and the relatively small volume of hypertonic saline administered should not correct hyponatremia too quickly.[5] In this case, a large fluid deficit is present. Correction of estimated dehydration, assuming a 500-kg horse, is 40 L; additional fluids would be required to address continued losses and maintenance. A reasonable approach would be 20 L of isotonic polyionic crystalloid fluids as a bolus over 2 to 4 hours followed by 3 to 4 L per hour. Polymyxin B (1.5–3 million units) was suggested as an additive to the first bag as an anti-endotoxemia treatment. Calcium (as 23% calcium gluconate) and KCL (20 mEq/L) were suggested as additives to follow-up fluids. Hetastarch was listed by some as an initial fluid to be administered along with the initial crystalloid fluids, because of presumed (and probable) hypoproteinemia.

The 8-year-old Thoroughbred broodmare, 338 days in foal presenting with severe colic and abdominal distention, was presumed by many to be a surgical candidate, prompting one respondent to reply: "Oh dear! Trocharize and bolus on the way to the surgical suite!" In this case, restoration of vascular volume and tissue perfusion was considered paramount, and both hypertonic saline (1 to 2 L) followed by high-rate isotonic polyionic crystalloid fluids and bolus administration of isotonic polyionic crystalloid fluids were suggested approximately equally. Respondents were concerned with improving perfusion before definitive treatment. Depending on the time needed before definitive diagnosis and treatment, administration of 20 to 40 L of isotonic polyionic crystalloid fluids might be required.

Finally, a 3-day postpartum mare presenting with mild to moderate colic with a foal at her side and in shock, had heart rate of 72; mm pale; CRT, 3 seconds; slow jugular fill; extremities cold. The assumption made by most in this case was postpartum bleeding. However, gastrointestinal accident, peritonitis, uterine tear, or other periparturient problems might be the primary concern. Respondents overwhelmingly chose to administer hypertonic saline (2 L) followed by bolus administration of isotonic polyionic crystalloid fluids in an effort to combat shock. Additives mentioned included aminocaproic acid (70 mg/kg over 20-minute loading dose, then 15 mg/kg per hour in 0.9% saline as a CRI), oxytocin (50 mg), calcium (120 mL 23% calcium borogluconate in 5 L crystalloid). Hetastarch was used by some, presumably to counteract soon-to-be-recognized hypoproteinemia caused by whole blood loss.

Critically Ill Neonatal Foals

Two cases representing critical illness in a neonatal foal were included. The first, a 10-minute-old foal

delivered from a prolonged dystocia (~120 minutes) with an APGAR score of 6 received a wide variety of fluid approaches ranging from none (with further close observation) to 500 mL to 1 L of isotonic polyionic crystalloid fluids. In this situation—unless blood loss occurred or there was severe in utero sepsis—vascular volume resuscitation is not necessarily required initially. The APGAR score suggests a mildly to moderately asphyxiated foal, and additives suggested by respondents were aimed at early intervention for this. Supplements suggested to be added to the first bag included (in order of frequency mentioned): dextrose (1% to 5%), thiamine (1 g/L), vitamin C, DMSO (1% to 2%), and 50% $MgSO_4$ (25 mL). Dextrose is aimed at providing energy support, thiamine supports normal intracellular energy metabolism, vitamin C and DMSO are provided as anti-oxidant treatment, and magnesium is thought to be neuroprotective.

The second foal case, an obtunded, hypothermic minimally responsive 24-hour-old foal, represents a variety of conditions of the critically ill neonate ranging from severe sepsis to hypoxic ischemic disease. Initial treatment of these foals is fairly uniform and aimed at stabilization and intravascular volume resuscitation in addition to providing an energy source. All respondents chose to administer isotonic polyionic crystalloid replacement fluids at bolus rates (20 mL/kg over 20 minutes, repeated as necessary), but the majority also recognized the need for almost immediate energy support. Energy support was supplied either as a piggyback CRI (4 mg/kg per minute, ~250 mL/h 5% dextrose solution to a 50-kg foal) or as a 1% additive to the first crystalloid bag followed by a CRI as described. I personally tend to include the first dose of any IV antimicrobial treatment in the first bag in addition to 1% dextrose while I begin preparing for dextrose CRI (5% dextrose at 250 mL/h initially will work for most foals) in these cases.

CNS Trauma

The 2-year-old Thoroughbred filly with a head injury from falling backward on its poll, with no epistaxis, represented a case of CNS trauma. This type of injury is not uncommon in practice and is probably seen most commonly in foals being halter-broken. The concern in these cases is the severity of injury, if there is basisphenoid injury, and if there is rectus capitis rupture or avulsion from the skull base. In this example, it was suggested that rectus capitis avulsion did not occur as a result of the absence of epistaxis. Coup–contra coup injuries also occur with these injuries, and fluid therapy is aimed at minimizing edema and further injury to the brain. Hypertonic saline is commonly recommended in the treatment of CNS injuries,[8,9] particularly those involving the brain, and the respondents were apparently aware of this as hypertonic saline was the fluid of choice for the majority. A few chose not to administer fluids immediately, whereas others opted for isotonic polyionic crystalloids or 0.9% saline. Additives included thiamine. $MgSO_4$ was also added to follow-up fluids at 20 mL 50% $MgSO_4$/L for a maximum of 50 mg/kg.

Renal Injury

The anuric 10-year-old Arabian mare, badly tied up after an apparently energetic trail ride, represented a case of acute kidney injury associated with pigment (myoglobin) released from the body and deposited in the renal tubules. Initiation of diuresis is the first-order treatment in these cases. Respondents chose to treat this mare with isotonic polyionic crystalloid replacement fluids as a bolus (generally 20 L), 0.9% saline, or hypertonic saline followed by isotonic polyionic crystalloid replacement fluids. Caution is required because continued administration of fluids to cases such as this without inducing urine production can be harmful and result in fluid overload with pulmonary edema. If an initial bolus does not result in urine production, other methods of inducing diuresis should be attempted, such as furosemide, and fluid administration must be slowed down or stopped.

5. Conclusions

Hypertonic saline (1-L bags), isotonic polyionic crystalloid fluids with a normal strong ion difference (1 L and 3- to 5-L bags) and 5% dextrose in water (1-L bags) appear to be the most commonly chosen IV fluids in this survey. Volumes that might be useful to have on hand for an initial IV fluid resuscitation in an ambulatory situation might include the following:

Foals: 1 to 4 L isotonic polyionic crystalloid; 2 L 5% dextrose

Adults: 2 L 7.5% hypertonic saline; 20 to 30 L isotonic polyionic crystalloid; hetastarch

Commonly mentioned additives included thiamine, calcium (23% calcium gluconate), magnesium (50% $MgSO_4$), and polymyxin B, and all are easily carried in an ambulatory practice. If practical and within the client's budget, hetastarch or a similar colloid may prove useful in some situations.

The reader is cautioned that additional fluids would be required for continued treatment of all cases listed above. Some specific fluid brands are listed in Table 1.

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[b]Hartman Solution, Hemofarm Pharmaceutical, Vršac, Serbia.
[c]PlasmaLyte A, Baxter Healthcare Corporation, Deerfield, IL 60015.
[d]PlasmaLyte 148, Baxter Healthcare Corporation, Deerfield, IL 60015.
[e]Vetivex 18, Dechra Veterinary Products, Shropshire, England.
[f]Hextend (Hetastarch), BioTime, Inc., Berkeley, CA 94710.
[g]HESPAN®, B. Braun Medical Inc., Irvine, CA 92614.

Review of Fluid Therapy in Acute Blood Loss

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Permissive hypotension and increased use of plasma and fresh, warm, whole blood instead of crystalloid fluids may benefit equine patients with acute blood loss. Author's address: Hagyard Equine Medical Institute, 4250 Iron Works Pike, Lexington, KY 40511; e-mail: mfrazer@hagyard.com. © 2013 AAEP.

1. Introduction

Acute blood loss in the veterinary patient is an emergency that many practitioners must manage in the field or hospital setting. Diagnosis may be obvious in cases of external blood loss, whereas internal blood loss may be more difficult to determine. Acute hemorrhage can occur into the peritoneal, pleural, or pericardial cavities; reproductive tract; gastrointestinal tract; guttural pouches; joints; and muscle tissue. In the equine patient, common causes include trauma, rupture of a vessel in the reproductive tract in pre- or post-foaling mares, fractured ribs in foals, and inadequate hemostasis during surgery. History, physical examination, ultrasound examination, and blood work aid in diagnosing acute hemorrhage as well as assessing the severity and determining the cause.

In hemorrhage, the number of circulating red blood cells decreases, and the oxygen-carrying capacity of the blood is compromised. Initially, physiological responses are able to compensate and maintain blood pressure with transcapillary refill, tachycardia, tachypnea, and systemic vasoconstriction. When significant volume is lost, the body can no longer compensate for the blood loss, and hemorrhagic shock occurs. In this situation, adequate tissue perfusion and oxygenation cannot be maintained. This eventually leads to organ dysfunction and cardiovascular collapse.

Patients with blood loss can be placed into one of four categories as defined by the American College of Surgeons.[1] Category 1 is loss of $\leq$15% of blood volume. Transcapillary refill typically compensates for this loss and maintains blood volume and blood pressure. Category 2 is loss of 15% to 30% of blood volume. Compensatory mechanisms such as tachycardia and tachypnea occur, and sympathetic vasoconstriction can typically maintain blood pressure. Category 3 is loss of 30% to 40% of blood volume. Compensatory mechanisms can no longer maintain blood pressure, and decompensated hypovolemic shock occurs. Organ dysfunction, such as acute renal failure, may occur from decreased tissue oxygenation. Decreased urine production and hypotension occur. Category 4 is loss of $>$40% of blood volume. Patients in this category require immediate emergency treatment, and changes in blood pressure and perfusion may not be reversible.

The goal of treatment in patients with acute blood loss is preventing hemorrhagic shock while also preventing further loss of blood. Two areas of controversy have occurred as to how best to accomplish this goal: fluid type and volume to be administered.

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2. Materials and Methods

A review of literature from the past 10 years describing fluid resuscitation for acute blood loss in human medicine was undertaken. Current trends in fluid choice and volume of fluid administered were reviewed.

3. Results

In human medicine, a strategy has been developed called Damage Control Resuscitation (DCR) to resuscitate patients with acute hemorrhage.[2,3] The goal of DCR is early prevention and/or treatment of the lethal triad. The lethal triad consists of coagulopathies, hypothermia, and acidosis.[3] Metabolic acidosis occurs from hypoperfusion leading to organ/tissue damage from decreased oxygenation and a switch to anaerobic metabolism. Hypothermia results from hypoperfusion and, if used in treatment, the use of cold resuscitation fluids. Coagulopathies primarily occur from hypoperfusion and tissue trauma. Other factors including loss of procoagulant proteases, dilution of blood from fluid resuscitation, and organ dysfunction from acidosis and hypothermia also potentiate coagulopathies.[4–6]

One of the key points in the DCR resuscitation plan is permissive hypotension. A minimum volume of intravascular fluid replacement is administered at a rate to maintain mean arterial blood pressure at 50 mm Hg and systolic blood pressure at 80 mm Hg. These pressures are considered sufficient to maintain organ perfusion without potentiating hemorrhage. Proponents of DCR cited a 40% survival rate in DCR-resuscitated patients versus a 16% survival rate in patients not undergoing DCR resuscitation strategies.[7–10] DCR is now the standard of care for resuscitating patients with blood loss in human medicine.[7] The DCR strategy has emerged over the past decade and is in opposition to the strategy used during the past century, when rapid volume expansion with crystalloid fluids was used in cases of hemorrhagic shock.

Permissive hypotension should not be confused with prolonged hypoperfusion. Prolonged hypoperfusion and failure to maintain blood pressure at DCR-recommended levels results in decreased perfusion to vital organs and tissues leading to metabolic acidosis, hypothermia, and coagulopathies. Coagulopathies are associated with increased mortality in patients with hemorrhagic shock.[11–13]

The second key point in DCR resuscitation is the type of fluid chosen for initial resuscitation. The DCR strategy recommends the use of plasma as the initial resuscitation fluid. This early use of plasma aids in prevention of a coagulopathy, one component of the lethal triad.[14] Human studies claim a 46% reduction in mortality rate when plasma was used in equal parts with whole blood as opposed to the use of more blood than plasma.[14] Also, fresh, warm, whole blood has been advocated over packed red blood cells.[7,15,16]

4. Discussion

Different strategies are used to treat acute blood loss in veterinary patients. Some clinicians limit intravenous fluid therapy in an attempt to maintain low blood pressure and limit further hemorrhage. The risk of limiting fluid therapy is hypovolemic shock and subsequent cardiovascular collapse. Without appropriate fluid volume, organs do not receive adequate perfusion and oxygenation. In opposition, some clinicians advocate more liberal use of crystalloid and colloid fluids to maintain blood pressure in a range closer to normal to preserve organ perfusion and prevent hypovolemic shock. The risk with this method is that further hemorrhage may occur.

Veterinarians also vary in what fluid should be used in resuscitation. Crystalloid fluids are typically inexpensive, easy to administer, and readily available to most practitioners. However, the majority of the fluid rapidly leaves the vascular compartment and moves to the interstitial space. Colloid fluids are more expensive and require specialized equipment to administer. Fresh, whole blood requires the presence of a donor and ideally, a laboratory to perform a cross-match. Plasma requires refrigeration for storage. Both require specialized IV lines with a filter designed for administration of blood products. Anaphylactic reactions may occur with either. Hydroxyethyl starch, a synthetic colloid, does not require refrigeration or specialized equipment to administer, but it has been associated with coagulopathies. Also, colloids may increase the blood pressure more than desired and may potentiate further bleeding.

Extrapolating data and strategies from human medicine and applying it to veterinary medicine may assist the veterinarian in establishing a treatment plan for resuscitative fluids in acute hemorrhage cases. Application of DCR strategy to veterinary medicine will guide the practitioner in choice of resuscitative fluid in hemorrhagic shock. Plasma and potentially warm whole blood should be the initial resuscitation fluid. Plasma provides additional clotting factors. Warm blood is used to prevent hypothermia because the coagulation cascade is less effective at lower body temperatures. Fresh, whole blood is preferred over packed red blood cells. Interestingly, this practice is already routinely performed in equine medicine because a blood bank with packed red cells from equids is not available.

DCR protocol recommends minimal administration of crystalloid fluids because they dilute coagulation factors and may increase coagulopathies. However, in the equine patient, financial constraints and availability of colloids may prevent administering the volume of plasma and whole blood needed to maintain mean arterial blood pressure at 50 mm Mg. Crystalloid fluids, therefore, are required in these cases.

Historically, hemoglobin and hematocrit levels have been utilized to determine when blood transfusions should be administered. However, these clinical parameters are not reliable transfusion triggers because they are unreliable indicators of blood loss severity. In acute blood loss, red blood cells and plasma are both lost; therefore hematocrit and hemoglobin do not change until transcapillary refill and other compensatory mechanisms occur. In addition, treatment with intravenous fluids will dilute but not alter the total number of circulating red blood cells. Therefore, the hematocrit decreases even if the number of red blood cells remains unchanged. In species that have a large reservoir of red blood cells in the spleen (such as the horse), the hematocrit alters rapidly with splenic contraction and may initially increase even in patients with severe blood loss.

Application of DCR strategy will also aid in determining the volume of fluid to administer in hemorrhagic shock. The goal of DCR is permissive hypotension. A volume of fluid is administered that maintains the mean arterial blood pressure at 50 mm Hg or the systolic blood pressure at 80 mm Hg. Measuring blood pressure in the equine patient to obtain the ideal pressure readings is difficult. Many field or hospital practitioners will not have equipment to accurately measure blood pressure. If a practitioner rarely encounters a case of hemorrhagic shock, justifying the cost of the equipment may be challenging. Even when equipment is available, obtaining frequent, reliable blood pressure readings may be impossible and potentially dangerous to the veterinarian or technician. Horses with blood loss may exhibit colic signs, ataxia, or change in mentation. Therefore, the blood pressure cuff may not be maintained in the correct position, and readings may be unreliable. Even if blood pressure readings are not obtained, other parameters may be utilized to determine if fluid resuscitation is adequate to maintain organ perfusion. DCR strategy advocates fluid therapy until the radial pulse can be palpated. In the equine patient, lack of peripheral pulses and cold extremities suggest hypoperfusion. Abnormalities in creatinine, blood urea nitrogen, and electrolytes are indicators of potential renal dysfunction. When these parameters are altered from the reference range, renal damage from hypoperfusion should be considered and fluid therapy must be adjusted.

Applying the DCR strategy of permissive hypotension and administration of plasma and fresh, warm blood to equine patients with acute hemorrhage may assist the veterinary practitioner in successful treatment of these cases. Limiting factors with this strategy are having the appropriate equipment to monitor blood pressure and having access to plasma or whole blood. However, physical examination and blood work parameters may compensate for lack of equipment. The DCR strategy needs further evaluation in equine medicine, but it may become the standard of care in resuscitation of equine patients with hemorrhagic shock.

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How to Maximize Standing Chemical Restraint

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1. Introduction

Sedation and analgesia are commonly required to help facilitate diagnostic and treatment procedures in standing horses. α_2-Adrenergic receptor (AR) agonists alone or in combination with opioids are used most commonly for this purpose.[1] Although such drug combinations in standing sedation are useful and mitigate some of the risks associated with general anesthesia,[2] they are not free of adverse effects. Dose-dependent respiratory depression, hypertension, bradyarrhythmias, decreases in cardiac output, hyperglycemia, polyuria, and decreased gastrointestinal motility are commonly caused by α_2-AR agonists.[3–8] Also, the presence of fear, stress, excitement, and pain can lead to failure to achieve satisfactory sedation with α_2-AR agonists.[3,5] The goal of this report is to present a practical technique that can help equine veterinarians in maximizing standing chemical restraint in patients undergoing diagnostic and/or surgical procedures.

2. Drugs and Drug Administration Techniques

Xylazine, detomidine, and romifidine are the most common α_2-AR agonists used to produce standing chemical restraint in equines, and the intravenous (IV) route of administration is preferred in most cases. An intravenous catheter is very useful for drug and fluid administration, especially for longer procedures. A single bolus administration can be used for short procedures (<30 minutes), whereas multiple boluses (ie, as needed) or a constant rate infusion (CRI) may be necessary for longer procedures. For longer procedures, a loading dose followed by a CRI is probably best because it produces more constant plasma levels and hence more stable sedation levels, and the overall dose used may be lower than with multiple doses. Administering small additional boluses can be used to intensify the level of sedation if required. Whereas α_2-AR agonists are generally considered potent and useful sedatives, some horses sedated with α_2-AR agonists may suddenly react to stimulation (even to touch), may become excited, or may develop aggression (especially with xylazine). Opioids can be used to modify some of these responses and improve the quality of the chemical restraint.

Combining opioids (morphine, butorphanol) with α_2-AR agonists will produce a state referred to as neuroleptanalgesia, in which the level of sedation and analgesia is more profound and greatly improves the chemical restraint. The opioids can be used as a single bolus for short procedures or, for longer procedures, as multiple intermittent boluses or as a loading dose followed by a CRI. Combinations of detomidine and morphine are very useful for procedures expected to be associated with strong noxious stimulation such as (but not limited to) ovariectomy and tooth extraction. For this tech-

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nique, the horse is first sedated with detomidine (0.005–0.01 mg/kg IV) and then given a slow (~5-minute) bolus of morphine (0.03–0.05 mg/kg IV). Morphine should be administered slowly to reduce possible risks of histamine release and to allow for early detection of an excitatory reaction. A CRI of both drugs can then be instituted (detomidine at 0.005–0.01 mg/kg per hour and morphine at 0.03–0.05 mg/kg per hour). Alternatively, morphine can be administered as intermittent IV boluses (slowly) as needed throughout the procedure. During the CRI, sedation level can be deepened with additional IV boluses of detomidine (0.002–0.005 mg/kg) or xylazine (0.1–0.2 mg/kg) as needed. Butorphanol can be used instead of morphine to improve sedation and analgesia produced by detomidine. It is administered as an initial IV bolus (loading dose) of 0.01–0.02 mg/kg that can be followed by a CRI (0.01–0.02 mg/kg per hour) or intermittent IV boluses (0.01–0.02 mg/kg IV) as needed. For the CRI, the drugs are best delivered with the use of syringe pumps as it allows for the most accurate dosing. However, drugs can also be added to a bag of crystalloid fluids and delivered through gravity flow with a drip set. For maximal control of infusion rates, drugs should be given through separate syringe pumps or fluid bags. For example, 10 mg of detomidine can be added to a 250-mL bag of saline (0.04 mg/mL) and dripped to effect through a 60-drop/mL drip set. With this setup, 1 drop per second will deliver 60 mL of fluid per hour or 2.4 mg of detomidine per hour. This is the equivalent to a dose of 0.005 mg/kg per hour of detomidine. It can be easily adjusted up or down from here as needed.

In patients undergoing urogenital procedures (ie, ovariectomy, removal of kidney stones), caudal epidural (S5-Cox1 or Cox1-Cox2) administration of morphine (0.1 mg/kg diluted into 15–20 mL of saline) produces useful analgesia during and after surgery.[9] Although not typically used for sedation of adult horses, very low doses of midazolam or diazepam (0.005–0.01 mg/kg) appear to relax the tongue and jaw and facilitate dental procedures.[3] Patients undergoing dental procedures may benefit from attenuation of visual (apply blindfold), auditory (apply cotton in ears), and oral tactile sensation (apply lidocaine gel on the surface of the tongue and oral mucosa). Patients with severe dental disease and suspected pathologic pain (ie, hyperalgesia, allodynia) may react severely to needle placement through the skin during the performance of nerve blocks. Application of a thick layer of eutectic mixture of local anesthetics (EMLA) cream on the skin over the site of the nerve block 20 to 30 minutes before the procedure greatly facilitates the performance of the nerve block. Patients undergoing prolonged sedation or expected to require deep sedation levels are in greater danger of development of significant ataxia and recumbency and therefore will benefit from a sling. Drugs and materials should be available to induce and maintain general anesthesia in the case that a patient undergoing standing chemical restraint becomes recumbent. For long procedures, a urinary catheter should be placed in equine patients sedated with α_2-AR agonists because these drugs increase urine production.

3. Discussion

Horses sedated with detomidine and morphine are typically less responsive to noxious as well as nonnoxious stimulation. However, local blocks are still required to facilitate most surgical procedures. When given as a CRI, this combination produces stable sedation that can be relatively easily adjusted as needed. In our institution, this is the preferred method of sedation of horses undergoing urogenital as well as head procedures.

We have observed that significantly more detomidine and morphine is required to produce satisfactory chemical restraint in horses undergoing extensive dental procedures (ie, root canals, multiple dental extractions, etc). Some of these horses may have significant bradycardia and require treatment with an anticholinergic such as butylscopolamine.[a] It can be given as small doses (0.05–0.1 mg/kg IV) as needed to maintain heart rate within an acceptable range for the patient.

Horses sedated with detomidine and morphine, especially those undergoing prolonged procedures, will also receive a crystalloid solution such as lactated Ringer's solution (2–5 mL/kg per hour) to maintain fluid balance and replace losses caused by polyuria. These horses also typically receive mineral oil through a nasogastric tube at the end of the procedure and are kept on colic watch for the following 24 hours. Morphine is avoided, used at the lowest possible doses, or replaced with butorphanol in horses with a recent history of colic. Antagonism of detomidine or morphine is typically not needed but could be performed with the appropriate antagonists.

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[a]Buscopan®, Boehringer Ingelheim Vetmedica, St. Joseph, MO 64506.

How to Use Local and Regional Anesthesia for Procedures of the Head and Perineum in the Horse

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1. Introduction

Combining local or regional anesthetic techniques with sedation will allow many diagnostic and surgical procedures to be safely performed in the horse. Drugs used for local anesthesia are designed to penetrate peripheral nerves and interrupt nerve conduction resulting in reversible anesthesia for a predictable period of time.[1] It is important for the clinician to have a thorough understanding of the pertinent anatomy in order to be successful with these techniques.

2. Local Anesthetics

The mechanism of action of local anesthetic drugs is to prevent transfer of nociceptive information by blocking sodium channels in excitable membranes.[1,2] Local anesthetics can be used topically, injected over nerves (perineural anesthesia), intrasynovial, regional infiltration, and injected into the epidural space. Common local anesthetics used in equine practice are lidocaine hydrochloride, mepivacaine hydrochloride, and bupivacaine hydrochloride.

Lidocaine is probably the commonly used because of its potency, rapid onset of action, moderate duration of action, topical anesthetic properties, and cost.[1] Peripheral nerve blocks and epidural anesthesia are typically performed with the use of a 2% lidocaine solution. The onset of action for peripheral nerve blocks is typically 5 to 10 minutes. The duration of action has been reported to be 1.5 to 3 hours, but clinical experience would suggest that the duration of action is shorter.[1,2] Repeated dosing or excessive amount of lidocaine can lead to toxicity. Total dosage should be kept less than 250 mL of 2% lidocaine in a 500-kg horse.

Mepivacaine is very similar to lidocaine. It has a similar onset of action, but the duration of action is longer.[1] Mepivacaine has less vasodilatory activity compared with lidocaine, and mepivacaine is less effective as a topical anesthetic compared with lidocaine.

Bupivacaine is two to four times as potent as lidocaine and mepivacaine. It has a much slower onset of action and a prolonged duration of action. The duration of action is reported to be greater than 3 hours.[1]

3. Anesthesia of the Head

Dental procedures are one of the most common reasons to use regional anesthesia of the head. Other indications include ophthalmic procedures, lacera-

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tion repair, and repair of incisive bone fractures. The maxillary, mandibular, alveolar, and mental nerve blocks are the regional nerve blocks of the head.[3] The horse should be sedated and properly restrained before the nerve block is given. The area over the injection site should be cleaned and prepped. Clipping of the hair is not necessary.

Maxillary Nerve Block

The maxillary nerve enters the infraorbital canal at the pterygopalatine fossa and becomes the infraorbital nerve.[3] Anesthetizing the maxillary nerve at the level of the pterygopalatine fossa will desensitize the ipsilateral dental structures of the maxilla and premaxilla, the paranasal sinuses, and the nasal cavity. There are two techniques to perform this block. The first method uses a 3.5-inch, 20- or 22-gauge spinal needle inserted perpendicular of the long axis of the head at the level of the caudal third of the orbit. The needle should be just ventral to the zygomatic process and dorsal to the transverse facial vessels. The needle is advanced until bone is contacted. The volume of local anesthetic to be injected is 15 to 20 mL.

The second technique uses a 6-inch spinal needle inserted just ventral to the most dorsal aspect of the zygomatic process. The needle is directed rostrally and ventrally, aiming toward the rostral aspect of the contralateral facial crest. The needle should be advanced until bone is contacted. The volume of anesthetic to be injected is 15 to 20 mL. Within 15 minutes, structures innervated by the maxillary nerve should be desensitized.

Infraorbital Nerve Block

The infraorbital nerve can be desensitized as it emerges from the infraorbital canal or within the infraorbital canal.[3] The effect is the same as achieved with the maxillary nerve block if local anesthetic completely fills the infraorbital canal. The skin of the ipsilateral lip, nostril, and face up to the level of the infraorbital foramen are desensitized when the infraorbital nerve rostral to the infraorbital canal is blocked. The left infraorbital foramen is located by placing the right thumb in the nasoincisive notch and the right middle finger on the rostral aspect of the facial crest. The right index finger should locate the foramen beneath the ventral margin of the levator labii superioris halfway between and 1 to 3 cm caudal to an imaginary line connecting the thumb and middle finger.

The infraorbital nerve is anesthetized by inserting a 22-gauge, 1.5-inch needle through the skin and into the canal for a distance of 1 inch. The volume of local anesthetic to be infused is 5 to 10 mL. Within 15 minutes, structures innervated by the infraorbital nerve should be desensitized.

Mandibular Nerve Block

The mandibular nerve enters the mandibular canal of the medial surface of the mandible.[3] The ipsilateral side of the mandible and all of its dental structures are desensitized when the mandibular nerve is blocked at the level of the mandibular foramen. The mandibular foramen is located by drawing an imaginary line from the lateral canthus of the eye ventrally and a second imaginary line that extends along and caudal to the occlusal surface of the mandibular cheek teeth. The point at which these two lines intersect is the mandibular foramen. A 6- to 8-inch, 20- to 22-gauge spinal needle is used for the injection. The needle is inserted at the ventral border of the ramus of the mandible and advanced dorsally toward the mandibular foramen. A second spinal needle of the same length can be held against the lateral surface of the mandible and used as a "depth guide" for the needle that was inserted on the medial surface of the mandible. Fifteen to 20 mL of local anesthetic should be infused. Within 15 to 30 minutes, structures innervated by the mandibular nerve should be desensitized.

Mental Nerve Block

The mandibular nerve travels through the mandibular canal and emerges at the mental foramen as the mental nerve.[3] The mental nerve provides innervation to the skin of the ipsilateral lip and chin. Branches of the mandibular nerve that lie within the mandibular canal provide innervation to the mandibular canine, incisor, and cheek teeth. The mental foramen is located on the lateral aspect of the horizontal ramus of the mandible in the interdental space. The foramen is beneath the tendon of the depressor labii inferioris muscle. This tendon must be elevated to palpate the foramen. To perform this block, a 1.5-inch, 22-gauge needle is inserted through the skin, and the tendon of the depressor labii inferioris muscle is then elevated. The needle is advanced into the foramen and 5 to 10 mL of local anesthetic is infused. Within 15 minutes, structures innervated by the mental nerve should be desensitized. Infusing large volumes (>10 mL) of local anesthetic into the mandibular canal is likely to desensitize the same structures as in performing a mandibular nerve block.

4. Anesthesia of the Perineum

Caudal epidural anesthesia is a common technique used to desensitize the perineum and associated anatomic structures. This technique has been used to facilitate many procedures, both medical and surgical. Examples include the following: to prevent or stop rectal tenesmus, assist with obstetric manipulations, and assist with surgical procedures of the caudal reproductive tract.[2]

Epidural Anesthesia

Epidural anesthesia is achieved when the local anesthetic solution is deposited between the dura mater and the periosteum of the spinal canal, which blocks conduction in the caudal nerve roots.[4] Caudal epidural anesthesia implies that sensory inner-

vation is lost but motor control of the hind limbs is not affected. However, the tail should be tied overhead to support the horse if ataxia develops. The sacrocaudal or first intercaudal vertebral space is selected as the site for injection for epidural anesthesia.[5] This location is found by grasping the tail and moving it up and down. The first articulation caudal to the sacrum is the first intercoccygeal space. The site should be clipped and aseptically prepared. The epidural should be administered with the use of aseptic technique. There are various injection techniques and drug combinations that can be used. The horse should be sedated and restrained in stocks (if available) during administration of epidural anesthesia. A small skin bleb of local anesthetic can be deposited at the proposed injection site to facilitate placement of the spinal needle. A 20-gauge, 7.5-cm spinal needle should be positioned just cranial to the dorsal spinous process of the second coccygeal vertebra. The needle is inserted through the skin at an angle 30 degrees relative to the tail and inserted cranially. If bone is encountered, the needle should be redirected. Once the needle is placed, the stylet should be withdrawn and the hub of the needle is filled with the local anesthetic to be injected. If the needle is positioned in the epidural space, the fluid will be aspirated (hanging drop technique). Minimal resistance is encountered during epidural injection. Horses that have had previous epidural injections may develop fibrous scar tissue over the intercoccygeal space, making needle placement difficult.[5] After injection, the needle should be removed. Caution should be exercised during placement of the spinal needle because some horses will kick during the procedure. An epidural catheter can be placed to facilitate readministration of anesthetic agents if needed during the surgery. Loss of anal sphincter tone is common when epidural anesthesia is achieved.

The type of blockade (motor and/or sensory) and the duration of effect is dependent on the type of drug(s) and the volume that is administered. Local anesthetics should produce motor and sensory blockade, whereas only sensory innervation is lost with other drug treatments. The use of 5 to 7 mL of 2% lidocaine hydrochloride per 500 kg body weight should produce analgesia within 5 to 15 minutes, and the duration of analgesia should be 60 to 90 minutes.[5] Two percent mepivacaine hydrochloride given at the same dose will produce analgesia in 10 to 30 minutes and provide analgesia for 90 to 120 minutes.[4] Ataxia is a complication when local anesthetics are used.

Epidural administration of α_2-adrenergic agonists such as xylazine will provide profound analgesia without the complication of ataxia.[6,7] The recommended dose of xylazine is 0.17 mg/kg. The onset of action is 10 to 30 minutes, and the duration of analgesia is 2.5 to 4 hours.[5] The xylazine should be diluted in saline to a total volume of 6 to 10 mL. Epidural administration of detomidine (30–60 μg/kg) provides analgesia lasting for 2 to 3 hours but produces sedation and ataxia.[5,6] The combination of lidocaine (0.22 mg/kg) and xylazine (0.17 mg/kg) produces significantly longer analgesia (approximately 5 hours) with only mild ataxia when compared with either agent used alone.[7]

Ataxia and the potential for recumbency must be considered when determining which drug or combination of drugs and the dosage that is to be administered.

5. Conclusions

The successful outcome of the use of local anesthetic drugs depends on the clinician to accurately deposit an appropriate volume and type of drug at the correct anatomic site. This can be achieved by a thorough understanding of the pharmacology of local anesthetics and equine anatomy.

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How to Provide Pain Relief for Laminitis in the Field

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1. Introduction

Laminitis is an extremely painful condition with still incompletely understood pathophysiology.[1–3] All too often, the uncontainable severe pain associated with the disease is the single most common reason for euthanasia of laminitic horses. Treatment is frequently unfinished because owners and health providers feel forced to end the extreme pain and suffering by ending the life of the afflicted animal. More positive treatment results could be obtained if pain and suffering could be successfully modulated.[4] Non-steroidal anti-inflammatory drugs (NSAIDs) such as flunixin meglumine and phenylbutazone have long been and still remain the primary pharmacologic agents used to treat pain and inflammation in horses with laminitis.[5]

Tramadol is emerging as an additional option toward providing multimodal analgesia in equine laminitis,[6] and its efficacy has been well established in neuropathic and inflammatory pain states.[7–10] Because it is now known that pain associated with laminitis has both inflammatory and neuropathic components,[11,12] targeted multimodal analgesic therapies probably will be necessary for effective pain modulation. Drugs such as ketamine and gabapentin may be very beneficial in modulating neuropathic pain as well as pathologic pain states characterized by hyperalgesia (ie, exaggerated response to a painful stimulus) and allodynia (ie, pain response to a normally nonpainful stimulus).[6,13–15] The goal of this report is to present techniques targeting such pathologic pain states that can be used to manage pain and suffering in horses with laminitis.

2. Recommended Agents and Routes of Administration

Tramadol tablets can be crushed into powder, mixed with syrup or molasses, and administered orally at a dose of 5 mg/kg q 12 hours.[6]

Ketamine can be administered intravenously as a constant rate infusion at a dose of 0.6 mg/kg per hour for 6 hours/day for 3 to 5 days (or longer as needed) through a syringe pump,[6] or it can be diluted in 0.9% saline and dripped by gravity. Alternatively, it can be administered intramuscularly at 0.5 mg/kg q 6 hours.[14]

Gabapentin can be administered orally at doses ranging from 2.5 to 20 mg/kg q 8 hours, q 12 hours, or q 24 hours, as needed.[13–15]

3. Evidence of Efficacy

When administered alone, tramadol was shown to produce significant initial improvement (3 of 7 days)

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in off-loading frequency in analgesic-naive horses with pain caused by chronic laminitis.[6] In this same study, ketamine administered intravenously for the first 3 days of treatment significantly improved off-loading frequency and forelimb load during (7 of 7 days) and after (3 days) tramadol therapy. Tramadol and ketamine had a modulatory role in the plasma levels of the pro-inflammatory cytokine tumor necrosis factor-α and the vasoconstrictor prostanoid thromboxane-A_2.[6] Gabapentin has been used as part of a multimodal analgesic protocol and was shown to help in the pain management of two horses with presumptive diagnosis of neuropathic pain.[14,15]

4. Discussion

Laminitis pain is associated with increased off-loading frequency, decreased forelimb load, and other behavioral and cellular changes congruent with pathologic pain states characterized by hyperalgesia and allodynia.[6,11] Pain relief with tramadol results from complex interactions with opioid, adrenergic, and serotonin receptor systems[16–18] and possibly through modulation of inflammatory mediators such as pro-inflammatory cytokines and prostanoids.[10,19] In analgesic-naive horses with chronic laminitis, co-administration of tramadol and ketamine resulted in significant improvement in off-loading frequency, forelimb load, and plasma levels of a major pro-inflammatory cytokine and a critical vasoconstrictor prostanoid.[3] It is possible that co-administration of tramadol with NSAIDs could result in greater modulation of inflammatory responses and superior pain management than when each drug is used alone.

Ischemia and inflammation in the early stages of laminitis probably cause neuronal injury that eventually shifts the acute inflammatory pain into a chronic syndrome with a prominent neuropathic component. The precise timing and nature of these events are not precisely known, but it may be established in as early as a few days. The neuropathic pain component in laminitic horses is not well responsive to NSAIDs and opioids but typically responds to modulators of N-methyl-D-aspartate receptors (ie, ketamine) and voltage-gated calcium channels (gabapentin) in nociceptive neurons in the spinal cord and brain.[13] The beneficial effects of these drugs have been demonstrated in horses with apparent neuropathic pain.[6,14,15] The equine practitioner should consider introducing these therapeutic modalities as soon as it is perceived that the current standard of care is not producing the desired pain control. The use of NSAIDs, tramadol, ketamine, and/or gabapentin appears to be a sound mechanistic-based approach in providing multimodal pain management in horses with laminitis.

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How to Produce Twenty Minutes of Equine Anesthesia in the Field

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1. Introduction

Every week, 50% of equine veterinarians anesthetize horses for short periods of time.[1] More than 90% of equine veterinarians use ketamine as the primary anesthetic drug for short-term anesthesia, with many incorporating diazepam to augment muscle relaxation.[1] The widespread application of ketamine for anesthesia in horses probably represents its relative efficacy and safety and thus the level of confidence that equine veterinarians have in its use. This report will provide recommendations on how to maximize the use ketamine for short-term field anesthesia in the horse.

2. Background Information

Ketamine is a dissociative anesthetic that produces its effects through antagonism at N-methyl-D-aspartate (NMDA) receptors in the brain and spinal cord. Ketamine anesthesia in most species is characterized by indirect stimulation of the cardiovascular system and maintenance of respiration and respiratory reflexes.[2] Animals receiving ketamine show varying degrees of muscle hypertonus, and purposeful or reflexive skeletal muscle movements may occur.[2] The presence of active ocular reflexes coupled with the increases in muscle tone complicate monitoring of anesthesia.[2] The use of ketamine for equine anesthesia was introduced to veterinary medicine in 1977.[3] The report described the use of the only available α_2 agonist at the time, xylazine, given before or in combination with ketamine for short term intravenous (IV) anesthesia. This combination produces an excitement-free induction and recovery, maintenance of normal cardiovascular function, moderate respiratory depression, and adequate muscle relaxation.[3] The average duration of anesthesia was approximately 16 minutes, and horses stood for 12 minutes after a single administration of the anesthetic combination.[3] Subsequent publications have confirmed the safety of the technique in horses but have noted some potential problems that the practitioner should recognize. Major reported problems associated with the use of xylazine and ketamine in the horse include inadequate sedation before ketamine administration producing induction failure, inadequate muscle relaxation during recumbency, and too short a duration of anesthesia.[4,5]

3. Alternative or Additional Sedatives and Analgesics

Since the introduction of xylazine-ketamine anesthesia, two additional α_2 agonists have been approved for use in horses in the United States: detomidine and romifidine. The combination of these agents with

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ketamine for short-term IV anesthesia has been investigated. Detomidine (0.02 mg/kg IV) in combination with ketamine produces induction of anesthesia similar to that of xylazine-ketamine with potentially better muscle relaxation.[6] The depth of anesthesia as shown by the ease of continuing anesthesia with thiopental was thought to be superior to xylazine-ketamine, but the recovery from anesthesia tended to be less coordinated when detomidine was used, presumably because of its greater duration of action. Romifidine (0.1 mg/kg IV) in combination with ketamine is not recommended unless additional muscle relaxation is provided. This is because romifidine may not produce sufficient sedation and muscle relaxation for satisfactory anesthesia.[7]

Acepromazine and butorphanol have been given in conjunction with xylazine or romifidine before ketamine administration. Acepromazine increases the sedation seen after α_2 agonist (romifidine) administration and may prevent hypertension.[8] Induction was more rapid than with romifidine alone. Butorphanol should provide additional analgesia, and, in one study, it appeared to reduce the response to stimuli and facilitate smooth recovery by prolonging the duration of the anesthetic period.[9]

4. Improving Muscle Relaxation and the Quality of Anesthesia

The addition of the benzodiazepines diazepam or midazolam to the xylazine-ketamine combination produces improved sedation and muscle relaxation at minimal cost to the cardiovascular system.[10] Benzodiazepines produce muscle relaxation and some sedation through stimulation of γ-aminobutyric acid receptor–chloride channel complexes in the central nervous system. Diazepam or midazolam is given at a dose of 0.05 to 0.1 mg/kg IV immediately before or in combination with the standard dose of ketamine (2.2 mg/kg IV). The addition of diazepam or midazolam increases the duration of anesthesia to 20 to 25 minutes. Guaifenesin (50 mg/kg IV) is a centrally acting skeletal muscle relaxant that is administered after sedation with an α_2 agonist just before an IV bolus of ketamine is given.[11] Five percent to 10% solutions of guaifenesin are given because of the risk of hemolysis; thus, administration of a large volume (250–500 mL) is required, resulting in a period of ataxia at induction.[12] Intravenous catheterization is particularly useful when guaifenesin is used because of the large volume and the potential for vasculitis and cellulitis if it is administered perivascularly.[13] Guaifenesin produces some sedation but primarily is used to augment muscle relaxation. The quality of anesthesia is similar to diazepam-ketamine.[10] One advantage of guaifenesin administration is that additional quantities of drug are easily given to effect.

5. Alternatives to Ketamine

Tiletamine-zolazepam[a] can be administered to sedate horses to produce good-quality anesthesia for 30 to 40 minutes.[14] Alpha-2 agonists are administered to produce sedation and relaxation. Tiletamine-zolazepam is given IV after the onset of full sedation. The quality of anesthesia is similar to that with xylazine-diazepam-ketamine in that muscle relaxation is excellent. Respiration is depressed but remains adequate for the period of recumbency. Recoveries are not as smooth as that seen with the xylazine-ketamine combination because of the greater degree of muscle relaxation. The IV administration of a combination of ketamine (0.5 mg/kg), tiletamine-zolazepam (0.7 mg/kg), and detomidine (0.01 mg/kg) has been investigated for anesthesia for castration.[15] The combination is prepared by reconstituting 500 mg of tiletamine-zolazepam powder with 4 mL of ketamine (100 mg/mL) and 1 mL of detomidine (10 mg/mL). The mixture is given after xylazine sedation at a rate of 0.007 mL/kg, IV (~3 mL/450 kg). The combination produces excellent induction to anesthesia with intraoperative arterial blood pressures higher than those seen with most other techniques. The duration of anesthesia is longer than that with the use of xylazine and ketamine, and recoveries may require assistance. Propofol is an IV anesthetic that has been investigated in the horse both alone and in combination with ketamine and other drugs. Propofol has not been widely adopted in equine ambulatory practice because of concerns related to respiratory depression, including apnea.[16] Recently, alfaxalone, a progesterone analog, has also been investigated as an anesthetic in sedated horses.[17]

6. Recommendations

A history should be obtained and physical examination should be performed and recorded before induction of anesthesia. Preanesthetic blood work does not need to be extensive for short procedures and should be guided by the physical status of the patient. Written permission for anesthesia should be secured from the responsible party before sedation. Intravenous catheterization improves the safety of anesthesia by ensuring that medications are administered appropriately and reduces the manipulation and stimulation associated with injection. It is important to choose an area for anesthetic induction that also provides good footing for the horse during recovery. Turf or most riding arenas provide good footing. The area should be at least 4×4 meters in size and should be free from obstruction. Provision should be made for recording of the anesthetic drugs administered and an evaluation of their effects. Observations (at a minimum heart rate and respiratory rate) should be made continuously and recorded at 5- to 10-minute intervals.[18]

Xylazine and ketamine have been successfully used to produce short-term intravenous anesthesia in horses for more than 30 years. Although deto-

midine and romifidine can be used for sedation before ketamine administration, their substitution does not appear to provide additional benefit. A key concept of equine anesthesia is the maxim: "Never anesthetize an excited horse." Initial doses of xylazine of 1.0 mg/kg IV usually produce profound sedation within 5 minutes of administration. The horse assumes a head-down posture, with its nose at a level below its knees. Frequently, some ataxia and muscle relaxation will be evident, with the horse shifting its weight back and forth and occasional buckling of the knees. If this level of sedation is not attained, additional IV xylazine should be administered in 0.2 mg/kg increments until the horse is completely obtunded. Butorphanol (0.01 mg/kg IV) can be added to augment sedation and analgesia.

Once the horse is sedate, diazepam (0.05–0.1 mg/kg IV) or midazolam (0.05–0.1 mg/kg IV) should be administered in combination with ketamine (2.2 mg/kg IV). Recumbency occurs approximately 60 seconds after administration. The horse should be rolled to lateral recumbency and then should be positioned as desired. The halter should be removed or padded to prevent facial paralysis. Approximately 20 minutes of anesthesia should be anticipated. Ideally, an assistant should remain near the horse's head during anesthesia to control sudden movements.

It is important to recognize that the assumption of recumbency is associated with the development of ventilation-perfusion mismatches and that the shunting of blood through the lungs results in less-than-optimal oxygenation. The suboptimal oxygenation is well tolerated for short periods, but oxygen supplementation should be used for procedures longer than 60 minutes. Respiratory function should be monitored closely. Persons who routinely perform IV anesthesia should consider the purchase of an oxygen tank and regulator to facilitate emergency oxygenation and ventilation of the patient. Horses can be ventilated by adapting a nasogastric tube onto a pressure-reducing valve attached to an oxygen tank. The tube is slid up one nostril, and the nasal openings are occluded. The nostrils are released when the chest wall rises to a normal inspiratory level. The process is repeated until spontaneous ventilation resumes.

Position the horse in lateral recumbency for recovery, and attach a lead rope to the halter to control the horse once it stands.[19] Most horses will roll to sternal recumbency within 45 minutes then stand shortly thereafter. Do not closely restrain the head of the horse as it attempts to stand. Horses tend to place their front feet forwardly and then push themselves to a standing possession with the use the rear legs.

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[a]Telazol, Zoetis, Florham Park, NJ 07932.

How to Safely Anesthetize a Horse for Sixty Minutes or More in the Field

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1. Introduction

Most equine veterinarians perform short-duration anesthesia frequently, but only 10% anesthetize horses for greater than 30 minutes weekly.[1] Approximately 50% of equine veterinarians anesthetizing horses for greater than 30 minutes use inhalants for maintenance of anesthesia, with the balance using repeated injections of induction drugs or guaifenesin recipes.[1] Almost all equine veterinarians (87%) anesthetizing horses for greater than 30 minutes use an assistant to monitor the depth of anesthesia and administer additional anesthetic drugs as required.[1] This report will provide recommendations on how to safely anesthetize a horse for 60 minutes or more in the field with the use of injectable agents.

2. Background Information

The risks of morbidity and mortality increase with increasing duration of anesthesia.[2] The increase in risk associated with longer anesthetic durations is probably heightened in larger horses such as warmbloods or drafts because of the difficulty of assisting in recovery in a field situation. Foals less than 1 month of age appear to be at increased risk for anesthetic complications.[2] Fortunately, most foals respond to sedation and thus many procedures can be accomplished with the use of small doses of sedatives, such as xylazine, and physical restraint combined with local anesthesia. The topic of anesthesia of the foal in the field has been addressed previously.[3]

Successful management of equine anesthesia beyond short, single-administration techniques requires careful evaluation of the patient and adept planning of the procedure to keep the duration of anesthesia to a minimum. An intravenous (IV) catheter should be placed because constant-rate or intermittent administration of anesthetic drugs is required to maintain an anesthetic plane.[4] The site of anesthesia and surgery should be chosen on the basis of avoiding hard surfaces, the availability of padding (especially for the head), and the quality of footing (nonslip), anticipating concerns during the recovery phase. Padding, if available, should be placed under the points of the shoulder and the hip. For horses in lateral recumbency, the lower front leg is traditionally pulled forward with the goal of reducing pressure on the radial nerve.

Much of the success of equine field anesthesia is predicated on the maintenance of light levels of anesthesia. The need to administer additional anesthetic agents to deepen the level of anesthesia is determined on the basis of changes in respiration,

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increases in muscle tone, or movement. Heart rate and ocular signs are less reliable indicators of anesthetic depth when a ketamine-based technique is used. Limiting movement while administering additional anesthetic drugs is important; thus, most practitioners combine restraint with ropes with the anesthetic techniques.[5]

No single IV bolus injection of a drug or drug combination safely produces 60 minutes of anesthesia in the horse. Anesthesia should be induced with the use of techniques described for short-term equine field anesthesia.[6–10] The induction techniques usually produce approximately 15 to 25 minutes of anesthesia. During this period, the horse should be positioned for surgery and the halter removed or loosened to prevent facial paralysis as a result of compression of the facial nerve.

The use of intravenous infusions is preferred, but intermittent boluses of the induction drugs can be used. Xylazine and ketamine can be given at the rate of 30% to 50% of the initial dose (xylazine 0.5 mg/kg and ketamine 1.1 mg/kg) combined in the same syringe.[11] The administration of a second dose of the combination extends the anesthetic period approximately 10 minutes. Subsequent boluses in response to movement appear to be less effective, perhaps because sympathetic activation is or may be associated with the variations in anesthetic depth. Co-infusions of xylazine and ketamine have been used to extend short-term IV anesthesia.[12] Induction is accomplished through the use of standard techniques, and an infusion of xylazine (2.1 mg/kg per hour) and ketamine (5–7 mg/kg per hour) is begun. The technique has been used for durations of 70 minutes.

Guaifenesin recipes (combinations) are widely used to extend anesthesia beyond 20 minutes in the horse. Guaifenesin (5%) solution is combined with xylazine and ketamine to produce a solution commonly referred to as "triple drip," or "GKX."[13] "Triple drip" is formulated by adding 1000 to 2000 mg of ketamine and 500 mg of xylazine to a liter of 5% guaifenesin. The combination is administered to effect up to a rate of 2 mL/kg of body weight per hour. The combination produces excellent muscle relaxation and suitable analgesia. The degree of muscle relaxation and lack of movement are the best indicators of the depth of anesthesia. The quality of recovery is generally good if the anesthetic period is kept to less than 1 hour. "Triple drip" should not be used for anesthetics greater than 1 hour in duration unless oxygen supplementation and respiratory support is provided. Guaifenesin in combination with ketamine and detomidine can also be used to prolong field anesthesia.[14,15] The recipe uses 10% guaifenesin supplemented with detomidine (0.04 mg/mL) and ketamine (4 mg/mL), given to effect. The required rate of infusion approximates 0.6 to 0.8 mL/kg per hour (approximately one half of the "triple drip" rate). Recoveries are generally longer than after inhalant anesthesia but have been judged good to excellent. Midazolam has been substituted for guaifenesin in recipes with ketamine and xylazine to produce IV anesthesia.[16] Midazolam (50 mg), ketamine (1000 mg), and xylazine (500 mg) are added to 1 liter of isotonic fluids and administered at a rate of 2.2 mL/kg per hour. The combination is infused at a rate equivalent to "triple drip" (2.2 mL/kg per hour) and is similarly effective, in the author's experience. The concentration of ketamine in the recipes has been varied, with increased concentrations (2 mg/mL) recommended when particularly painful procedures are contemplated.[3]

The assumption of lateral recumbency at induction is associated with ventilation/perfusion mismatches that can cause hypoxemia. Horses positioned in dorsal recumbency have further deterioration of pulmonary function. Oxygen supplementation through insufflation or the use of a demand valve[a] is recommended, particularly when anesthesia is extended beyond 60 minutes.[17,18] Persons performing IV anesthesia routinely should consider the purchase of an oxygen tank and regulator to facilitate emergency oxygenation and ventilation of the patient.[19] Placement of an orotracheal tube helps to ensure a patent airway and facilitates the administration of oxygen or the delivery of assisted ventilation. Orotracheal intubation should be considered if there is risk of respiratory obstruction or regurgitation or if the horse is to be positioned with the neck in an abnormal position. Horses can be ventilated by adapting a nasogastric tube onto a pressure-reducing valve attached to an oxygen tank. The tube is inserted into one nostril and the nasal openings are momentarily occluded. The nostrils are released when the chest wall rises to a normal inspiratory level. The process is repeated until spontaneous ventilation resumes. The techniques described usually produce tolerable levels of cardiovascular depression, but, as anesthesia is extended, the importance of monitoring increases.

3. Recommendations

Equine anesthesia should not be undertaken lightly. The risk of morbidity or mortality in equine anesthesia is greater than that in the other commonly anesthetized domestic species.[2,16,17] A history must be obtained, a physical examination must be performed, and the results must be documented. The horse's body weight is estimated, and the amount of drug to administer is based on that weight. If significant fluid deficits are present, they should be corrected before anesthetic induction and supplemented during the anesthetic procedure. All drugs administered with the route and time of administration are recorded in the medical record. Indices of cardiac and respiratory function are recorded at least every 10 minutes during the anesthetic period, and results are recorded. The horse should be monitored until it returns to a standing position.

The assumption of lateral recumbency is associated with the development of ventilation-perfusion

mismatches and the shunting of blood through the lungs resulting in less than optimal oxygenation. This level of oxygenation is well tolerated for short periods, but oxygen supplementation should be considered if anesthetic periods greater than 30 minutes are anticipated. Induce anesthesia using drugs and techniques that are familiar to you. The intravenous combination of diazepam or midazolam and ketamine administered to horses fully sedated with xylazine or detomidine is useful. Once the horse is positioned for surgery (approximately 10 minutes after induction), an infusion of guaifenesin in combination with xylazine and ketamine at a rate of 2.2 mL/kg per hour is begun. The horse's respiratory rate, heart rate, and muscle tone are monitored, and the rate of infusion is adjusted up or down on the basis of the operator's assessment of anesthetic depth. The infusion is discontinued as the surgery is completed. The horse is rolled to lateral recumbency for recovery. The halter and attached lead rope are replaced.

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[a]Equine demand valve, JD Medical Distributing Co, Inc, Phoenix, AZ 85029–4914.

How to Sedate and Anesthetize the "Untouchable" Horse

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1. Introduction

Most practitioners have been presented, at some time or another, with an animal that has never been haltered or halter-broke and is in need of some type of veterinary care. This is a common occurrence in my area, and I have several ways that I deal with these types of horses. I will discuss methods on how I do this further, keeping in mind that the safety of both the horse and the human handler is always the first priority when dealing with this type of animal. There are a few variations in normal drug doses that I use on intractable horses, and these will be discussed. Keep in mind that most are based on trial and error and have served me well in my experience.[1–4]

2. Methods

When attempting to treat intractable animals that require sedation, the placement of a needle in the jugular vein can be extremely difficult. I use several methods to accomplish this.

First Method

The use of a broke, quiet, or gentle horse can be helpful when working on another horse that has not been halter-broke or handled. I put the problem horse in a sturdy stall or run and begin to introduce the gentle horse to him. The presence of a quiet animal tends to provide a calming influence. By placing the intractable horse between a wall or very sturdy fence and the gentle horse, I am usually able to get a needle into the jugular vein. The size of the needle to be used is often related to volume of medication to be administered and is an individual preference. I have no particular needle preference in this setting and often use whatever will get the job done safely and quickly at the time. It has been my experience that these animals react less to this approach than to any attempt at petting or rubbing. I try to get the needle placed with as little direct contact with the horse as possible. In most scenarios, with a little time, a jugular stick is possible. In some cases, however, I may have to use an intramuscular injection.

It is very important that the horse used is quiet and accustomed to being around other horses. This is not a good time to work with a young colt as an aid to sedate an intractable horse. Oftentimes, sedation is accomplished with no halter on the intractable horse, and a broke, quiet horse is an invaluable assistant.

Once I have sedated the problem horse, I will still use the broke horse as a "blocker" to halter the horse that I am working on. This technique will protect

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Table 1. Drugs and Dosages for Sedation

Drug	IV Dose (mg/kg)	IV Dose/450 kg	IM Dose (mg/kg)	IM Dose/450 kg
Xylazine	2–3	900–1500 (9–15 mL)	3–5	1200–2000 (12–20 mL)
Detomidine	0.03–0.04	15–20 (1.5–2 mL)	0.04–0.06	20–40 (2–4 mL)
Butorphanol	0.01–0.04	5–20 (0.5–2 mL)	0.02–0.05	5–25 (0.5–2.5 mL)

Note: Doses are considerably greater than those used in horses that are accustomed to interaction with humans.

from any sudden breaks in sedation until you can better control the animal with a halter and lead rope. Once the horse is haltered and sedated, I can usually work with them. Occasionally, I will use a saddled horse so that I can dally the lead rope of the problem horse to the saddle horn to help control the un-broke animal. If this is the case, be sure that the saddle horse is accustomed to having something large dallied on the saddle horn.

Second Method

In some cases, the problem horse must be roped. I only attempt this if I have a very sturdy stall or small pen and enough available time to complete the process. I prefer not to rope these horses, because this is a trainer's area more than it is mine. Often during this scenario, horses become so excited that they will override the effects of drugs quite easily. Still, in some cases, I have no other options and am often able to get a needle in the jugular vein this way. Again, a good saddle horse can sometimes be invaluable as a stabilizing and calming influence.

Third Method

The third method I will discuss is an approach that I use mainly with bucking horses. In the traditional sense, true "bucking horses" are by and large unable to be handled without proper chutes and alleys. The majority of these types of horses are owned by rodeo stock contractors, and in most instances, the equipment and help necessary to work on them is available. With the proper equipment and help, the job can be done safely and in a timely, efficient manner.

Bucking chutes are a must and extremely beneficial when castrating or working on the distal limbs of bucking horses. Unless dealing with a head or dorsal body wound, anesthesia will be required. These horses do benefit from a blindfold on occasion. A good, strong halter and lead rope are a necessary requirement.

Most bucking horses are accustomed to being haltered in the chutes, and once haltered, they can be restrained and allow a needle to be placed in the jugular vein. The chutes allow you to administer the sedation and induction drugs, but be careful not to get your head or arms in a position to be injured. Once sedation and induction have been achieved, you can then open the chute gate as soon as they start to go down. It is very important not to let them out of the chute too early because you probably will end up chasing a horse that is overriding the drugs given.

Again, with true "bucking horses," very few procedures can be performed without general anesthesia.

Drugs and Dosages

Drugs and dosages for sedation are given in Table 1.

Drugs and dosages for anesthesia are as follows.

Ketamine: 2 to 4 mg/kg IV (150–200 mg/45 kg (100 lb)

Diazepam: 0.1 mg/kg IV (40 mg (8 mL)/450 kg (1000 lb) horse—maximum, 8 mL

Guaifenesin Recipes (Triple Drip)

For adult horses, add 5 to 10 mL of xylazine (100 mg/mL) and 15 to 20 mL of ketamine (100 mg/mL) to 1 liter of 5% guaifenesin.

For young foals, add 5 mL of xylazine (100 mg/mL) and 10 mL of ketamine (100 mg/mL) to 1 liter of 5% guaifenesin.

Yearlings and mature horses require higher doses of xylazine and ketamine.

3. Discussion

These are by no means the only ways to work with an intractable horse; however, they represent what has worked best thus far in my experience. I try, first and foremost, not to get myself or anyone else injured and to be as safe as possible with the horses. You must be able to adapt the doses of the drugs from case to case—the more intractable animals require much larger doses of drugs to achieve the same result as a quiet animal. The most intractable horses will also require close monitoring of sedation or anesthesia levels.

Keep in mind that these animals are highly reactive to external noise and stimuli. Be quiet and get the job done. These animals wake up quickly. It is best to leave the eyes covered and let the horse lay as long as possible in an area that is safe for it to recover. I often remove the halter and lead rope before the horse wakes up because any type of human assistance during recovery usually makes the situation worse.

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Risk Factors Associated With Gastrointestinal Dysfunction in Horses Undergoing Elective Procedures Under General Anesthesia

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Gastrointestinal dysfunction occurs commonly after elective anesthesia. Identification of risk factors may allow implementation of preventative measures to reduce the prevalence of colic after elective anesthetic procedures. Authors' address: Department of Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO 80523; e-mail: Brad.Nelson@colostate.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The purpose of this retrospective, case series study was to examine risk factors associated with post-anesthetic colic (PAC) or gastrointestinal dysfunction for horses undergoing elective surgical or diagnostic procedures under general anesthesia.

2. Materials and Methods

Medical records were collected from adult horses undergoing general anesthesia from January 1, 2008, to December 31, 2010, through the use of a retrospective cohort design. Potential risk factors were examined by means of univariable logistic regression with a limit of a $P < 0.25$. Through backward elimination, the final multivariate model was created to determine variables significantly associated with gastrointestinal dysfunction.

3. Results

Colic or delayed fecal output was reported in 36 of 416 (8.7%) horses undergoing general anesthesia in the study period. In the final multivariable model, horse breed ($P = 0.05$), peripheral blood lactate ($P = 0.02$), right lateral recumbency during general anesthesia ($P = 0.04$), post-anesthetic rectal temperature ($P = 0.03$), and hours to first passage of manure ($P < 0.01$) were statistically significant between horses that exhibited colic compared with those that did not exhibit colic. Arabians were more likely to have colic compared with other horse breeds (4/13, 30.8%). Horses that exhibited gastrointestinal dysfunction passed manure later than those that did not exhibit colic (7.2 ± 0.8 hours and 5.4 ± 0.2 hours, respectively). As blood lactate increased at the end of anesthesia, the odds of colic also increased (odds ratio, 1.4; 95% confidence interval, 1.04–1.83, $P = 0.02$).

4. Conclusions

This study demonstrated the prevalence of gastrointestinal dysfunction in horses undergoing elective general anesthetic procedures in our hospital population. Arabian horses, increasing blood lactate levels, and delayed passage of feces were signifi-

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cantly associated with an increased risk of gastrointestinal dysfunction. These results will help identify horses undergoing anesthesia that are at increased risk for colic and may allow implementation of preventative measures.

Acknowledgments

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Acute-Phase Proteins as Diagnostic Markers in Horses With Colic

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1. Introduction

The objective of this study was to investigate the ability of the acute-phase proteins serum amyloid A, haptoglobin, and fibrinogen to differentiate between horses with infectious nonsurgical colic and surgical colic.

2. Materials and Methods

The performance of the acute-phase proteins was evaluated individually and in combination with clinical examination and with traditional biomarkers in blood (white blood cell count, packed cell volume [PCV], total plasma protein [TPP], lactate) and peritoneal fluid (hemolysis, white blood cell count, total protein).

Admission data collected prospectively from 148 horses with severe colic in one hospital were used to construct multivariate logistic models to predict if a horse had an infectious nonsurgical colic. The models were based on 1) clinical evaluation, 2) clinical and blood evaluation, and 3) clinical, blood, and peritoneal fluid evaluation. Each model was independently validated against admission data from 78 horses in another hospital.

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3. Results and Discussion

The variables included in the final clinical model were lethargy, temperature increase from 38°C, gastric reflux 5 to 10 L, and normal rectal findings. All variables except gastric reflux 5 to 10 L were positive predictors of infectious colic. Diagnostic specificity and sensitivity was 98% and 57%, respectively. When serum amyloid A concentration in serum was added to the model on the basis of clinical evaluation, the specificity and sensitivity improved to 98% and 64%, respectively. No additional blood or peritoneal fluid variables improved the model significantly. The models had a satisfying integrity and diagnostic performance when validated.

Postoperative Complications After Colic Surgery in Geriatric Versus Mature Non-Geriatric Horses

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1. Introduction

Many believe that geriatric horses have prolonged, complicated recoveries from colic surgery. Studies have evaluated survival rates in geriatrics undergoing colic surgery or used age in multivariable analyses when evaluating specific postoperative complications (eg, postoperative reflux [POR]); however, there have been no comprehensive studies, to the authors' knowledge, comparing such complications or associated outcomes in geriatric (≥20 years) and mature (4–15 years) horses.

2. Materials and Methods

Medical records were evaluated for horses age ≥20 years (geriatric, $n = 78$) and 4 to 15 years (mature, $n = 156$) surviving ≥24 hours after colic surgery from 2000 to 2010. Postoperative complications included POR, diarrhea, fever, leukopenia, incisional infection, incisional dehiscence, thrombophlebitis, colic, re-laparotomy, pneumonia, and laminitis. Short-term outcomes of horses with complications were compared. Data were analyzed by means of a χ^2 test or an analysis of variance. The level of significance was $P < 0.05$.

3. Results

For most complications, proportions of geriatric and mature horses with complications and short-term outcome were similar.

A higher proportion of geriatrics had a small intestinal strangulation and small intestinal resection and anastomosis, leading to an overall higher proportion with development of POR. There was no difference, however, in the proportion of geriatric versus mature horses with a small intestinal strangulation with development of POR.

A higher proportion of geriatrics was inappetent after surgery, which remained significant for horses with small intestinal strangulations.

4. Discussion

Geriatric horses have colic surgery recoveries, in terms of short-term outcome and postoperative complications, similar to mature horses, a useful finding when discussing surgical options with owners.

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Comparison of Laparoscopic and Conventional Cryptorchidectomy on Rate of Intra-Operative and Postoperative Complications, Length of Surgery and Anesthesia, and Hospital Stay

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Horses undergoing laparoscopy for cryptorchidism had increased anesthesia and surgery time and more intra-operative and postoperative complications. Further work is needed to assess the relative invasiveness of the procedures. Authors' address: Department of Clinical Studies, Ontario Veterinary College, University of Guelph, Guelph, ON, Canada N1G 2W1; e-mail: ncribb@uoguelph.ca. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Advantages of laparoscopy include superior visualization, less surgical morbidity, and faster return to work. Disadvantages include expense of equipment and familiarity with techniques. To our knowledge, no previous study has compared intra-operative and postoperative surgical parameters between conventional surgery and its laparoscopic equivalent in horses.

The objective was to perform a case-control study to determine any differences in intra-operative and postoperative complications associated with laparoscopic cryptorchidectomy compared with conventional surgery and if surgery time, anesthesia time, and length of hospital stay are different.

2. Materials and Methods

Thirty horses that underwent laparoscopic cryptorchidectomy were matched with 30 control horses that had open conventional surgery. Horses were matched according to history of previous surgery, location of testicles, and type of closure after removal of scrotal testicles. Length of surgery time, anesthesia time, duration of hospital stay, and intra-operative and postoperative complications were compared.

3. Results

Significantly more postoperative complications were encountered in horses undergoing laparoscopy ($P = 0.03$), and a tendency for more intraoperative complications was noted (0.1). Horses that had laparoscopic surgery had a significantly longer anesthesia time ($P < 0.01$) and surgery time ($P < 0.01$). No statistically significant difference was seen for the duration of hospitalization ($P = 0.4$).

4. Discussion

The advantages of laparoscopy may not always outweigh the disadvantages in horses requiring surgery for cryptorchidism. Further work is required to better determine the relative invasiveness of the two procedures.

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Long-Term Outcome After Laser-Assisted Modified Forssell's in Cribbing Horses

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The laser-assisted modified Forssell's procedure is a very effective (84.4%) surgical treatment of cribbing behavior in horses. Authors' address: Equine Health Studies Program, Department of Veterinary Clinical Sciences, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA 70803; e-mail: djburba@vetmed.lsu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The objectives of this study are to report on the outcome of long-term follow-up (range, 1–18 years; median = 6) after laser-assisted modified Forssell's procedure (LARMF), to describe the prevalence of postoperative complications, and to identify risk factors associated with outcome.

2. Materials and Methods

Medical records of horses undergoing LARMF between 1994 and 2012 were reviewed. Signalment, preoperative duration of cribbing, postoperative complications, and outcomes were recorded. Logistic regression analyses were used to analyze the data.

3. Results

Follow-up was available in 90 of 119 horses; 76 horses stopped cribbing for more than a year. Of the 14 horses that resumed cribbing, the median time to cribbing relapse was 6 months. The preoperative duration of cribbing was significantly associated with an increased probability of unsuccessful outcome when horses cribbed for more than 3 years before surgery (odds ratio of positive outcome, 0.12). Postoperative complications reported in 20 of 88 included hematoma (n = 3), seroma (n = 3), infection (n = 6), and prolonged drainage (n = 6) and dehiscence (n = 2) and were also associated with a decreased probability of obtaining a successful outcome (odds ratio, 0.22). Quarter Horse and Warmblood breeds were more likely to be presented for cribbing surgery than for other conditions when compared with other breeds.

4. Conclusions

The LARMF is a very effective (84.4%) surgical treatment of cribbing behavior in horses. Preoperative duration of cribbing and the postoperative complications were identified as significant risk factors associated with unsuccessful surgical outcome, which has not been reported previously.

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How to Manage Axillary Wounds

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1. Introduction

Axillary wounds are common in horses and often result from a traumatic insult, which may include running into a stationary object or being impaled by a sharp object, such as a fencepost or stick; or kick injuries from other horses.[1] At first, these wounds may appear to be relatively minor; however, they must be monitored carefully because they can result in severe complications. The most common complication is the development of subcutaneous emphysema. The importance of subcutaneous emphysema is that it can lead to pneumomediastinum and eventually pneumothorax, which can be life-threatening. Infection is another complication of these wounds.[2] Initial management of axillary wounds can affect what complications may develop.

2. Materials and Methods

Clinical Assessment

Clinical assessment begins with evaluation of the general condition of the horse and a thorough physical examination, including temperature, heart rate, respiratory rate, intestinal borborygmi, and evaluation of mucous membranes. The patient should then be sedated for full evaluation of the wound. After cleaning the surface of the wound with warm water and povidone iodine solution, sterile gloves should be worn to palpate the wound. The depth and direction of the wound should be determined. Potential involvement of the elbow joint, cranial mediastinum, and cranial thorax should be evaluated.

Treatment

Once the extent of the wound has been determined, it should be cleaned, debrided, and lavaged. The overuse of high-pressure lavage should be avoided to prevent further dissemination of contaminants into deep fascial planes.[2] Methods to lavage the wound include the use of a catheter-tipped syringe or a 35-mL syringe with an 18-gauge needle on the end (Fig. 1). Lavage should be performed with povidone iodine solution diluted in water. After the wound is cleaned, a sterile gauze or laparotomy sponge should be packed into the wound. Packing the wound is important because it will prevent air from migrating into the subcutaneous fascial planes. At this point, an initial attempt at partial primary closure to appose the edges of the wound may be attempted; however, dehiscence should be expected. The closure can be performed in two layers: the first layer should attempt to close any muscle and fascial layers and the second layer to close the skin. An opening in the wound closure should be left for the sterile gauze packing to be changed. After partial primary closure is performed, stay sutures can be placed with non-absorbable suture material to create a lattice, to shoe-string–tie a stent in place over the wound. After the stent is place, a "figure 8"

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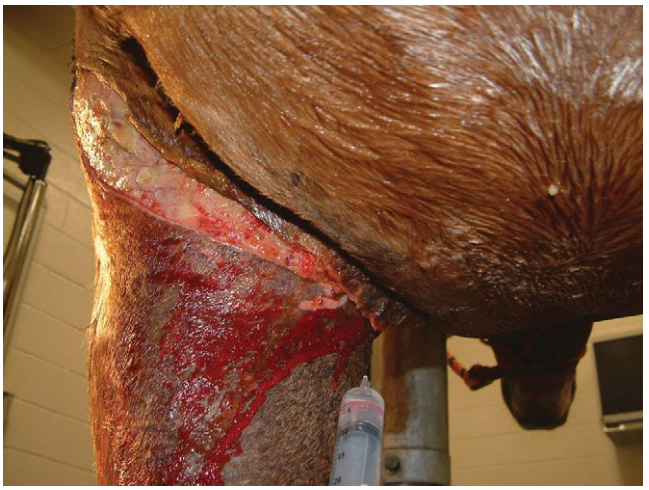


Fig. 1. Caudoventral-craniodorsal view of a horse with a wound in the left axilla. A 35-mL plastic syringe with an attached 18-gauge needle is used to irrigate the wound with sterile saline.

bandage should be placed around the thorax and axillary region to keep the stent in place and covered (Fig. 2).

The horse should be administered flunixin meglumine (1.1 mg/kg, q 12 h, IV) and broad-spectrum antibiotics. A tetanus toxoid should also be administered. To help avoid progressive accumulation of air within the soft tissue, limiting movement is indicated. Therefore, the horse should be on strict stall rest until the wound is healed. The horse can be allowed to lie down, but if he rolls or is very active in the stall he should be cross-tied to prevent excessive movement.

The bandage should be changed every 24 to 48 hours or as needed, on the basis of the amount of drainage and condition of the bandage. The wound should be lavaged and cleaned at each bandage change and new sterile gauze packing inserted into the wound. To promote granulation tissue formation, topical medications can be applied to the gauze before it is packed into the wound. Additionally, 15 to 30 mL of procaine penicillin G (300,000 IU/mL) can be applied to the gauze to provide local administration of antibiotics.



Fig. 2. Horse with an axillary wound that has been cleaned, packed, and sealed. A stent and thoracic bandage have been applied to hold the packing in place.

Secondary Complications

Subcutaneous emphysema may develop as a secondary complication. Subcutaneous emphysema may not be present on initial evaluation of the horse; it develops as air progressively accumulates in the subcutaneous tissue as the wound acts as a one-way valve. Once the source of the subcutaneous emphysema has been addressed, residual subcutaneous emphysema is usually self-limiting and rarely requires additional treatment. However, if the subcutaneous emphysema is extensive and the risk of complications from the amount of subcutaneous emphysema is high, small intravenous catheters may be inserted subcutaneously to aspirate some of the air.[3] Inserting subcutaneous catheters is not without its own potential complications (ischemia of overlying skin, local infection); therefore, this technique should only be performed when deemed necessary to prevent complications from subcutaneous emphysema. Additionally, the horse's temperature should be monitored because the subcutaneous emphysema has an insulation-like effect. At warmer times of the year or in warm climates, there is a potential for serious hyperthermia.

When subcutaneous air dissects through the muscle layers and fascial planes into the mediastinum, pneumomediastinum ensues. A diagnosis of pneumomediastinum is based on radiographic findings. Otherwise, pneumomediastinum in the horse is often clinically silent and there is no specific treatment beyond treatment of the underlying cause. Cases should be monitored for complications including pneumothorax, impairment of venous return to the heart, and rupture of the mediastinal pleura, which has been reported in humans but is yet to be reported in horses.[4]

If pneumomediastinum progresses, it can result in pneumothorax and specifically a tension pneumothorax. Tension pneumothorax occurs when a flap of skin or soft tissue acts as a one-way valve, allowing air into the cavity on inspiration but preventing its escape on expiration. This is a rapidly progressive form of pneumothorax that classically consists of progressive respiratory distress, tachycardia, hypotension, and absent lung sounds. Delays in the development of pneumothorax are commonly encountered; therefore, serial evaluations should be performed to identify progressive changes in pulmonary function. Radiographs or ultrasound can be used to confirm the diagnosis. However, when subcutaneous emphysema is present, ultrasound has limited diagnostic value to evaluate a pneumotho-



Fig. 3. Horse with 24F chest trocar[a] and Heimlich valve placed in left caudodorsal thorax at the ventral level of the tuber coxae between the 13th and 14th ribs.

rax. Pneumothorax can be unilateral or bilateral. The development of bilateral pneumothorax should be considered because the mediastinum of horses is generally described as incomplete, having small fenestrations in the caudal and ventral portions of it.[1] Diagnostic radiographic features of bilateral pneumothorax include complete absence of pulmonary parenchyma and hyperlucency in the caudodorsal lung fields and retracted dorsal margins of both right and left lung lobes.[1] Diagnostic features of unilateral pneumothorax include retraction of one lung margin and visualization of the vessels in the contralateral lung.[5] Ultrasonographic diagnosis is based on a static, hyperechoic line that does not move with the respiratory cycle. The presence of subcutaneous emphysema may limit or prevent ultrasonographic examination because the ultrasound waves will not penetrate the subcutaneous air.

Pneumothorax that is causing respiratory distress or is severe on radiography should be treated. Aspiration of air from a dorsally located chest trocar in the 12th to 15th intercostal space is therapeutic. The chest trocar is placed midway between two adjacent ribs at the level of the ventral aspect of the tuber coxae (Fig. 3). Intravenous catheters may also be used to aspirate air from the thorax. The decision to use a catheter versus a chest trocar is based on the clinician's expectation of the pneumothorax resolution. A catheter is appropriate if the clinician expects the pneumothorax to resolve after aspirating the air one time, but, if not, a chest trocar is more appropriate because it can be sutured in place and used for repeat or continuous aspiration. Furthermore, if the pneumothorax does not resolve with passive aspiration of air through a chest trocar, suction may be applied. If suction is used, a low negative pressure is preferable to minimize re-expansion pulmonary edema/hemorrhage; alternatively a system that uses the three-bottle technique[a] should be used. Removal of the chest trocar is based on the clinical condition of the horse and radiographic evidence of pneumothorax resolution. Pneumothorax may be treated conservatively if the horse does not show signs of respiratory distress[6] and there is a mild pneumothorax on radiographic evaluation.

3. Results

Medical records from seven horses with a diagnosis of an axillary wound that were examined at Texas A&M University Veterinary Medical Teaching Hospital (VMTH) were reviewed. Ages ranged from 8 months to 16 years and included four geldings, one mare, one colt, and one stallion. All seven cases presented to the hospital with subcutaneous emphysema. The time between wound occurrence and development of subcutaneous emphysema was able to be determined in five of seven cases (3.2 ± 0.84 days; range, 2–4 days). In the remaining two cases, the development of subcutaneous emphysema was recorded as "progressive." Radiographs were taken in five cases. Pneumomediastinum was diagnosed in three cases and suspected in two cases. A bilateral pneumothorax was diagnosed in three cases and a unilateral pneumothorax was diagnosed in one case. Treatment at the VMTH was similar across all cases. All wounds were initially cleaned, explored, packed, and bandaged. All patients received nonsteroidal anti-inflammatories (NSAIDs) and antibiotics. The antibiotics that were used included trimethoprim sulfa (30 mg/kg, q 12 h, PO), procaine penicillin G (22,000 IU/kg, q 12 h, IM) in combination with gentamicin (6.6 mg/kg, q 24 h, IV) and chloramphenicol (50 mg/kg, q 6 h, PO). Phenylbutazone (2.2 mg/kg, q 12 h, PO) and flunixin meglumine (1.1 mg/kg, q 12 h, IV) were the NSAIDs chosen for pain management. Treatment to relieve subcutaneous emphysema was performed in two cases. In one case, 14-gauge hypodermic needles were inserted subcutaneously over the shoulders bilaterally to release air, and, in the other case, incisions were made lateral to the withers to allow air to escape from the subcutaneous space. Three of the four cases with pneumothorax were treated by aspiration of the air from the pleural cavity. In each of these cases, chest trocars were placed bilaterally in the caudodorsal thorax. All patients survived to discharge.

4. Discussion

An important finding of this study is recognition that there is a repeatable association between equine axillary wounds and the development of secondary complications. These complications include the development of subcutaneous emphysema, pneumomediastinum, and pneumothorax. On the basis of the results of this study, subcutaneous emphysema develops to a clinically significant extent approximately 3 days after injury and progresses extensively if the primary cause is not treated.

Another important finding of this study is that optimal treatment of axillary wounds should include packing and sealing the wound with a sterile laparotomy sponge or gauze that is changed daily until healing by second intention occurs. Primary closure is insufficient to prevent secondary complications. Restriction of movement and close monitoring are necessary to limit the occurrence and severity of complications.

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[a] Pleur-evac, Chest Drainage System, Teleflex, Research Triangle Park, NC 27709.

Use of Silver Sodium Zirconium Phosphate Polyurethane Foam Wound Dressing on Wounds of the Distal Forelimb

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Application of a silver sodium zirconium phosphate polyurethane semi-occlusive foam wound dressing improved measures of wound healing compared with a control dressing. Authors' address: 1202 Calle Maria, San Marcos, CA 92069; e-mail: maurkell@gmail.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The SPF dressing is a semi-occlusive polyurethane foam, impregnated with an ionic silver exchange resin and antimicrobial dyes proposed to be useful in the management of open wounds. The objective of this study was to determine if the SPF dressing wound improved second-intention healing of experimental wounds of the distal forelimb in horses.

2. Materials and Methods

A full-thickness 6.25-cm^2 wound was created on each metacarpus, with one limb for treatment and control. During the study period, granulation tissue was graded, wound area was calculated, and granulation tissue was cultured. Wound areas and granulation scores were analyzed with the use of a mixed-effects linear regression model. Time for complete wound healing was compared with the use of a log-rank test. Significance levels were set at $P < 0.05$.

3. Results

SPF wounds had significantly decreased wound area ($P = 0.035$) and decreased granulation tissue scores ($P = 0.010$), although healing times were not significantly different. Bacterial contamination was seen on all wounds at varying times throughout the study period.

4. Discussion

The proposed mechanism of action for the SPF dressing is release of antimicrobial elements to the wound, reduction of contamination, and absorption of microbial products. The SPF dressing was associated with significantly improved measures of wound healing in this experimental model. Further studies are needed to document the benefit of the SPF dressing in clinical equine wounds.

Research Abstract

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Regional Limb Perfusion of Amikacin Sulfate Alone and in Combination With Ticarcillin/Clavulanate in Horses

Ashlee E. Watts, DVM, PhD, Diplomate ACVS*; Alanna J. Zantingh, DVM; Wayne S. Schwark, DVM, PhD; and Susie L. Fubini, DVM, Diplomate ACVS

The results of our study indicate that antimicrobial activity of amikacin is reduced when used for regional limb perfusion with ticarcillin and that this combination should be avoided in regional limb perfusion when optimal amikacin activity is desired. Authors' addresses: Department of Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX 77845 (Watts); Murdoch University Veterinary Hospital, 90 South Street, Murdoch, WA 6150 Australia (Zantingh); Department of Molecular Medicine (Schwark) and Department of Large Animal Clinical Sciences (Fubini), College of Veterinary Medicine, Cornell University, Ithaca, NY 14853; e-mail: awatts@cvm.tamu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The purpose of this study was to determine the effect of regional limb perfusion with amikacin alone and in combination with ticarcillin/clavulanate on synovial fluid concentration and antimicrobial activity of amikacin.

2. Materials and Methods

Regional limb perfusion with amikacin alone (group A: 2.5g) or amikacin and ticarcillin/clavulanate (group AT: 2.5 g of amikacin, 7 g of ticarcillin/clavulanate) was performed with a tourniquet placed at mid-antebrachium in sedated horses. Perfusate blood was collected immediately after injection and again before tourniquet release. Blood from the jugular vein was collected before tourniquet release. Synovial fluid from the middle carpal joint was collected at 0, 30, and 60 minutes after tourniquet release. Amikacin concentration and antimicrobial activity of synovial fluid against amikacin- and ticarcillin-resistant and susceptible cultures were determined.

3. Results

There was significantly lower amikacin concentration in the middle carpal joint synovial fluid in group AT compared with group A at 30 minutes (group AT: median, 4.4 μg/mL; group A, 17.5 μg/mL) and 60 minutes (group AT: median, 4.6 μg/mL; group A: 15.0 μg/mL) after tourniquet release. The zones of inhibition against ticarcillin-resistant *Klebsiella pneumoniae* from group AT were significantly smaller than group A from synovial fluid at 30 minutes and 60 minutes and in the perfusate serum before tourniquet release.

Research Abstract

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4. Conclusions

The combination of amikacin with ticarcillin/clavulanate during regional limb perfusion resulted in significantly lower amikacin synovial concentration and antimicrobial activity on amikacin-susceptible and ticarcillin-resistant cultures compared with amikacin alone.

This study was approved by the university's institutional animal care and use committee.

How to Perform Diagnostic Stifle Arthroscopy With the Use of a Needle Arthroscope in Standing Horses

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1. Introduction

It has been suggested that athletic injuries to the stifle may account for >40% of injuries in certain disciplines.[1] In the authors' experience, the prevalence of stifle injuries in most disciplines is not accurately known. Furthermore, until the past 10 to 15 years, very little attention has been given to athletic injuries in the stifle, in part because of diagnostic limitations and lack of successful treatment options.

Limiting definitive diagnosis is the inability to accurately image the soft tissue and bone with high sensitivity and specificity. Although routine radiography can be helpful in certain cases, many lesions, particularly those of soft tissues, go undetected. Ultrasonographic examination of the equine stifle joint has helped broaden diagnosis of stifle lesions. This is especially true of soft-tissue structures, namely the collateral ligaments, patellar ligaments, and menisci. Ultrasound examination of the cruciate ligaments still poses a problem because of the orientation and deep anatomic location of these structures. Moderate to severe pathology appears to be readily detected with the use of ultrasonography,[2,3] but detection of slight to moderate lesions is difficult. In a recent study, Cohen et al[4] nicely defined the sensitivity (79%) and specificity (56%) of ultrasonography in the equine stifle joint, with surgery used as the gold standard. A positive predictive value (73%) and negative predictive value (62%) were also determined for equine ultrasonography on the basis of this study. These results point out the current limitation of ultrasonography of the equine stifle. Other current methods for diagnostics in the stifle currently require general anesthesia, specifically computed tomography, magnetic resonance imaging, and routine arthroscopy, which remain the gold standards. It is also worth noting that these later diagnostics are typically only available at large referral centers, limiting their availability in a more routine practice setting.

Additional methods to safely and quickly assess the stifle in a typical practice setting would be advantageous to aid a more accurate diagnosis of stifle

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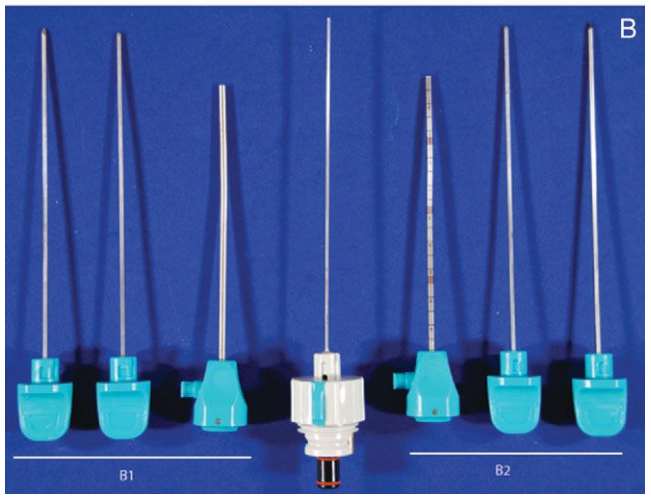


Fig. 1. A, Integrated fiber optic xenon light source, 6.4-inch high-resolution LCD monitor and light cable/480-line high-resolution camera; B, 18-gauge, flexible arthroscope; B1, sharp and blunt obturator and stiff cannula; B2, sharp and blunt obturator and standard cannula.

disease. This report outlines the use of an 18-gauge, disposable arthroscope to safely and efficiently provide a complete visualization of the stifle joint in the standing horse.

2. Materials and Methods

Equipment

The 18-gauge needle arthroscope[a] (1.3-mm diameter) is a compact and portable unit that consists of a light source and imaging processor in one console and a camera attached to a cable, which connects with the base console (Fig. 1). The standard 100-mm-long arthroscope and cannula/obturator (2.0-mm outer diameter, OD) systems are disposable and come in a 10° and special-order 30° configuration. A separate, stiffer cannula/obturator (2.5-mm OD) with a 30° scope lens system is available from the manufacturer and was used in all clinical cases (Fig. 1). Fluid

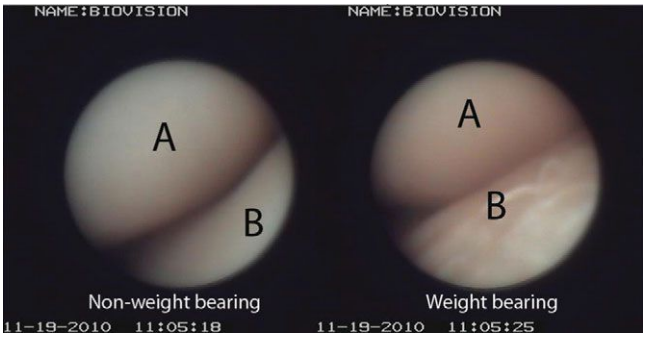


Fig. 2. Comparison of the cranial aspect of the medial femorotibial joint in non–weight-bearing and weight-bearing positions in the same horse. The configuration of the medial meniscus changes considerably when the joint is being loaded. This figure demonstrates the medial femoral condyle (A) and medial meniscus (B).

distention of the joints is necessary and may be achieved through either the use of a 60-mL syringe, a fluid pressure bag on a 1-L fluid bag, or an automated pressure-sensitive arthroscopic fluid pump system. The fluid used to distend the joint during surgery should have 200 mL of 2% lidocaine/mepivacaine added per liter of fluid.

Patient Preparation

Patients are typically given 2 g of phenylbutazone before surgery and then 2 g daily for three additional postoperative days. Throughout the procedures, horses are administered light to moderate sedation; they are also typically restrained with a nose twitch and in some cases with the use of stocks. Horses are routinely sedated with 10 mg of detomidine intramuscular at the time of initial stifle preparation. The joints to undergo the procedures are blocked separately with the use of approximately 20 to 30 mL of local anesthetic per joint, optimally at least 20 minutes before the onset of the procedure. Next, depending on the amount of organic debris and hair length, the stifle area is clipped with a No. 40 clipper blade and prepared in an aseptic manner as for routine arthroscopic surgery. In some horses that have very short coats, the hair is not clipped. Just before local skin and tissue anesthesia, the horses are typically given 3 mg of detomidine and 5 mg of butorphanol intravenously. Approximately 5 to 10 mL 2% mepivacaine hydrochloride is used to block the skin and deeper tissues at each of the entry portals described in the next section. An additional aseptic preparation of the portal sites follows, with final positioning of the limb. The team typically consists of a surgeon and assistant wearing sterile gloves as well as an assistant to position and help balance the hind limb. The procedure can and has been performed with or without stocks.

The preferred limb positioning is flexed similar to standard arthroscopy.[3] In refractory horses, the

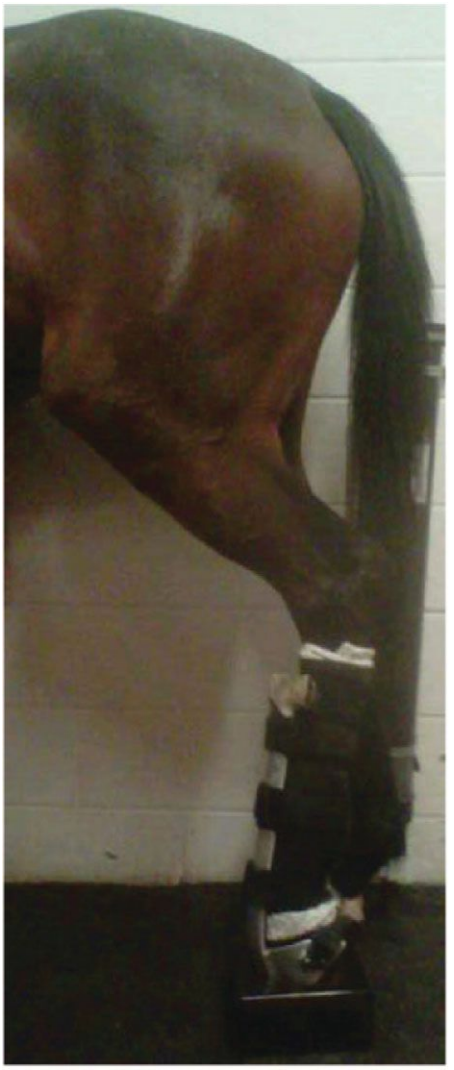


Fig. 3. Mobile limb-positioning device.

stifle joints can be entered in a weight-bearing position, but the surgeon must be aware that this changes the shape and position of soft-tissue structures, most notably the appearance of the menisci (Fig. 2). If the limb is entered in a weight-bearing position (minority of the cases), the limb is flexed manually for a short period to visualize the more distal extent of the condyle.

A customized stand is used to hold the limb in flexion (Fig. 3). With the use of this technique, the distal limb is bandaged with a quilt and bandage, then placed in a splint.[b] A custom base[c] has been manufactured to accept the splint and allow for variable flexion of the limb on the basis of how high the base is from the ground (Fig. 3).

At the end of the procedure, either 125 mg of amikacin sulfate or 600 mg of ceftiofur sodium is administered into the joint undergoing needle arthroscopy. The small size of the skin incisions indicates that no suture or other method of closure is necessary; however, the authors have used tissue glue in cases in which bleeding was an issue. Horses are normally stall-confined and observed twice daily for 3 days, and the level of work appropriate to the arthroscopic findings is then outlined.

Portal Placement

A stab incision just large enough to introduce the sharp tip of the trocar can be made with the use of a No. 15 or No. 11 blade. However, the authors have more recently used the sharp trocar to introduce the stiff arthroscopic cannula through the skin and soft tissues before advancement of the cannula into the joint with the use of the blunt obturator. Distention of the joint is not necessary before cannula placement; this is aided by the previously administered 20 to 30 mL of local anesthetic. Additionally, the caudal aspect of the medial femorotibial joint is usually done after the cranial compartment, thus providing some caudal distention, which the authors believe aids entrance.

The cranial compartment of the medial femorotibial (MFT) joint is assessed from a standard approach lateral to the lateral patellar ligament and then from a cranial approach between the lateral and middle patellar ligaments[3] in either flexed or extended limb positions (Fig. 4A). The lateral approach optimizes visualization of the cranial ligament and axial portion of the medial meniscus. The cranial approach allows easier visualization of the intercondylar area (cruciate ligaments) and the medial collateral ligament (Fig. 5).[5] The caudal approach to the MFT uses the description of Trumble et al[6] and can be used in both flexed and weight-bearing positions[3] (Fig. 4). The authors' approach of choice to the cranial compartment lateral femorotibial (LFT) joint is that first described by Moustafa et al,[7] with the use of the previous lateral portal (lateral to the lateral patellar ligament) to the cranial MFT joint, although the portal for the cranial MFT joint also can be used[3] to help visualize more lateral structures. The caudal pouch of the LFT joint is entered through an approach that is 2.5 cm proximal to the tibial plateau and 3 cm caudal to the lateral collateral ligament; the authors have only performed this in the flexed position. The femoropatellar joint is entered through a standard craniolateral approach with the limb extended.[3]

A combination of these approaches allows a complete examination of the stifle. It should be noted that all movements of the cannula around the joint should be made by placing pressure on the cannula itself and not on the scope or camera because these are prone to breakage (Fig. 6). The authors have not experienced this and typically use a single scope for four to five procedures or until visible damage to the scope is observed.

3. Results

The proof of principle for this technique has been confirmed and included diagnostic examination of

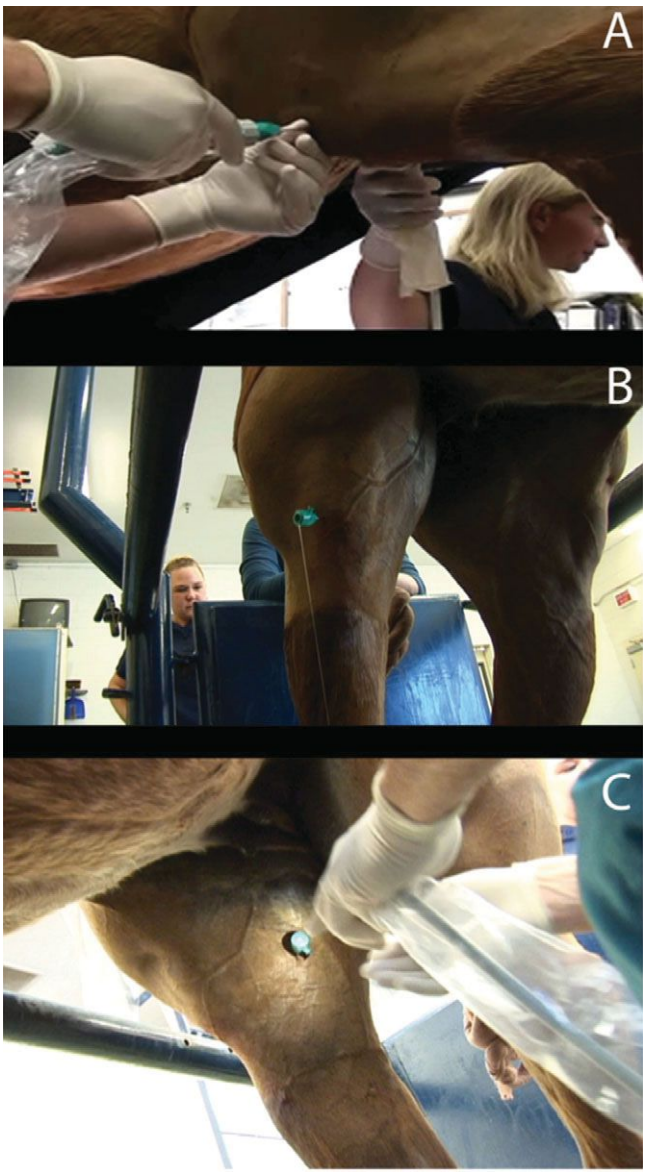


Fig. 4. A, Simultaneous diagnostic arthroscopy and ultrasonographic examination in a standing horse with the operated limb flexed. B, Synovial fluid draining from a stiff (2.5-mm) cannula inserted in the cranial compartment of the MFT joint from a lateral portal in a standing horse. C, Cannula placement in the caudal compartment of the MFT joint in a standing horse.

cadaver stifles followed by examination in normal stifles of standing horses.[8]

To date, the authors have identified a broad range of lesions with the use of the needle scope. These include full-thickness cartilage erosion of articular cartilage (Fig. 7) and horizontal articular cartilage lacerations, an axial lesion of the medial meniscus (Fig. 8), and a loose body identified in the intercondylar area (undetected on radiographs and ultrasound) (Fig. 9) have been seen. Other syndromes include a horizontal meniscal lesion (in a weight-bearing stifle) (Fig. 10) and tearing of the cranial

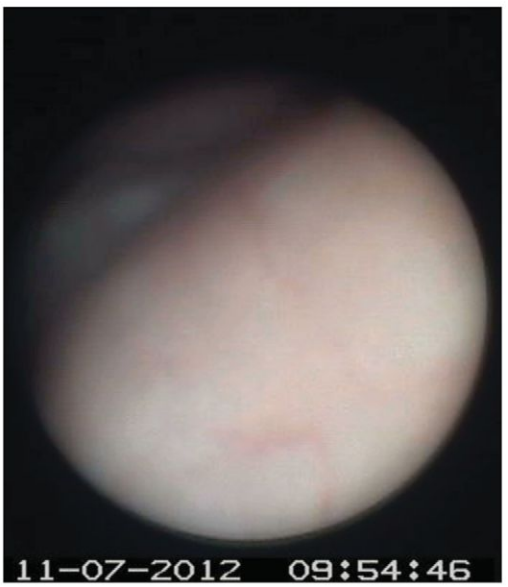


Fig. 5. View of a normal cranial cruciate ligament just caudal to the attachment at the medial tibial eminence as it courses through the intercondylar notch area.

ligament of the lateral meniscus (Fig. 11). A vertical radial tear of the cranial horn of the medial meniscus was identified in a weight-bearing stifle. The severity of the lesion could not be fully appreciated during the standing procedure. The lesion was confirmed through standard arthroscopy in which the full extent of the tear was detected by probe manipulation. In cases in which therapeutic arthroscopy (under general anesthesia with the use of

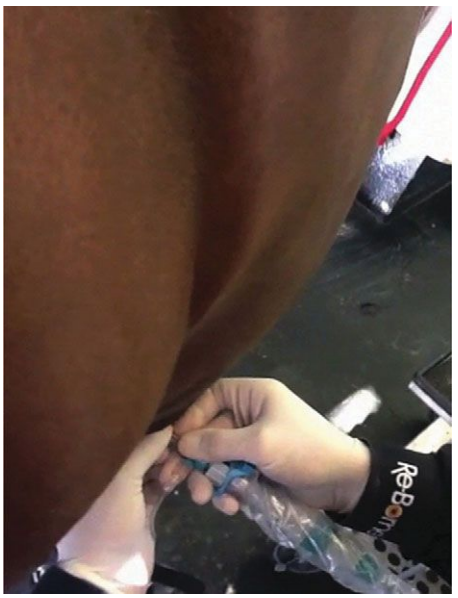


Fig. 6. Correct hand placement on the cannula, which is used to manipulate the scope in the joint. No torque is placed on the scope or camera.

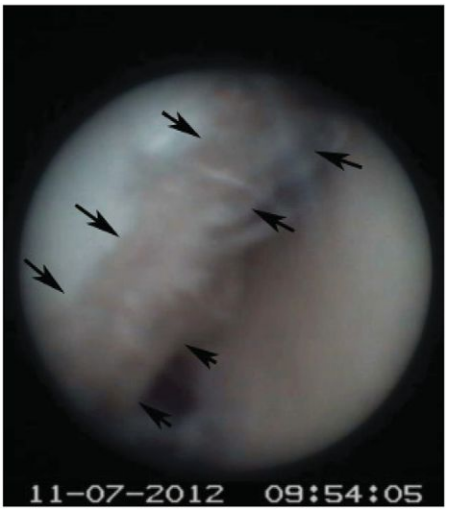


Fig. 7. Full-thickness erosion on the medial condyle (outlined by black arrows) just adjacent to the medial tibial eminence (right side).

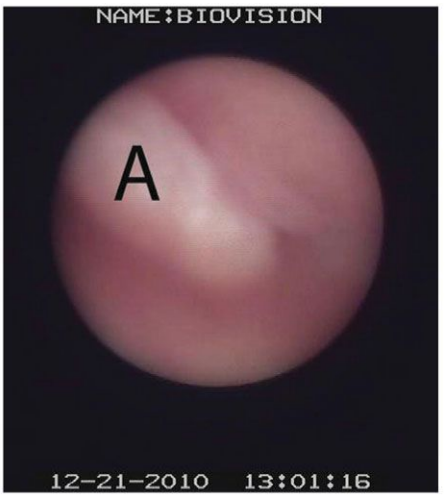


Fig. 9. Arthroscopic view of loose osteochondral fragment (A) in the intercondylar area.

a standard 4-mm arthroscope) has been indicated, on the basis of the diagnostic evaluation with the use of the needle arthroscopy standing, abnormalities identified have been similar with both techniques. Although the authors have not specifically recorded procedure time on the basis of the length of video captured during the procedure, the time in the joint is estimated as 4 to 10 minutes per joint. The preparation time is estimated at approximately 30 minutes.

4. Discussion

To date, the authors have found this procedure to be well tolerated by most horses, and no morbidity has been ascribed to the procedure (90+ stifle joints). In the authors' experience, how the horse responds to the intra-articular anesthetic procedure is a reasonable "gauge" of how the horse will tolerate the standing procedure. All horses that have been scheduled to undergo the procedure have been cooperative enough to allow for diagnostic evaluation of the intended joints, with the exception of one horse.

The procedure has allowed diagnostic information to be gained on horses with questionable diagnoses on the basis of other conventional diagnostic techniques. The procedure has benefit in the quick and relatively atraumatic format of the technique, allowing the clinician to provide a more accurate diagnosis for the patient. Currently, if a surgical lesion is diagnosed, routine therapeutic arthroscopy can be recommended. If a surgical lesion is not identified, medical treatment can be recommended. In the authors' opinion, a better rehabilitation procedure can

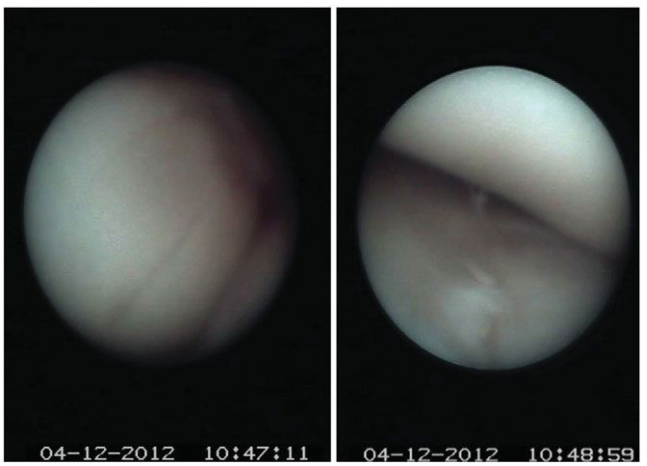


Fig. 8. Full-thickness lacerations in the medial condyle (left) adjacent to a medial meniscal lesion (right).

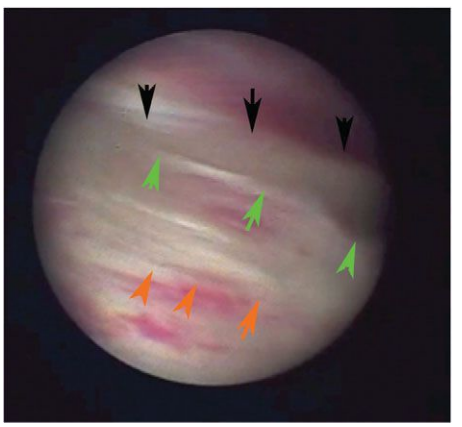


Fig. 10. Image of the cranial compartment of the MFT, weight-bearing position. Black arrows delineate the margin of the proximal articular margin of the medial condyle and reflection of synovial membrane. Green arrows delineate the proximal border of the medial meniscus. Orange arrows delineate the horizontal tear in the medial meniscus, which was noted to become larger as the horse became more non–weight-bearing.

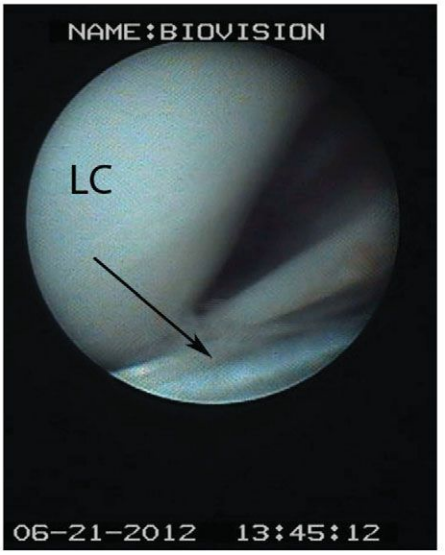


Fig. 11. Lateral condyle (LC) and longitudinal tear in the cranial ligament of the lateral meniscus (arrow).

be constructed with a more accurate knowledge of the health of the articular cartilage and intra-articular soft-tissue structures.

As expected, the field of view is smaller than that of a standard 4-mm arthroscope, but this was not considered to be a significant limitation. In fact, in the authors' opinion, the 18-gauge arthroscope provides a better exploratory of the stifle joint than would be obtained with a 2.7-mm arthroscope. Further, the resolution of the system was good at 640 × 480, for still and video capture. Because an egress portal is not routinely used, joint fluid is not flushed through the joint, which can cause visibility issues and require egression of the joint fluid.

In summary, the authors believe that clinical cases that have the following components are good candidates for this procedure: (1) pain regionalized to the stifle with the use of intra-synovial anesthesia, (2) lack of a definitive radiographic diagnosis, (3) definitive ultrasonographic diagnosis missing or unclear, and (4) owner's unwillingness to allow general anesthesia without a clear diagnosis.

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[a]18-Gauge needle arthroscope (1.3-mm diameter), BioVision Technologies, 221 Corporate Circle, Suite H, Golden, CO 80401.
[b]Kimzey Leg Save Splint, Kimzey, Inc, 164 Kentucky Avenue, Woodland, CA 95695.
[c]BioVision Technologies, 221 Corporate Circle, Suite H, Golden, CO 80401.

Review of Serum Chemistry Interpretation in Neonatal Foals

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Interpretation of serum chemistry results in foals can be very confusing because reference ranges are usually based on adult samples, and many values are increased in foals compared with adults. Understanding the expected differences in serum chemistry results between foals and adult horses improves the recognition of abnormalities and facilitates making an accurate diagnosis. Author's address: PO Box 1843, Georgetown, KY 40324; e-mail: bwaldridge@earthlink.net. © 2013 AAEP.

1. Introduction

Reference ranges for many serum chemistry values in adult horses cannot reliably diagnose disease in foals.[1–4] Placental abnormalities may influence clinicopathologic results in neonates during the first few days of life.[1] Most hepatic indices in foals are significantly different from adult horses, especially during the neonatal period. Ideally, age-specific reference ranges should be used in foals, but it is difficult to sample a large enough population of clinically normal foals and few laboratories are able to generate age-specific references. Whenever possible, the reference ranges in this report have been obtained from clinically healthy foals.

2. Hepatic Indices

Sorbitol dehydrogenase and aspartate transaminase are reliable indicators of hepatocellular disease in foals.[2,5] Sorbitol dehydrogenase and aspartate transaminase have more narrow standard deviations in foals, and their normal reference ranges are closer to those of adults.

Serum alkaline phosphatase (ALP) activity is highest during the first 2 to 8 weeks of life[2,3,6,7] and remains elevated for 8 weeks[7] to at least 90 days.[6] There is a wide individual variation and large standard deviation of ALP activity in foals, which limits its effectiveness to diagnose hepatobiliary disease.[2]

Hank et al[8] measured activity of specific ALP isoenzymes and determined that 80% to 92% of total ALP activity is from bone. However, Dumas and Spano[9] reported that increased ALP activity in foals is mainly of hepatic origin. Although the contribution of the various ALP isoenzymes to total ALP activity remains unclear, most of the increased ALP activity in foals is attributed to increased osteoblastic activity and bone formation in young, growing animals.[2,4,6,8]

Gamma glutamyltransferase (GGT) activity in foals ranges from 1.5 to 3 times normal adult values for the first 3 to 4 weeks of life.[2,10] Gamma glutamyltransferase activity in foals is most increased from adult horses at 7 to 21 days of age.[6] Similar to ALP activity, there is a normal wide individual variation in GGT activity between foals, which restricts its

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diagnostic use to identify hepatic disease.[2] Although mare colostrum contains a low amount of GGT, postsuckle GGT activity in foals is not associated with serum immunoglobulin G concentration[10] and cannot be used to determine colostrum intake.[11] Hepatic GGT activity is increased in young animals and may account for some of the increased GGT activity in foals.[2,10]

Neonatal hyperbilirubinemia is normal during the first 2 to 3 weeks of life.[4–6] Bilirubin concentration is highest in young foals and reduces to normal adult horse ranges by 7 to 14 days of age.[5,6] Both total and unconjugated bilirubin concentrations in foals are increased over adult values from birth through 7 to 14 days postpartum.[6] Neonatal hyperbilirubinemia may be the result of immature hepatic function or hemolysis of fetal erythrocytes.[3,4,6] The ability of the fetal liver to excrete bilirubin is minimal, and bilirubin must be excreted across the placenta by the fetus.[6] Foals less than 5 days of age have less hepatic glucuronyl transferase activity than adults and therefore a slower rate of bilirubin uptake and conjugation.[4] Increased direct bilirubin concentration in foals 2 days of age or less may be due to a lack of bilirubin transport proteins.[3]

Serum bile acid concentration is highest at birth and gradually declines for at least 6 weeks postpartum.[5] Serum bile acid concentration in foals may be elevated because of increased hepatic production, decreased excretion, differences in gastrointestinal flora, or enhanced intestinal absorption. Hepatic uptake of serum bile acid and excretion into bile require active transport, which may not be fully functional in foals.

Triglyceride concentration in foals tends to be highest during the first 2 weeks of life and moderately increased for up to 6 weeks of age.[5,6] Triglyceride concentrations in foals may decrease as hepatic function matures and triglycerides are used to synthesize other lipoproteins.[6] Triglycerides can be very elevated in foals that have recently nursed, as the result of digestion of fat in mare's milk.

3. Renal Function

Increased creatinine concentration in newborn foals is usually caused by placental pathology and/or fetal stress rather than renal disease.[1] Placental pathology appears to affect serum blood urea nitrogen (BUN) concentration less than creatinine, probably because urea is a smaller and more diffusible molecule.

Spurious hypercreatininemia in foals <2 days of age is defined by serum creatinine concentration >5 mg/dL, and creatinine concentration decreases by $\geq 50\%$ in the first 24 hours of treatment and each day until it is within normal range by 72 hours.[12] Serum creatinine concentration in foals with spurious hypercreatininemia normalizes regardless of fluid administration, including foals that are only nursing free-choice. Seventy-one percent of foals affected by spurious hypercreatininemia were also diagnosed with neonatal encephalopathy in one report.[12] It is unknown if placental abnormalities predispose newborn foals to spurious hypercreatininemia.

Blood urea nitrogen concentration is near normal adult ranges at birth, declines during the first 48 hours of life, and remains low until 1 to 18 weeks of age.[4,7,13] Very young foals may be azotemic, but renal indices and plasma osmolality should decrease as the foal nurses and fluid intake expands plasma volume and stimulates diuresis.[13] Increased BUN concentration often occurs when foals are in a catabolic state and using endogenous protein as an energy source.[1] Deamination of protein results in increased urea production and excretion. Low BUN concentration in growing foals may be the result of increased amino acid utilization for protein synthesis.[3]

4. Electrolytes

At birth, both total and ionized calcium concentrations are 25% to 30% higher than in adult horses.[1] Hours after birth, blood calcium concentration is approximately 20% lower than in adults and then gradually returns to normal limits in the first few days of life. Edwards et al[13] reported that serum calcium concentration decreased significantly during the first 48 hours of age and returned to normal ranges by 7 days.

Inorganic phosphorous concentration is elevated from birth until at least 18 weeks of age.[7] Schmitz et al[4] found that serum inorganic phosphorus concentration was initially slightly higher than expected adult ranges and peaked between 2 to 3 weeks of age. Serum inorganic phosphorous concentration then gradually decreased but remained above normal adult ranges at 6 months of age. Hyperphosphatemia is apparently related to skeletal ossification and osteoblastic activity.[4,13]

5. Conclusions

Interpretation of serum chemistry values in neonatal foals can be complicated when normal ranges from adult horses are used. Comparison of reference ranges from textbooks and between different laboratories and serum chemistry analyzers is not always reliable. Ideally, the normal expected ranges used in foals are stratified by age and are based on a large sample of healthy foals. However, this is often not possible, and knowing the inherent differences in serum chemistry values between foals and adult horses helps to determine if an abnormality is significant.

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Disposition and Metabolic Profile of the Weak Androgen Dehydroepiandrosterone After Administration as Part of a Nutritional Supplement to Exercised Horses

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After oral administration of a nutritional supplement containing dehydroepiandrosterone (DHEA), horses are capable of producing detectable levels of testosterone, which can result in a positive regulatory finding. Authors' addresses: School of Veterinary Medicine (Arthur), K.L. Maddy Equine Analytical Chemistry Laboratory (Knych, Stanley), University of California, Davis, CA 95616; e-mail: rmarthur@ucdavis.edu. *Corresponding author; †presenting author. © 2013 AAEP.

1. Introduction

Dehydroepiandrosterone (DHEA) is a weak androgen as well as a precursor to the much more potent androgen, testosterone. Although DHEA is not specifically banned from administration to racehorses, the potential for metabolism to substances such as testosterone that are not permitted in racing make its presence in nutritional supplements fed to horses a regulatory concern. In the current study, we sought to describe the metabolic profile after administration of a nutritional supplement containing precursors to non-permitted androgens to exercised female horses.

2. Materials and Methods

Eight healthy, exercised adult female horses received a single administration of 15 mL of a nutritional supplement containing DHEA (500 mg total dose) and pregnenolone (500 mg total dose). Blood and urine samples were collected at time 0 and at various times up to 48 hours after drug administration. Plasma concentrations of DHEA, testosterone, and pregnenolone as well as their sulfated conjugates were measured by means of liquid chromatography-mass spectrometry.

3. Results and Discussion

Peak plasma concentrations of DHEA, DHEA sulfate, and testosterone sulfate were 10.1 ± 8.84 ng/ml, 25.7 ± 10.2, and 0.670 ± 1.17, respectively. All compounds were below the limit of detection by 24 hours after supplement administration. Peak urine concentrations of DHEA and testosterone sulfate were 1947 ± 1045 ng/mL and 256 ± 163, respectively. Both compounds remained above the limit of detection of the assay through the 48-hour time point. Results of this study warrant careful use of nutritional supplements containing androgen precursors to racehorses.

Research Abstract

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Sequential L-Lactate Concentration in Hospitalized Equine Neonates: A Prospective, Multicenter Study

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L-lactate measurements, single and sequential, serve as strong independent predictors of mortality in critically ill foals. Authors' addresses: University of Pennsylvania, New Bolton Center, Kennett Square, PA 19104 (Borchers, Wilkins, Boston); Hagyard Equine Medical Institute, 4250 Iron Works Parkway, Lexington, KY 40511 (Marsh); Scone Equine Hospital, 106 Liverpool Street, Scone NSW 2337, Australia (Axon); Equine Medical Center of Ocala, 7107 County Road 326, Ocala, FL 34482 (Read); University of Bologna, Via Zamboni, 33, 40126 Bologna, Italy (Castagnetti); Woodford Equine Hospital, 3550 Lexington Road, Versailles, KY 40383 (Pantaleon); Peterson Smith Equine Hospital, 4747 SW 60th Avenue, Ocala, FL 34474 (Clark); University of Illinois, College of Veterinary Medicine, Urbana, IL 61802 (Wilkins, Qura'n); Mid Atlantic Equine Medical Center, 40 Frontage Road, Ringoes, NJ 08551 (Belgrave); University of Zurich, Rämistrasse 71, 8006 Zurich, Switzerland (Schwarzwald); Purdue University, College of Veterinary Medicine, West Lafayette, IN 47907–1240 (Levy); Tufts Cummings School of Veterinary Medicine, North Grafton, MA 01536 (Bedenice); University of Pretoria, Blackmoor 357-Jr, Pretoria 0002, South Africa (Saulez); e-mail: pawilkin@illinois.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Serial blood L-lactate concentration [LAC] changes are of prognostic value in critically ill neonatal foals but have not been prospectively evaluated in a large, multicenter study.

2. Materials and Methods

For this prospective, observational study, 13 university and private equine referral hospitals enrolled 643 foals over the 2008 foaling season. [LAC] was measured at admission ([LAC]ADMIT) and 24, 48,

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72, and 120 hours after admission. [LAC] changes over time ([LAC]Δ) were calculated between sampling points.

3. Results

Nonsurvivors had significantly greater [LAC]ADMIT, [LAC] 24 hours, and [LAC] 48 hours compared with surviving foals ($P < 0.001$). [LAC]Δ in nonsurviving foals did not decrease over time, whereas survivors showed significant positive [LAC]Δ between [LAC]ADMIT through 24 hours and all other time periods ($P < 0.001$). Logistic regression analysis showed that the odds of survival decreased for each 1 mmol/L [LAC] increase at all time points for all sick foals, independent of major final diagnoses as potential confounders. Septic foals showed significantly greater [LAC] at all time points compared with nonseptic foals ($P < 0.001$). [LAC]Δ in septic foals was significantly more positive (suggesting better clearance) at [LAC]ADMIT through 24 hours and [LAC] 72 through 96 hours ($P < 0.01$), whereas in nonseptic foals, [LAC]Δ was significantly positive between [LAC]ADMIT through 24 hours compared with all other time periods ($P < 0.001$).

4. Discussion

L-lactate metabolism is impaired in nonsurviving and septic foals, and [LAC]Δ can be used to identify patients that are at high risk for mortality.

Hematologic and Biochemical Reference Intervals in Adult Friesian Horses From North America

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Friesian-specific reference intervals make diagnosis, treatment, and assessment of response to therapy more accurate and provide equine veterinarians greater confidence in evaluating the health of the Friesian horses under their care. Authors' addresses: Fenway Foundation for Friesian Horses, N3398 State Road 76, Hortonville, WI 54944 (Fox); Department of Pathobiological Sciences, School of Veterinary Medicine, University of Wisconsin, 2015 Linden Drive, Madison, WI 53706 (Sample, Friedrichs); IDEXX Laboratories, 3 Centennial Drive, Suite 1, North Grafton, MA 01536 (Wunn); IDEXX Laboratories, 300 East Wilson Bridge Road, Suite 200, Worthington, OH 43085 (Roth); e-mail: friedrik@svm.vetmed.wisc.edu. *Corresponding author; †presenting author. © 2013 AAEP.

1. Introduction

Friesian horses originated in The Netherlands and have become popular in North America over the past 20 years. Purebred Friesian horses were reintroduced to North America in 1974, and it is estimated that between 10,000 and 12,000 Friesian horses currently reside here. Several well-documented conditions affect the Friesian breed, with ongoing research directed at understanding and reducing their incidence. Because there is little published work on breed-specific reference intervals (RI) in horses, the impact this may have on the care and research in Friesian horses is unknown. The goal of this project was to determine hematologic and biochemical RI for Friesian horses residing in North America.

2. Materials and Methods

American Society for Veterinary Clinical Pathology (ASVCP) guidelines for establishing RI in veterinary species were followed. Strict inclusion and exclusion criteria were established for selection of reference subjects and blood specimen collection and handling. Complete blood counts and biochemistry profiles were performed at IDEXX Laboratories, Columbus, Ohio.

3. Results

Nonparametric RI with 90% confidence intervals were subsequently determined from 123 healthy, adult Friesian horses of either sex. IDEXX's equine RI are appropriate (transferrable) to Friesian horses for 30 of 37 variables. However, Friesian-specific RI are recommended for hemoglobin, red blood cell

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count, hematocrit, glucose, and lactate dehydrogenase. The use of Friesian RI for other analytes also may lead to more accurate interpretation in health and disease.

Acknowledgment

This work was made possible through financial and inspirational support of the Fenway Foundation for Friesian Horses, Hortonville, Wisconsin.

Evaluation of the Pre-Analytical Stability of Adrenocorticotropic Hormone in Plasma and Whole Blood From Horses

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Adrenocorticotropic hormone (ACTH) measurements from the equine patient are stable until 24 hours in samples stored at 21°C or 4°C, whereas storage as whole blood or plasma has no effect. Freezing samples maintains adrenocorticotropic hormone levels for at least 30 days. Authors' addresses: The William R. Pritchard Veterinary Medical Teaching Hospital and the Department of Medicine and Epidemiology (Prutton, Pusterla, Watson) and Department of Population Health and Reproduction (Kass), School of Veterinary Medicine, University of California, Davis, CA 95616; e-mail: j-prutton@hotmail.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Equine pituitary pars intermedia dysfunction is a disease of aged horses that is most easily diagnosed through the use of an endogenous adrenocorticotropic hormone (ACTH) blood sample. The stability of ACTH in a collected blood sample has not been fully elucidated, and the goal of the study was to address this.

2. Materials and Methods

Eleven horses were blood-sampled, and ACTH levels were assessed after storage at 4°C, 21°C, −20°C, and −80°C for up to 30 days either as whole blood or plasma.

3. Results

Detection of ACTH was similar between whole blood and plasma. Time affected ACTH levels, with storage beyond 24 hours dramatically reducing ACTH recovery. Freezing at both −20°C and −80°C did not depreciate ACTH levels.

4. Discussion

ACTH measurements in the equine patient were subject to degradation, but appreciable changes were only seen at 48 hours and longer in samples stored at 21°C or 4°C, whereas storage as whole blood or plasma had no effect. Freezing samples maintained ACTH levels for at least 30 days. This information allows practitioners to reasonably store samples without centrifugation for at least 24 hours and appears to negate the need for protease inhibitors in the samples.

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Comparison of Transverse Facial Venous Sinus and Jugular Blood Values in Healthy and Critically Ill Horses

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Findings support the use of the transverse facial venous sinus (TFVS) as a blood sampling site in horses. Authors' address: Large Animal Clinical Sciences, Oregon State University College of Veterinary Medicine, Corvallis, OR 97331; e-mail: barbara.hunter21@gmail.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The transverse facial venous sinus (TFVS) can be used for blood collection in the horse, but information on the validity of blood values from this site is limited.

The objectives of the study were to determine whether packed cell volume (PCV), total solids (TS), and blood lactate concentrations in blood drawn simultaneously from a TFVS and the jugular vein of critically ill horses is correlated and to determine the effect of serial TFVS sampling on the same parameters in healthy horses.

2. Methods

Critically ill horses had simultaneous blood samples drawn from a TFVS and the jugular vein. Blood was drawn from the left TFVS and the jugular vein from six healthy adult horses every 6 hours for 24 hours, then every 24 hours for 72 hours. Blood was drawn from the right TFVS and the jugular vein every 24 hours for 96 hours. All samples were analyzed for PCV, TS, and blood lactate concentration. Data were analyzed by means of two-way repeated-measures analysis of variance. Significance was set at $P \leq 0.05$.

3. Results

There were no significant differences in PCV, TS, or blood lactate concentrations of TFVS samples compared with jugular blood in critically ill horses. Serial TFVS sampling in healthy horses had no significant effect on TS or blood lactate concentrations. PCV in the TFVS was significantly lower than jugular blood on serial sampling, but the difference was not considered clinically relevant.

4. Conclusions

Blood values for PCV, TS, and blood lactate concentrations were comparable between the TFVS and the jugular vein.

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Variation of *VapA*-Specific Immunoglobulin G in Mares and Foals on Different Farms in Kentucky

Macarena Sanz, DVM, MS, Diplomate ACVIM*; Aline Oliveira, PhD; Allen E. Page, DVM; and David W. Horohov, MS, PhD

Mares have overall low *VapA*-specific immunoglobulin (Ig) G regardless of the *Rhodococcus equi* incidence on the farm. Foals are exposed to *R equi* within the first month of age. Authors' address: Maxwell H. Gluck Equine Research Center, Department of Veterinary Science, University of Kentucky, Lexington, KY 40546–0099; e-mail: sanzmaca@gmail.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Rhodococcus equi causes pneumonia in young foals and major economic losses to the horse industry. Our objective was to evaluate serum *VapA*-specific immunoglobulin (Ig)G in mares and foals from farms that administered *R equi*–specific hyperimmune plasma and farms that did not administer the plasma.

2. Materials and Methods

Serum from mare and foal pairs from four farms in Kentucky was evaluated for *VapA*-specific IgG with the use of enzyme-linked immunoassay. Blood was collected at foaling (mare and foal) and for a period of 5 to 6 months thereafter (foals).

3. Results

A total of 161 pairs of serum were included: 121 from a farm that used hyperimmune plasma and 40 from three farms that did not use the plasma. The level of *VapA*-specific IgG of the mares was overall low and was not significantly different between farms. Foals given hyperimmune plasma had significantly higher *VapA*-specific IgG than untreated foals. All foals had an increase in *VapA*-specific IgG over time. Only moderate correlation between *VapA*-specific IgG between mares and foals was observed when plasma was not given.

4. Discussion

The moderate correlation between mare and foal *VapA*-specific IgG may explain why vaccination of mares has not always been effective in preventing *R equi* infection. Foals produced *VapA*-specific antibodies after the first month of age, which strongly suggests that *R equi* exposure occurs early in life.

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Serologic Responses of West Nile Virus Sero-Negative Mature Horses to West Nile Virus Vaccines

Kevin G. Hankins, DVM, MBA

Inclusion of West Nile virus (WNV) with equine encephalitis viruses and tetanus in vaccines has a detrimental impact on WNV antibody production. To maximize the humoral immune response against WNV, monovalent WNV vaccines should be considered. Author's address: 12685 McIntyre Creek Road, Olsburg, KS 66520; e-mail: Kevin.hankins@zoetis.com. © 2013 AAEP.

1. Introduction

The first vaccine against West Nile virus (WNV) was licensed as a monovalent viral inactivated vaccine. Since then, incorporation of the WNV antigen in multivalent vaccines has become common. This study was conducted to assess, under field use conditions, the impact on serological response of three commercially available WNV vaccines either given as a monovalent injection or as a multivalent WNV vaccine incorporated with equine encephalitis and tetanus vaccine.

2. Materials and Methods

Two hundred forty mature, WNV sero-negative (less than four) horses were followed serologically before and after primary and secondary vaccination with six different vaccination programs, all including WNV antigens. Forty horses were left as unvaccinated sentinel horses.

3. Results

All vaccines stimulated both a primary and secondary (booster) response to vaccination that was significantly higher than sero-negative controls. However, inclusion of WNV with equine encephalitis viruses and tetanus toxoid in vaccines had a significant detrimental impact on WNV serum neutralization antibody production to both the primary and secondary vaccinations.

4. Discussion

This study showed that West Nile monovalent vaccines stimulated a 2- to 3-fold increase in the serologic response to WNV compared with West Nile multivalent vaccines incorporated with equine encephalitis and tetanus. The impact of the dramatic differences seen in post-vaccination WNV titers on protection in the horse is not known. Several studies have demonstrated the correlation between antibodies against WNV and protection against challenge. One author suggested the use of post-vaccination WNV antibody levels as a determinant of duration of immunity. These studies suggest that antibodies may play a role in speed of onset, level, or duration of protection against WNV infection.

Research Abstract

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Junctional Epidermolysis Bullosa in Belgian Draft Horses in North America: Results of Genetic Testing (2002–2012)

John D. Baird, BVSc, PhD*; Lee V. Millon, BS; and M. Cecilia T. Penedo, PhD

Junctional epidermolysis bullosa (EB) in the Belgian draft horse and other draft horse breeds is a genodermatosis inherited as an autosomal recessive trait. The mutation responsible is a cytosine insertion (1368 insC) in the *LAMC2* gene, which results in the absent expression of the laminin α-2 polypeptide chain of the heterotrimer laminin-332 (previously laminin-5). A polymerase chain reaction (PCR) test was developed to identify carriers of the mutation through the use of mane hairs. Mandatory testing of breeding stallions was instituted by the Belgian Draft Horse Corporation of America on November 1, 2002, and by the Canadian Belgian Horse Association on January 1, 2003. In the following 10 years (until December 31, 2012), a total of 2176 registered Belgian draft horse stallions have been tested. The number of stallions identified as carriers of the *LAMC2* mutation was 319 (12.5%). The genetic testing of breeding mares for the *LAMC2* mutation has been on a voluntary basis. Over the first 10 years of genetic testing, there has been no statistically significant change in the percentage of carriers of the *LAMC2* mutation. Authors' addresses: Department of Clinical Studies, Ontario Veterinary College, University of Guelph, Guelph, ON N1G 2W1 Canada (Baird); Veterinary Genetics Laboratory, University of California, One Shields Avenue, Davis, CA 95616 (Millon, Penedo) e-mail: jbaird@ovc.uoguelph.ca. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Epidermolysis bullosa (EB) is an inherited mechanobullous disorder characterized by skin fragility and blister formation after minor trauma or traction on the skin.[1] There are many clinically distinctive phenotypes, all of which have skin blistering as a major feature, but variable risks of extracutaneous manifestations and premature death. In humans, more than 1000 different mutations involving 14 structural genes within the skin have been documented to lead to the clinical phenotypes of EB.[1,2] Mutations result in either abnormal, absent, or significantly reduced levels of a specific protein that is important in epidermis to dermis adhesion. The result is shearing of the skin or blistering with ultrastructurally uniform cleavage planes.[3]

EB is currently classified into four major types, on the basis of the level of the skin where the missing or abnormal structural skin protein is located and the corresponding ultrastructural level of cleavage.[1,3] In EB simplex, cleavage and blister formation occurs within the epidermis. In junctional EB (JEB), blister formation occurs within the lamina lucida, an electron-lucent region that contains anchoring filaments that connect the basal keratinocytes to the underlying lamina densa. In dystrophic EB, blis-

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Fig. 1. Newborn Belgian foal shows characteristic skin lesions of junctional epidermolysis bullosa-Herlitz form.

tering occurs in the dermis (or sublamina densa). The fourth type of EB is the Kindler syndrome, which is a mixed type of EB that exhibits multiple cleavage planes within the affected skin.[1,3]

JEB-Herlitz (JEB-H) represents the most severe form of EB, which is characterized by generalized, extensive mucocutaneous blistering at birth with erosions of the skin and mucous membranes, and dental enamel hypoplasia.[3,4] In humans, the disease is lethal in early childhood.[1] JEB-H is most often caused by homozygous null mutations in the genes *LAMA3*, *LAMB3*, or *LAMC2*, each gene encoding for one of the three chains of the heterotrimer laminin-332 (previously laminin-5).[3] The Herlitz form of JEB is the result of complete absence of laminin-332.[4,5]

Laminin-332 is a major adhesion protein within the basement membrane zone of the skin and mucous epithelia that provides stable anchorage of basal epithelial cells (keratinocytes) to the underlying dermis by connecting the hemidesmosomal component $\alpha 6\beta 4$ integrin to collagen VII–containing anchoring fibrils.[4] Laminin-332 is an essential component of the dermal-epidermal basement membrane.[5] The usual mode of transmission is autosomal recessive.[3,4]

In North America, reports of a junctional mechanobullous disease in Belgian foals were first published in the late 1980s.[6–8] The most consistent skin changes are irregular, round, red, and ulcerated areas over the bony prominences of the hocks, stifles, hips, carpi, elbows, and fetlocks (Fig. 1). The severity and extent of the skin lesions progress with age. Affected foals usually have very extensive oral erosions and ulcers, especially around the base of the incisor teeth. One of the most characteristic findings is that the temporary incisor teeth, which are not usually noticed until 8 to 14 days of age, are visible at birth. The teeth are very white and have irregular serrated edges, with pitted enamel. Excessive amounts of blood-tinged saliva may occur as a result of oral ulceration. Irregular areas of ulcers are along the coronary bands, which may progress to sloughing of the hoof.[6–9]

The clinical, histopathological, ultrastructural, and immunohistochemical findings have shown that the disease in North American Belgian draft foals fits all the criteria of the JEB-H form of EB.[10] The mutation responsible for this particular form in Belgian draft horses was first identified on January 24, 2001, by researchers at the INSERM 634 Laboratory, University of Nice, France.[10] The mutation is a cytosine insertion in the genomic nucleic acid sequence of affected horses at position 1368 of the laminin $\alpha 2$-encoding polynucleotide, resulting in a frame shift that leads to a premature termination codon and absent expression of the *LAMC2* gene.[10] An autosomal recessive mode of inheritance of this mutation was verified.[10]

After the identification of the *LAMC2* mutation, a commercial polymerase chain reaction (PCR) test was developed at the Veterinary Genetics Laboratory (VGL), School of Veterinary Medicine, University of California-Davis. This test was performed on DNA samples with fluorescence-labeled primers designed to amplify the region containing the mutation. The mutation is a single base insertion, and thus carriers have a PCR product that is one base longer than the normal allele. The single base difference is detected by analysis of the PCR products by capillary electrophoresis on ABI 3730 DNA sequencer.[9]

The availability of a commercial PCR test and appropriate genetic counseling made it possible for Belgian draft horse breeders to avoid the financial and genetic losses associated with the birth of JEB-H foals. Commencing on November 1, 2002, the Belgian Draft Horse Corporation of America (BDHCA) instituted rules that require the sire of a foal being registered to have been DNA-profiled and JEB-tested. This testing program is organized by the corporation office. The breed association provides hair sample collection information and forms to the owners of horses to be tested. Samples are submitted to the VGL for testing, and results are reported directly to the breed association office. The JEB results are printed on the Certificate of Registry. The Canadian Belgian Horse Association (CBHA) instituted the same requirements that commenced on January 1, 2003. The testing of mares for the *LAMC2* mutation has been on a voluntary basis; however, when mares are bred by artificial insemination with frozen semen they are required to be DNA- and JEB-tested. Foals resulting from frozen semen insemination are also required to be DNA- and JEB-tested. The aim of the genetic testing program is (1) to prevent the birth of JEB-H Belgian foals and (2) to reduce the number of carrier animals in the population.

2. Materials and Methods

Until December 31, 2012, mane hair root samples from 1785 stallions, 301 mares, and nine geldings

registered with the BDHCA and from 391 stallions, 65 mares and three geldings registered with the CBHA were forwarded to the VGL for the commercial PCR test for the *LAMC2* mutation. Logistic regression models were used to examine the relation between year of testing and *LAMC2* mutation carrier status, the binary outcome (SAS 9.2). We also allowed for quadratric effect, breed registry, and interactions.

3. Results and Discussion

In the BDHCA registry, 206 (11.5%) of 1785 stallions tested between 2002 and 2012 were found to be carriers of the *LAMC2* mutation. In the CBHA registry 47 (12.0%) of 391 stallions tested between 2003 and 2012 were found to be carriers of the *LAMC2* mutation. Over the same time period, the number of mares JEB-tested has been low. In both registries, only 336 mares have been tested, with 65 (17.8%) identified as carriers of the *LAMC2* mutation. In the past 3 years (2009–2012) in the BDHCA registry, of the 42 horses found to be carriers, 21 (50%) were from mating in which the stallion was a JEB-tested non-carrier and the mare had not been JEB-tested.

On statistical analysis, there were no quadratic effects or interactions (P values >0.25). There was also no effect of breed registry (P value 0.8149) (odds ratio = 1.041, 95% confidence interval = 0.743, 1.459) and no trend over years of testing (P value 0.2339) (slope = −0.275, 95% confidence interval = −0.0729, 0.0178). There has been no statistically significant difference in the *LAMC2* mutation carrier rate in the Belgian stallion population in both the BCDHA and CBHA registries since testing over the 10-year period of testing.

There is anecdotal evidence from equine practitioners, the breed association offices, and breeders that the occurrence of JEB-H foals has declined markedly. The last two cases of JEB-H that the authors are aware of were born in Indiana in 2008 and in Ontario in 2009. The lack of JEB-H foals has been attributed to Belgian draft horse breeders selecting sires that are not carriers of the *LAMC2* mutation. In 2012, of 132 animals that were JEB-tested 12 (10 stallions, two mares) were identified as carriers. Of the 132 tested animals, 100 (75.8%) were sired by non-carrier stallions, 30 (22.7%) were sired by stallions that were born before the introduction of mandatory testing (not JEB-tested), and two were sired by one known carrier stallion.

Acknowledgments

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First Report of the Use of Skin Prick Test Diagnostic Technique in Horses

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The skin prick test (SPT) was a valuable means of identifying potential allergic triggers in horses with recurrent airway obstruction and may be a step toward the establishment of successful eviction measures and eventually, specific immunotherapy. Authors' addresses: CIISA, Department of Clinics, Faculdade de Medicina Veterinária, Universidade de Lisboa, Avenida da Universidade Técnica, 1300 – Lisboa, Portugal (Tilley, Luis); Medicina II, Faculdade de Medicina, Hospital Santa Maria, Universidade de Lisboa, 1100 – Lisboa, Portugal (Ferreira); e-mail: paulatilley@fmv.utl.pt. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The authors evaluated the response to skin prick tests (SPT) with common aeroallergens in horses with recurrent airway obstruction (RAO).

2. Materials and Methods

Thirty-six horses with RAO and 10 healthy control horses were studied, taking into account the medical history and physical examination, supported by thoracic radiography, respiratory tract endoscopy, and bronchoalveolar lavage cytology. SPT to 16 locally relevant aeroallergens (molds, mites, pollens, and dander) were performed.

3. Results

All horses with RAO had positive SPT results to at least five aeroallergens. Four control horses had all negative SPT results, and six had positive SPT to one to three aeroallergens, although with much lesser reactivity: mean wheal diameter was 30% to 50% of histamine, contrasting with 30% to 127% of histamine for the positive SPT results in horses with RAO.

4. Discussion

The SPT was a valuable means of identifying potential allergic triggers to reduce the allergen load. The SPT may be a step toward accurate determination of allergens to which horses with RAO may be sensitized to establish successful eviction measures and eventually, specific immunotherapy.

Research Abstract

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Effect of Pre-Insemination Uterine Lavage on Fertility in a Population of Subfertile Mares

Marco Livini, DVM*; Armena Zamboni, DVM; and Denis Necchi, DVM, ECAR

Uterine lavage is commonly used in the peri-ovulatory period to improve pregnancy rates in mares with reduced fertility. Previous work has shown that uterine lavage with lactated Ringer's solution performed immediately before insemination does not affect the fertility in reproductively healthy mares. In the present field study, uterine lavage using lactated Ringer's solution was performed on a population of barren mares (n = 97) immediately before insemination with fresh, cooled, or frozen semen. Sixty-four (64/97, 68%) mares delivered live foals after this treatment. These data suggest that uterine lavage immediately before breeding is not detrimental to fertility and can improve pregnancy rates in previously barren mares. Authors' addresses: Veterinari Associati Ippovet, Cascina Longora, Carpiano (Milano) Italy 20080 (Livini, Zamboni); Selecet Breeders Services, Europe via Argine Capoluogo 39 San Daniele Po, Cremona 26046 Italy (Necchi); e-mail: marcolivini@libero.it. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Uterine lavage with the use of lactated Ringer's solution or saline (0.9% NaCl), is a well-documented therapy for mares with post–mating-induced endometritis. Uterine lavage is often combined with administration of oxytocin or cloprostenol in the peri-ovulatory period[1–4] to improve physical clearance of uterine fluid in susceptible mares that are unable to resolve the physiological inflammation that occurs after breeding. Uterine lavage in normal or susceptible mares 4 to 6 hours after insemination[3] did not show negative effects on fertility rates, and this practice is frequently used. More recently, Vanderwall and Woods[5] demonstrated that uterine lavage with lactated Ringer's solution performed immediately before insemination with cooled semen did not affect fertility in reproductively normal mares. The objective of this study was to determine the usefulness of flushing before insemination (FPI) in mares with known fertility problems. We hypothesized that in mares with fertility problems, a uterine lavage with lactated Ringer's solution performed immediately before the insemination (FPI) would improve the uterine environment for survival and ascent of the spermatozoa to the oviducts, in particular when there is the presence of fluid in the uterus at the time of insemination. Flushing can also remove biofilms, which can act as a substrate for pathogenic organisms introduced at the time of insemination. Uterine lavage before breeding might also aid in removal of excess mucus on the surface of the endometrium that may interfere with the motility of the spermatozoa and mucociliary function.

2. Materials and Methods

A total of 97 mares from various breeds (32 Standardbred, 43 Warmblood, 17 Quarter Horses, 1 Haflinger), from ages 4 to 23 years, were used in the

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study during the breeding seasons 2009 to 2011. Ninety-one mares were inseminated with fresh or cooled semen; six of the mares were inseminated with frozen semen. Good-quality semen from known stallions was used.

Mares included in the study were classified in the following categories:

- Barren in the previous year (inseminated at least for two cycles)
- Barren for the present breeding season after being inseminated at least once (not at the first cycle of the season)
- Barren for two or more cycles in the current breeding season

All mares treated with FPI were managed similarly to previous cycles with the only addition being lavage immediately before insemination. Furthermore, all mares were inseminated with semen from the same stallion that was used in previous cycles that did not result in pregnancy. Therefore, mares served as their own controls.

The perineal area of all mares was prepared by securing the tail away from the perineal area and washing the vulva with soap, rinsing with water, and drying with a paper towel. Immediately before insemination, the uterus was flushed with 2 L of lactated Ringer's solution[a] maintained at room temperature with the use of a Foley[b] catheter. Fluid was recovered using gravity flow. At least 90% of the lactated Ringer's solution was recovered from all mares. If the effluent fluid was cloudy, extra flushes were performed until the fluid recovered was clear. Immediately after the lavage, the mare was inseminated following the same criteria of the previous cycles. All the mares inseminated with fresh or cooled semen ovulated within 48 hours of semen collection, whereas the mares inseminated with frozen semen ovulated 6 hours before or after the insemination. The mares were checked by transrectal ultrasound examination at 14 days after ovulation to detect the pregnancy. Mares determined to be pregnant at 14 days were rechecked at 21, 30, and 45 days of pregnancy and periodically during the fall months. Live foal delivery rate was recorded.

3. Results

Seventy of 97 (72%) mares treated with FPI were diagnosed as pregnant at 14 days after ovulation.

Three of the pregnant mares were diagnosed with twins. All mares with twins successfully underwent manual reduction of one embryonic vesicle.

One mare lost her pregnancy at 21 days, three mares reabsorbed at 30 days of pregnancy, one mare died, and two mares aborted in autumn; 64 (66%) mares foaled. Twenty-six mares submitted to FPI had fluid in the uterus before the treatment and four of those mares did not become pregnant.

Seven mares had cloudy return fluid, and two of these mares received an extra flush. Two of the seven mares did not become pregnant, one of which had received an extra flush.

4. Discussion

Previous work by Vanderwall and Woods[5] has shown that uterine lavage with lactated Ringer's solution performed immediately before insemination does not adversely affect fertility in reproductively healthy mares. This study also showed that pregnancy rates were not affected by remaining fluid in the uterus after lavage. In our study, pre-insemination lavage also showed a positive effect on pregnancy (72.2%) and foaling (66%) rates in previously barren mares. All mares included in this study were not pregnant for two or more previous cycles, with the use of similar breeding management and semen from the same stallion as prior cycles. A lack of untreated control mares was a flaw of this trial but was unavoidable, given the field conditions. As such, the previous poor pregnancy rates in this population of mares provide a solid comparison to very good pregnancy and live foal rates achieved in this study. Improvement in pregnancy rates after FPI in subfertile mares could be related to the clearance of inflammatory cells, cellular debris, and excess mucus, thus providing a more suitable environment for sperm transport to the oviducts.[6] It is possible that pre-insemination uterine lavage might also make mucociliary clearance more effective.[7,8] Uterine lavage is primarily used early in the post-insemination period to modulate the effects of post–breeding-induced endometritis by clearing the uterus of inflammatory byproducts after breeding.[3,9,10]

The goal of post-mating uterine lavage is to improve the uterine environment for eventual embryo entry. Flushing mares immediately before insemination with lactated Ringer's solution optimizes the uterine environment for the sperm, thus improving survival and ultimately fertility. Both techniques can be useful for improving pregnancy rates in barren mares.

This technique can be very useful in routine stud practice when fluid is detected at the planned time of insemination. Generally, insemination cannot be delayed under these conditions. However, most veterinary practitioners are reluctant to put semen into an inflamed environment, as would be assumed if the uterus contained fluid at the time of breeding. In this study, pre-insemination uterine lavage positively affected pregnancy and live foal delivery rates in mares with known history of reproductive failure. Further studies would be useful to investigate if FPI may positively affect pregnancy rates after natural cover and in mares inseminated at foal heat.[11]

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Hysteroscopic Hydrotubation of the Equine Oviduct

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The equine oviduct can be evaluated by hysteroscopic hydrotubation. Author's address: Inoue Equine Clinic, Shizunai-Mena 453-48, Shin-Hidaka, Hokkaido, 056-0001, Japan; e-mail: equine@mopera.net. © 2013 AAEP.

1. Introduction

Current methods to assess oviductal patency involve laparoscopy or laparotomy. Development of a method that does not require surgery would be of great diagnostic and prognostic merit. This study evaluated the hysteroscopic-guided hydrotubation of the equine oviduct through the use of videoendoscopy in standing mares.

2. Materials and Methods

Both oviducts from each of 10 sedated mares were used in this study. A catheter was inserted into the uterotubal junction with a guide wire under endoscopic observation, and a dye was flushed into the oviduct. Peritoneal fluid was collected by ultrasound-guided abdominocentesis and was inspected visually and by spectrometry for the presence of dye. A colpotomy was performed, and the endoscope was inserted into the abdominal cavity to inspect for the presence of the dye in the oviduct.

3. Results

In 15 of 20 attempts, the catheter was successfully inserted into the uterotubal junction, and dye was observed at the ampulla and fimbria. In two mares, the videoendoscope could not be manipulated to insert the catheter. Only one of two oviducts was flushed in an additional mare because insufflation of the uterus could not be maintained. The color of the dye was evident macroscopically and spectrophotometrically in four of eight mares from which peritoneal fluid was successfully collected.

4. Discussion

The results suggest that hysteroscopic-selective hydrotubation can be a less invasive tool for diagnosis and treatment of suspected blockage of the equine oviduct.

Research Abstract

NOTES

Concentrations of Testosterone and Estrone Sulfate After Castration and After Human Chorionic Gonadotropin Stimulation in Stallions

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After castration, testosterone and estrone sulfate reached castrate levels after 24 hours. After human chorionic gonadotropin (hCG) stimulation, the largest increase in testosterone concentrations were at 48, 72, and 96 hours, compared with pre-stimulation levels. Authors' address: Gluck Equine Research Center, Department of Veterinary Science, University of Kentucky, Lexington, KY 40546; e-mail: aestellervico@uky.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Human chorionic gonadotropin (hCG) stimulation is used in suspected cases of cryptorchidism and to assess testis function in stallions. Basal testosterone and estrone sulfate concentrations are used as markers of testicular tissue and to ensure complete castration. To the author's knowledge, the half-lives of these hormones are not reported for the horse, and the best time points to sample after hCG stimulation are controversial. The objective of this study was to determine (1) half-lives of endogenous testosterone and estrone sulfate after castration and (2) testosterone concentration after hCG stimulation.

2. Materials and Methods

Pony stallions ($n = 8$; age $\geq$2 years) were randomly assigned to control (5 mL of saline) or treatment groups (5000 IU hCG IV). Blood samples were drawn at 0, 1, 2, 6, 12, 24, 48, 72, and 96 hours. Stallions were castrated, and blood samples were drawn at 0, 1, 2, 3, 4, 5, 6, 8, 10, 12, 24, 36, 48, 72, 96, and 120 hours after castration. Estrone sulfate and testosterone were determined through the use of an enzyme-linked immunosorbent assay. Repeated-measures analysis of variance and exponential decay analyses were used.

3. Results and Discussion

Testosterone concentrations were significantly greater at 1, 12, 24, 48, 72, and 96 hours after hCG, compared with pre-stimulation values. The largest increases from baseline were at 48, 72, and 96 hours, which indicates that later sampling times may better identify the presence of testicular tissue. Half-lives of testosterone and estrone sulfate were 1.1 and 0.7 hours after castration, respectively, and both hormones reached castrate levels by 24 hours after castration.

Acknowledgments

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Research Abstract

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How to Collect Equine Oocytes by Transvaginal Ultrasound-Guided Follicular Aspiration

Hunter Ortis, DVM*; and Rob Foss, DVM

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1. Introduction

Assisted reproductive techniques such as oocyte transfer and intracytoplasmic sperm injection (ICSI) can be used clinically to produce foals from mares with reproductive abnormalities that prevent normal breeding or production of embryos.[1–3] ICSI can also be used to produce foals from stallions with poor fertility or from semen with limited quantity.[4–6] Both of these procedures require the collection of oocytes. Ultrasound-guided transvaginal oocyte aspiration (TVA) can be used by the practitioner to recover maturing oocytes from dominant gonadotropin-stimulated follicles (DSF) or immature oocytes from smaller or subordinate follicles (IMM) for use in these techniques. DSF oocytes are generally required for oocyte transfer, whereas both DSF and IMM oocytes are suitable for ICSI. Once collected, the oocytes can be used on-site or shipped to a referral laboratory for embryo production.

2. Materials and Methods

Equipment required for TVA includes the following:

(a) Transvaginal ultrasound probe with needle guide,[a] preferably with a micro-convex ultrasound probe

(b) 60-cm, double-lumen, 12-gauge oocyte aspiration needle[b]

(c) Aspiration pump with a vacuum/pressure relief valve[c] capable of maintaining a regulated negative pressure of 150 mm Hg[d]

(d) Collection bottle(s)[e] and water bath[e] set to 37°C; 250-mL bottles are a convenient size

(e) 150-mm sterile petri dishes[f]

(f) Warming tray[g] set to 37°C

(g) Dissecting microscope with transmitted light source as used for embryo search and identification

(h) 0.25-mL semen straws

(i) Pipettor and pipettes

(j) Embryo filter[h]

(k) Complete embryo flush medium[i] with 5 IU heparin[j]/mL

(l) Controlled flushing set[k]

(m) 20-mL all-plastic syringe[l]

(n) Glutaraldehyde[m] for sterilization of ultrasound probe and needle guide

Equipment Setup

A 2-L bag of complete embryo flush medium with 5 IU heparin/mL that has been pre-warmed to 37°C is attached to the controlled flush set and is suspended from an IV stand. A 20-mL, all-plastic syringe is attached to the female connector of the controlled flush set and the male connector of the controlled flush set is attached to infusion tubing of the aspi-

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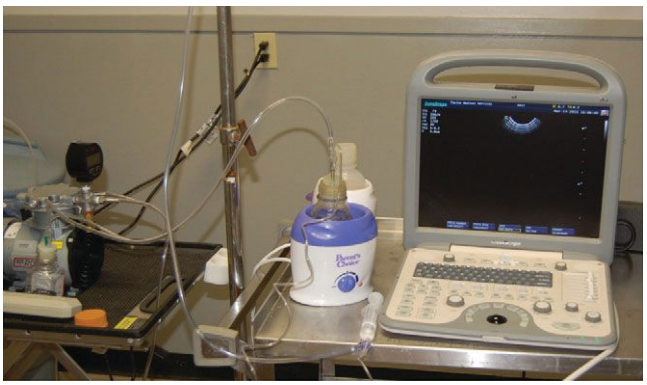


Fig. 1. Simple aspiration setup with the use of thermostatically controlled baby-bottle warmers as water baths.

ration needle. The controlled flushing set has an automatic three-way valve that allows an assistant to alternatively rapidly fill the syringe and then infuse flush medium into the follicle by simply moving the syringe plunger. Tubing from the aspiration port of the needle (the center lumen) is attached to the collection bottle placed in a 37°C water bath. The vacuum pump, regulated to −150 mm Hg, supplies the vacuum through the collection bottle (Fig. 1). The needle is inserted into the needle guide, with care taken to avoid contamination and to prevent dulling the needle.

Mare Preparation

DSF Oocytes

Oocytes are firmly adhered to the wall of the follicle. After the luteinizing hormone (LH) surge, as ovulation approaches, the attachment loosens in dominant follicles. Stimulation of the LH surge with an ovulatory agent such as deslorelin acetate[n] allows a timed collection of the oocyte 24 to 36 hours later.[7,8] This timing allows collection of an oocyte that has become easier to flush and has also started maturation and will not need additional hormonal stimulation. A typical mare is administered 1.8 mg of deslorelin acetate after detection of a 35-mm actively growing follicle during estrus in association with endometrial edema, but this will vary with breed, individual, and time of year.

IMM Oocytes

Immature oocytes can be collected from small follicles at any stage of the estrous cycle or from subordinate follicles during estrus. Equine oocytes are especially firmly adhered to the follicle wall in a broad-based hillock of cumulus cells. Considerable intra-follicular turbulence is necessary to dislodge the oocyte to allow it to be flushed out of the follicle. The greatest productive oocyte yield will generally be from follicles of 10 to 20 mm diameter, because it is difficult to create the necessary turbulence in larger follicles, and oocytes in smaller follicles are often not meiotically competent. Regular examination can allow scheduling of the follicular aspiration when the largest population of follicles in the appropriate size range are present.

Follicular Aspiration

All Mares

Mares are restrained in stocks and tranquilized to effect with approximately 0 01 mg/kg detomidine HCl[o] or 0.66 mg/kg xylazine[p] intravenously. *N*-butylscopalammonium bromide[q] (0.9 mg/kg IV) is administered to encourage rectal relaxation and to prevent peristaltic waves from interfering with trans-rectal ovarian manipulation. The tail is tied up, the rectum is evacuated, and the vulva and perineum is cleaned. With a sleeved hand, approximately 2 oz of obstetrical lubricant is distributed over the face of the ultrasound probe and holder with the needle guide. A thin rectal sleeve is placed and then stretched over the probe and holder to prevent vaginal mucus and debris from being introduced into the needle guide channel. Obstetrical lube is applied to the exterior of this sleeve and the probe is guided into the vagina and placed against the dorsal anterior vaginal wall lateral to the cervix. The sleeved hand is withdrawn from the vagina and introduced into the rectum. The ovary is grasped and guided to the dorsal vaginal wall next to the transducer. This requires manipulating the ovary and lifting it up over the uterus and is best accomplished by grasping the ovarian suspensory ligament between two fingers. The ovary and probe are aligned in such a manner that the follicle(s) to be aspirated are adjacent to and aligned with the needle guide. Penetration of the vaginal wall, ovary, and follicle with the needle is facilitated by firm pressure of the ultrasound probe against the vaginal wall opposed by firm pressure of the ovary against the peritoneal surface of the vaginal wall. The needle is advanced to the center of the follicle, and the aspiration pump is started (Fig. 2).

Initially, the practitioner may find it easiest to simply manipulate the probe and the ovary while having an assistant advance the needle into the follicle. This works well with dominant follicles, but smaller follicles are more efficiently aspirated when the operator handles the needle as well as the probe and ovary. This allows all to move or be stabilized in synchrony with the timing of needle puncture; which is especially important if the animal is moving. The probe handle is held in the palm of the hand with forward pressure maintained by the heel of the hand. The fingers are then free to advance and manipulate the aspiration needle (Fig. 3).

DSF Oocytes

Once penetrated, the follicle is completely evacuated and then re-filled as rapidly as possible while the

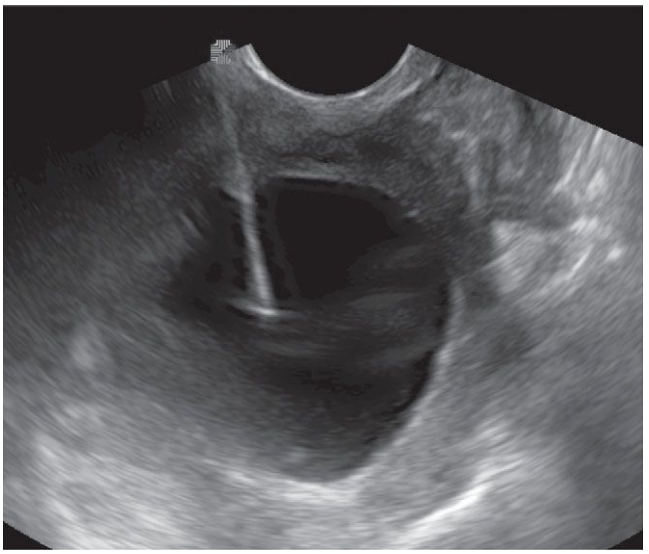


Fig. 2. DSF with needle advanced to center of follicle.

pump remains running. The speed of fluid refilling the follicle encourages the turbulence that is responsible for dislodging the cumulus oocyte complex from the follicle wall. When possible, the operator ballots the emptying follicle to increase turbulence during the evacuating phase as well. The follicle is sequentially filled and evacuated approximately 10 times as the volume in the collection bottle allows.

Oocytes from dominant stimulated follicles are quite temperature sensitive. Small decreases in temperature for even a short period can cause depolymerization of the meiotic spindle and subsequent fertilization abnormalities.[9,10] Equipment, labware, and solutions that the oocyte will come in contact with should be kept as close to body temperature as possible to prevent spindle damage. The flush solution should be pre-warmed, the collection bottles maintained at body temperature, and petri dishes for searching should be kept on a warming tray (Fig. 4).

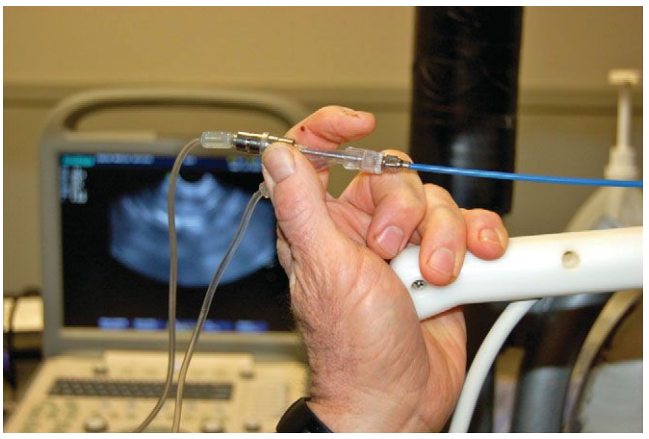


Fig. 3. Probe handle is held with the palm of the hand, allowing the thumb and first two fingers to manipulate the needle.

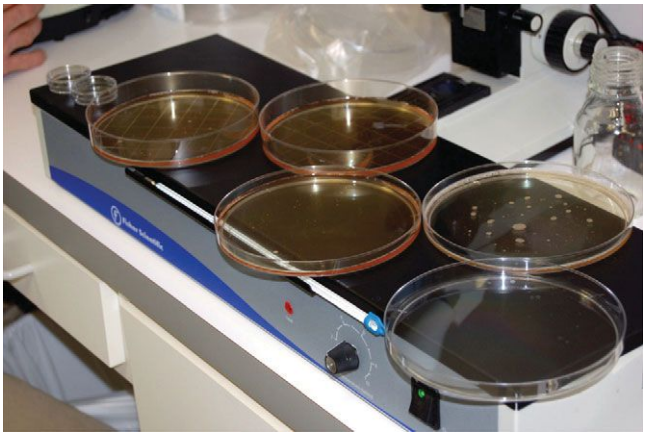


Fig. 4. Fluid from DSF aspiration distributed in 150-mm petri dishes on a warming tray.

IMM Oocytes

Once penetrated, the follicle is completely evacuated, then refilled as quickly as possible while the pump remains running, creating as much intra-follicular turbulence as possible. The follicle wall is lightly scraped with the needle by gently manipulating the probe and the ovary during aspiration. The needle can also be rotated to assist in freeing the oocyte from the follicle wall.

Generally, several small follicles can be aspirated with a single needle puncture. The ovary and needle guide can be manipulated to allow the needle to be advanced through the ovarian stroma into successive follicles. The aspiration pump is kept running as the needle is advanced from one follicle to the next but is turned off when the needle is withdrawn from the ovary to prepare for another puncture, or at the end of the procedure.

IMM oocytes are not as temperature-sensitive as DSF oocytes, but temperature shock is avoided to decrease cellular stress.

Search and Identification of Oocytes

DSF Oocytes

Fluid recovered from stimulated dominant follicles is usually bloody; to effectively search for a cumulus oocyte complex (COC), the fluid is distributed into 150-mm petri dishes on a warming tray. Blood is generally present from the aspirated fluid of dominant follicles that have responded to gonadotropins; therefore bloody aspirations are more likely to yield a maturing oocyte. Clear recovered fluid is more commonly recovered from follicles that have not responded to gonadotropins, although this is not always the case.

The petri dishes are individually searched for the COC with the use of the dissecting microscope at



Fig. 5. Clear COC from dominant gonadotropin-stimulated follicles (DSF) aspiration in search dish.

×10 to ×25 magnification. The COC often appears as a large, clear, fluffy mass of cells containing the oocyte and is easy to identify in the bloody fluid (Fig. 5). If it is not readily found, all cell masses should be closely examined. The oocyte appears as a partially dark sphere surrounded by the corona radiata in the center of the clear cumulus mass (Fig. 6). The cytoplasm of the meiotically competent oocyte generally appears heterogeneous in nature; it has dark areas and light areas caused by accumulations of lipids. Handling the COC, because of its large size, is performed with a 0.25-cc semen straw or a fire-polished glass pipette of similar diameter (1.5 mm).

IMM Oocytes

Recovered fluid from small follicles is variably contaminated by blood. Oocytes are located by filtering the fluid through an embryo collection filter and then further clarifying by passing more flush medium through the filter. The clarified fluid is rinsed into a 150-mm petri dish, which is then searched for



Fig. 6. COC from DSF aspiration.

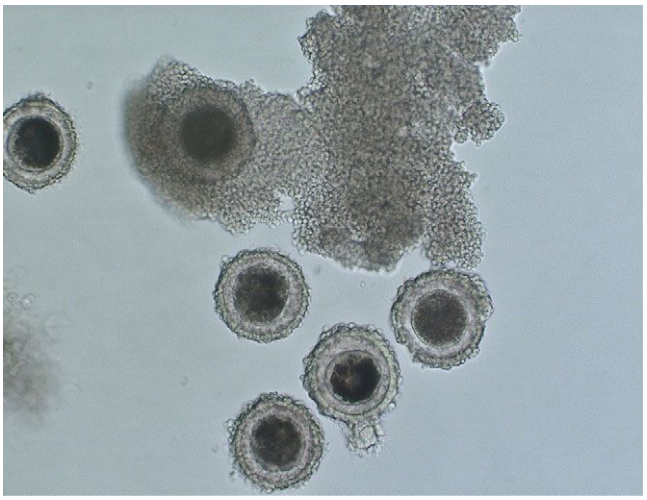


Fig. 7. Compact COC from IMM follicle aspiration.

oocytes. The COC of the IMM is usually quite dense, although occasionally an expanded cumulus is noted, but the expansion is not usually as dramatic as those of the DSF. Oocytes are usually found individually in the dish, or clumped with cellular debris, surrounded by only a few layers of cumulus cells or contained in a small, dense, relatively flat mass of cumulus cells (Fig. 7). These small COC can be handled effectively with a pipettor and 10-μL tips.

3. Results

Oocyte recovery rates are generally approximately 75% or more for DSF oocytes[11,12] and 50% to 60% for IMM oocytes,[3,13] although in the authors' experience, selection of follicles between 10 and 20 mm in diameter can increase percent recovery of IMM oocytes. Mares and breeds prone to multiple dominant follicles and larger numbers of subordinate follicles provide an opportunity for larger numbers of oocytes to be recovered per aspiration session, whereas older mares tend to have fewer subordinate follicles and will generally yield fewer IMM oocytes per aspiration session.

During 2012, in the authors' clinical practice, 223 aspiration sessions performed on 42 client-owned mares, between the ages of 12 and 26 years, yielded 126 intact DSF oocytes and 829 IMM oocytes. Of the 829 IMM oocytes that were collected, 611 matured to metaphase II (73.7%) as evidenced by extrusion of a first polar body. ICSI was performed on intact DSF oocytes and metaphase II IMM oocytes, resulting in 126 blastocysts; 44 of the blastocysts were produced from DSF oocytes (0.35 blastocyst per oocyte) and 82 from IMM oocytes (0.13 blastocyst per injected oocyte). Blastocysts (n = 106) were transferred non-surgically into recipient mares, resulting in 83 pregnancies (78.3%) at 14 days. Fifteen pregnancies were lost by 30 days

(18.1% embryo loss), and 20 of the blastocysts produced were cryopreserved.

4. Discussion

Ultrasound-guided transvaginal follicular aspiration is a useful technique for collection of oocytes for advanced reproductive techniques. The ability of the practitioner to successfully retrieve oocytes from mares will allow more owners access to these reproductive techniques. TVA is not a complex procedure, but most individuals require some practice to become proficient at the technique, and, to some extent, increased practice will continue to increase proficiency. One should endeavor to obtain this practice, preferably under the guidance of someone already experienced, and master the technique before attempting TVA on a client's animal.

TVA has been shown to be a safe procedure; rare but reported negative effects include internal hemorrhage,[14] adhesion development,[15] and ovarian abscessation.[16] The authors have only noted one case of ovarian abscessation in an estimated 1000+ clinical, research, and teaching TVA sessions. Repeated aspiration sessions have also shown no negative impact on cyclicity[15] or future fertility[17–19] of mares studied. This does not mean that the technique should be taken lightly; attention should be paid to proper technique, restraint, and aseptic technique. The risk of rectal tears is present with any rectal palpation procedure and ovarian manipulation.[16] Many of the mares presented for oocyte collection have chronic endometritis or pyometra, contributing to the likelihood of vaginal contamination that could be carried into the abdomen through needle puncture. Prophylactic treatment with antibiotics has been reported,[20] and the administration of ampicillin and gentamicin 10 minutes before TVA has been shown to have no adverse effect on blastocyst formation rates after ICSI,[21] although the administration of antibiotics may be considered unnecessary by some because of the relative infrequency of TVA-associated infection.

Restraint and analgesia for TVA are easier to attain in some mares than others. A dose of 300 mg of xylazine administered intravenously will provide adequate sedation for most pluriparous mares, but mares that are more fractious may require an increased dose of xylazine or detomidine or use of a muzzle twitch. Mares with DSF that are aspirated near ovulation may be sensitive to manipulation, requiring additional analgesia, as are mares in which significant tension on the ovarian suspensory ligament is necessary to position the ovary for TVA. Pain caused by tension of the suspensory ligament may be alleviated somewhat by the administration of flunixin meglumine before TVA, whereas ovarian sensitivity is best handled by increased dosage of detomidine or xylazine.

The TVA technique requires the ability of the operator to manipulate the ovary back to the cranial surface of the vaginal wall. This can be difficult in mares with short ovarian suspensory ligaments and short vaginal vaults, which is often the case in maiden mares. Micro-convex ultrasound probes better facilitate aspiration in these mares compared with linear-array probes because the orientation of the probe in the needle guide allows the ovary to be in front of the micro-convex probe, whereas it must be pulled farther caudally to be on top of the linear probe (Fig. 8).



Fig. 8. Demonstration of the orientation of the needle exiting both the micro-convex probe and linear probe needle guides.

Scheduling of an aspiration session depends somewhat on the intended type of recovered oocyte. TVA for recovery of DSF oocytes or DSF with IMM oocytes is generally scheduled on the basis of ultrasound examinations and progression of the donor mare's estrous cycle, although aspiration on a 14-day interval without ultrasound examination[21] was found to be effective for both DSF and IMM oocytes. Aspirations performed for collection of only IMM oocytes can generally be performed at the convenience of the mare manager and veterinarian, although periodic ultrasound examinations can help select a time with higher follicle numbers. TVA performed at an interval less than every 10 to 11 days may deplete follicle numbers, resulting in fewer follicles aspirated per session.[19,20]

Maintenance of aspiration equipment is paramount for mare safety as well as the effectiveness of the procedure. Sterilization of the aspiration needles is somewhat problematic because of their size as well as the combination of metal and plastic parts. Gas sterilization can be used, but a significant de-gassing time period is necessary because of the extreme sensitivity of oocytes to toxins. Dry-heat sterilization has been an effective alternative in the authors' practice. Heating in a common household oven at 170° F for 2 hours or longer sterilizes the needle and tubing as long as it is com-

pletely dry, although the authors typically "bake" them overnight. Needles will dull with repeated uses, so replacing or re-sharpening is necessary. The authors sharpen needles with an ultra-fine ceramic stone[r] under direct visualization through a dissecting microscope as necessary or after three to four aspiration sessions. Sterilization of the ultrasound probe and needle guide is accomplished through immersion in glutaraldehyde for 20 minutes. The probe and guide assembly are then thoroughly rinsed with distilled or deionized water and allowed to air-dry.

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[a]Needle guide, Minitube of America, Inc, Verona, WI 53593.
[b]60-cm, double-lumen, 12-gauge oocyte aspiration needle, Minitube of America, Inc, Verona, WI 53593.
[c]CDI Control Devices Valve, Relief, ¼-inch, 5z763, Grainger, Lake Forest, IL 60045.
[d]Gast Roc-R Vacuum Pump 1/8 hp model 5z669, Grainger, Lake Forest, IL 60045.
[e]Kimax, Kimble Chase Life Science and Research Products, LLC, Rockwood, TN 37854.
[f]Corning, Inc, Corning, NY 14831.
[g]Fisher Scientific, Waltham, MA 02454.
[h]Embryo transfer low volume filter, Veterinary Concepts, Inc, Spring Valley, WI 54767.
[i]Vigro Complete Flush, Bioniche Animal Health, Belleville, Ontario K8N5J2, Canada.
[j]Heparin sodium injection, Sagent Pharmaceuticals, Schaumburg, IL 60195.
[k]Controlled flushing set CFS36, Mila International, Inc, Erlanger, KY 41018.
[l]20-mL Norm-Ject. Restec Corp, Bellefonte, PA 16823.
[m]CidexPlus, Johnson and Johnson, Irvine, CA 92618.
[n]SucroMate, Thorn BioScience, LLC, Louisville, KY 40204.
[o]Dormosedan, Orion Corp, Espoo, Finland; Zoetis, Madison, NJ 07940.
[p]AnaSed, Lloyd Laboratories, Shenandoah, IA 51601.
[q]Buscopan, Boehringer Ingelheim Vetmidica, Inc, St. Joseph, MO 64506.
[r]Spyderco, Inc, Golden, CO 80403.

Effect of Potential Oocyte Transport Protocols on Blastocyst Rates After Intracytoplasmic Sperm Injection in the Horse

Rob Foss, DVM*; Hunter Ortis, DVM; and Katrin Hinrichs, DVM, PhD, Diplomate ACT

Intracytoplasmic sperm injection (ICSI) is used to produce foals from otherwise infertile mares and from stallions with limited sperm stores. Data from this study provide practical information supporting transport of oocytes for ICSI. Shipment of oocytes to an ICSI laboratory would allow mares to remain under the care of their primary veterinarians. Authors' addresses: Equine Medical Services, Inc, 5851 Deer Park Road, Columbia, MO 65201 (Foss and Ortis); Departments of Veterinary Physiology and Pharmacology and Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX 77483 (Hinrichs); e-mail robertfoss@centurytel.net. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Intracytoplasmic sperm injection (ICSI) is used clinically to produce foals, but only at limited locations. The objectives of this study were to determine and define protocols that would allow successful transport of oocytes for ICSI.

2. Materials and Methods

Oocytes were recovered from dominant follicles 24 hours after deslorelin administration (DSF follicles) or from subordinate follicles (immature, IMM). DSF oocytes were incubated overnight under differing conditions before ICSI to model transport. IMM oocytes were placed in various conditions overnight before in vitro maturation, followed by ICSI. The rate of blastocyst production was compared among treatments.

3. Results

Blastocysts were produced in all groups. The highest blastocyst production in DSF oocytes (70%) was obtained after holding in sealed tubes in pre-equilibrated control maturation medium maintained at 37°C. IMM oocytes were held overnight for one or two nights in modified M199 medium or for one night in commercial embryo holding solution, in air at room temperature, yielded 35% to 37% blastocyst production.

4. Conclusions

A commercially available medium can be used for shipping IMM oocytes with good resulting blastocyst rates. Better blastocyst rates are obtained from DSF oocytes, but these are more sensitive to temperature changes and delays. Further research is needed to determine the ability of blastocysts produced from the various protocols to establish normal pregnancies.

Research Abstract

NOTES

Autogenous Transfer of Intracytoplasmic Sperm Injection–Produced Equine Embryos Into Oocyte Donor Uteri

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Oocytes can be collected from late-estrous mares for intracytoplasmic sperm injection (ICSI); the resulting embryos can be transferred back into the uterus of the oocyte donor to establish pregnancy. Authors' address: Colorado State University, 3103 Rampart Road, Equine Reproduction Lab, Fort Collins, CO 80521–3003; e-mail: elaine.carnevale@colostate.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Intracytoplasmic sperm injection (ICSI) has been used to produce embryos from stallions with poor-quality sperm or limited sperm availability, but the procedure is expensive and typically requires a recipient to carry the ICSI-produced embryo. The aim of this project was to establish pregnancy by transfer of ICSI-produced embryos into the uteri of oocyte donors.

2. Materials and Methods

Oocytes were collected from estrous mares ($n = 9$) during 17 cycles (one to three cycles per mare). When a follicle >33 mm was present, an ovulation-inducing agent was administered, and oocytes were collected 22 to 27 hours later. Oocytes were matured and injected with sperm 44.5 to 50.5 hours after collection. Resulting embryos were cultured until blastocyst development. Embryos were transferred into their respective oocyte donor's uterus at 6 to 7 days after ICSI. Pregnancy was diagnosed by transrectal ultrasonography at 5 and 9 days after embryo transfer.

3. Results and Discussion

Twelve oocytes were collected, and 10 embryos were produced. Two embryos failed to develop past the early morula stage, and eight embryos developed into blastocysts. Five pregnancies resulted from the 12 oocytes (5/12, 42% pregnancy per oocyte) and from the eight transferred blastocysts (5/8, 62.5% pregnancy per transferred blastocyst).

4. Conclusions

Pregnancy was established after the transfer of ICSI-produced embryos into the uteri of the respective oocyte donors.

Research Abstract

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Estrogens as Potential Diagnostic Markers in Mares With Experimentally Induced Ascending Placentitis

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These preliminary results suggest that determination of 17β-estradiol sulfate but not estrone sulfate may be a useful diagnostic marker for ascending placentitis in mares. Authors' addresses: Gluck Equine Research Center, Department of Veterinary Science, University of Kentucky, Lexington, KY 40546 (Ball, Canisso, Esteller-Vico, Troedsson); K.L. Maddy Equine Analytical Chemistry Laboratory, University of California, Davis, CA 95616 (Stanley); e-mail: b.a.ball@uky.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

There is a critical need for sensitive measures for early detection of ascending placentitis in mares. The objective of this study was to evaluate plasma concentrations of estrogens in mares: (1) with experimentally induced placentitis (with/without fetal fluid sampling); and (2) carrying normal pregnancies (with/without fetal fluid sampling).

2. Materials and Methods

Mares (260–280 days of gestation) were assigned to the following experimental groups: (1) control mares with ($n = 2$) or without fetal fluid sampling ($n = 2$) and (2) mares with induced ascending placentitis with ($n = 4$) or without fetal fluid sampling ($n = 6$). Placentitis was induced by means of intracervical inoculation of *Streptococcus equi spp. zooepidemicus*. Blood samples were obtained at inoculation (day 0) and then daily for 6 days. The concentration of estrone sulfate was determined by immunoassay, and the concentration of 17-β estradiol sulfate was determined by mass spectrometry. The data were analyzed by means of a mixed model with mare as a random effect.

3. Results and Discussion

Treated mares (9/10) aborted 6.7 $\pm$ 0.5 days after inoculation, and one control mare aborted subsequent to fetal fluid sampling. The treated mare that failed to abort and the control mare that did abort did not have signs of placentitis and were excluded from the analysis. Estrone sulfate concentrations did not differ between control and inoculated mares; however, 17β-estradiol sulfate decreased significantly ($P < 0.05$) within 1 day after

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inoculation in treated mares compared with control mares. These data indicate that estrone sulfate was not a useful biomarker for acute placentitis in mares, whereas 17β-estradiol sulfate decreased precipitously in mares subsequent to experimental induction of ascending placentitis.

Acknowledgments

This research was supported by the Albert G. Clay Endowment in equine reproduction, by the Department of Veterinary Science, and by the Geoffrey Hughes Fellowship at the University of Kentucky.

Decreasing pH of Mammary Gland Secretions Predict Foaling and Are Correlated With Electrolyte Concentrations in Prefoaling Mares

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Mammary secretion pH declined significantly in mares within 24 hours before foaling, with 11 of 14 mares foaling in <24 hours when the pH level was ≤7. The pH of mammary secretions was correlated ($P < 0.001$) with Na^+ ($r = 0.87$), Cl^- ($r = 0.85$), Ca^{2+} ($r = -0.88$), and K^+ ($r = -0.8$). These results suggest that serial assessment in the pH of mammary gland secretions can replace serial electrolyte measurements. Authors' addresses: Reproduction Laboratory, The Maxwell H. Gluck Equine Research Center, Department of Veterinary Science, University of Kentucky, Lexington KY 40546-0099 (Ball, Canisso, Davolli, Troedsson); Faculdade de Medicina Veterinária e Zootecnia, Departamento de Radiologia e Reprodução Animal, Universidade Estadual Paulista, Botucatu, São Paulo, 18618-970, Brazil (Silva); e-mail: b.a.ball@uky. *Corresponding author; †presenting author. © 2013 AAEP.

1. Introduction

Methods to predict impending parturition in mares allow close supervision and provision of support to the mare and foal during parturition. Serial electrolyte measurements in mammary gland secretions are commonly used to predict foaling. A recent study reported that a reduction in the pH of mammary gland secretions could predict parturition; therefore, the objectives of this study were to determine the pH of mammary gland secretions and corresponding associations with electrolyte concentrations in prefoaling mares.

2. Materials and Methods

Fourteen normal foaling mares were monitored daily from 310 to 320 days of gestation age until parturition. The mares underwent daily physical examinations between 5:30 and 6:30 PM. Prefoaling mammary gland secretions were collected, and the pH was immediately determined by a portable device[a] coupled with a semi-micro electrode. The pH readings and pH meter calibrations were performed according to manufacturer's instructions with the use of buffer solutions (ie, acid buffer pH = 4, neutral pH = 7). A small aliquot of mammary secretion was frozen (−20°C) until further analyses. After parturition, samples from day −4 to day 0 (day of foaling) were thawed, and electrolyte concentrations were determined with an automated analyzer.[b] The electrolytes analyzed included Ca^{2+}, Mg^{2+}, Na^+, K^+ and Cl^-. The data were analyzed by means of repeated-measures analysis of variance, and individual means were compared by means of a

Research Abstract

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t test.[c] Correlations were determined between pH and electrolyte concentrations[c] by the Pearson product-moment for each pair.

3. Results and Discussion

There was significant reduction in the pH of mammary secretions on the day of foaling ($P < 0.0001$), and most mares (11/14) with a pH $\leq$7 foaled within 24 hours. The pH of mammary secretions of all mares on the day of foaling was 7.0 $\pm$ 0.16 (mean $\pm$ SEM). In addition, there were significant ($P < 0.05$) increases in Ca^{2+} and K^{+} concentrations and significant decreases in Na^{+} and Cl^{-} concentrations from 1 day before to the day of foaling. The pH of mammary secretions was highly and significantly ($P < 0.001$) correlated with Na^{+} ($r = 0.87$), Cl^{-} ($r = 0.85$), Ca^{2+} ($r = -0.88$), and K^{+} ($r = -0.8$) concentrations. These results suggest that serial assessment in the pH of the mammary gland secretions can replace serial electrolyte measurements.

Acknowledgments

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Footnotes

[a]Fisher Scientific Accumet AP 115 Portable pH Meter, Thermo Fisher Scientific, Inc, Marietta, OH 45750.
[b]AU480 Beckman Counter, CH 1260 Nyon Switzerland.
[c]JMP 9, SAS Institute, Cary, NC 27513.

Effects of Ergot Alkaloids on the Breeding Stallion

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Ergot alkaloid ingestion causes problems in pregnant mares. In stallions, there was a lower gel-free volume but no negative effects of ergot alkaloid on the spermiogram (sperm motility, concentration, cell morphology, and total number of cells), testosterone concentration, and body and scrotal temperature. However, in colts, ergot alkaloids induced a significant increase in the proportion of pachytene spermatocytes showing unpaired sex chromosomes. Authors' addresses: SoRho Vet Fertility Services, PO Box 217, Winterville, GA 30683-0217 (Fayrer-Hosken); College of Agricultural Economics (Hill); College of Agriculture (Heusner, Traylor, Turner); University of Georgia, Athens, GA 30602; e-mail: richardfh@me.com. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

The objective of this study was to investigate the effect of ergot alkaloids from endophyte-infected tall fescue on the stallion's reproductive functions, including the breeding soundness examination (BSE) spermiogram.

2. Materials and Methods

Six stallions were fed either endophyte-infected fescue seed or a nontoxic endophyte fescue seed[a] in a crossover study. The stallions were fed the diet for 70 days, then rested for at least 70 days (no fescue seed), and then fed fescue seed for a second 70 days.

3. Results

Maximal systemic levels were reached 8.33 hours after starting seed ingestion, and the systemic alkaloid levels fell to control levels within 48 hours after cessation. Between the experimental and control stallions, there were no statistical differences in the spermiogram, testicular volume, baseline, and human chorionic gonadotropin (hCG) stimulation of testosterone levels. Only the stallion's' ejaculate had significantly ($P < 0.01$) lower gel-free volume than did controls.

4. Discussion

Ergot alkaloids have minimal detectable effects on the spermiogram parameters, and the only reproducible change is a decreased gel-free volume of stallions consuming high levels of ergot alkaloids.

Footnote

[a]MaxQ, Pennington Seed, Inc., Madison, GA 30655.

Research Abstract

NOTES

Progression of Reproductive Changes Accompanying Testicular Dysfunction in Aging Thoroughbred Stallions: Case Studies

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Regular breeding soundness examinations in aging stallions are warranted to detect early changes that might predict onset of declining fertility. Management changes could then be instituted that might improve pregnancy rates during this period and until testicular dysfunction becomes so pronounced that a stallion must be retired for infertility. Authors' addresses: Department of Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX 77843-4475 (Blanchard, Brinsko, Varner, Love), and Equine Medical Associates, PSC, Lexington, KY 40583-3166 (Morehead); e-mail: tblanchard@cvm.tamu.edu. *Corresponding and presenting author. © 2013 AAEP.

1. Introduction

Superior stallions can command a sizeable book of mares to be bred each year throughout their reproductive lifespan. Many stallions achieve good pregnancy rates when bred beyond 20 years of age, but eventually fertility begins to decline. A myriad of causes can contribute to declining fertility in aging stallions, including testicular dysfunction.[1] As the ability to produce sufficient numbers of normal, motile sperm in ejaculates declines, so do pregnancy rates until the stallion eventually becomes so subfertile that it is no longer commercially feasible to continue breeding the horse. The purpose of this study was to evaluate our findings in three aging Thoroughbred stallions over this period of declining fertility to determine when testicular dysfunction began as well as to characterize the progression of the disorder. These data have been used to formulate changes in breeding management for aging stallions with testicular dysfunction.[2]

2. Materials and Methods

Stallions, Semen Collection, and Testicular Measurement

Three aging Thoroughbred stallions were evaluated for breeding soundness over several years as their fertility declined. Stallions were usually evaluated at least once yearly (typically between November and January): Stallion 1 from 20 through 24 years of age; Stallion 2 from 18 through 27 years of age; and Stallion 3 from 18 through 24 years of age. Stallions were mated several times to mares in estrus to stabilize extragonadal sperm reserves before

NOTES

semen was collected. After 1 to 2 days of sexual rest, semen collections (two to four ejaculates over a 1- or 2-day period) were accomplished with the use of an artificial vagina[a] equipped with an in-line nylon micromesh filter[b] to permit collection of gel-free semen.[1] Before semen collection, each stallion was exposed to a mare in estrus to stimulate sexual interest. The erect penis was rinsed thoroughly with water and cotton immediately before semen collection and was dried with paper towels. Each stallion was then allowed to mount a mare in estrus, and the penis was deflected into the artificial vagina. After semen collection, the gel-free semen was transported to an adjacent laboratory and placed in an incubator (37°C) before processing (see "Semen Evaluation"). Daily sperm output (DSO) was estimated as previously described.[3]

Testicular measurements were obtained by means of transcutaneous ultrasonography, and the length, width, and height of each testis was used to calculate testicular volume. Total testicular volume was represented by adding the volume of the left and right testis. Predicted DSO was calculated on the basis of total testicular volume.[4]

Semen Evaluation

Volume of gel-free semen for each ejaculate was measured in a graduated cylinder. Sperm concentration was determined with the use of an Equine Densimeter,[c] unless concentration was dilute (ie, $<100 \times 10^6$/mL), in which case hemacytometer counts were used to determine sperm concentration.[1] The total sperm number was determined by the product of semen volume $\times$ concentration. Gel-free semen was extended in one of two extenders[d,e] at a 9:1 ratio (extender:semen), and placed in an incubator for 15 minutes. Sperm motility (total/progressive; percentage) was assessed subjectively by one experienced examiner with the use of a phase-contrast microscope with a warming stage at $\times$250 magnification.[1]

Aliquots of raw semen were mixed with buffered formol saline and stored until evaluated for sperm morphologic characteristics with the use of a differential interference contrast microscope at $\times$1250 magnification.[1] One hundred sperm were evaluated per specimen, and all morphologic features (normal, proximal droplet, distal droplet, abnormal head, bent midpiece, swollen or irregular midpiece, bent or broken tail, detached head, coiled tail, abnormal acrosome, or premature germ cell) were recorded and expressed as percentages.[5]

Determination of Spermatogenic Efficiency

Spermatogenic efficiency was calculated by as a percentage, on the basis of predicted sperm output ([actual DSO/predicted DSO from testicular measurements] $\times$ 100).[6]

Sperm Chromatin Structure Assay

One-milliliter aliquots of raw semen from the last ejaculate collected at each examination period were pipetted into 1.2-mL cryovials, placed on dry ice, and shipped frozen to the laboratory for storage at −80°C until assayed for sperm DNA integrity by the Sperm Chromatin Structure Assay as previously described.[7] Values recorded for each assay were mean α-t and %COMP α-t.

Hormonal Assays

When assays were performed to determine circulating hormone concentrations, jugular blood was collected into 7-mL heparinized tubes once hourly for 4 hours in the morning. Tubes were centrifuged, and equal volumes of plasma were pipetted from each hourly sampling into 2-mL cryovials that were frozen and shipped to a commercial endocrine laboratory on dry ice. Radioimmunoassays were performed to determine circulating concentrations of testosterone, total estrogens, luteinizing hormone (LH), and follicle-stimulating hormone (FSH) and, in some instances, inhibin.

On some occasions (when resting testosterone concentration was low), a gonadotropin (GnRH; 100 μg, IV) stimulation test was performed. Jugular blood samples were collected immediately before GnRH administration and at 15, 30, 60, and 120 minutes after GnRH administration for assay of testosterone concentrations.[8]

Pregnancy Rates

Pregnancy rates per cycle (PR/C) and per season (SPR) were determined from analysis of breeding records.

Normalized Data

Additionally, PR/C, spermatogenic efficiency, percentage of the maximal testicular volume (Max volume; the volume obtained on the first year each stallion's testes were measured), and total number of progressively motile, morphologically normal (PMMN) sperm at DSO were normalized to the last year each stallion bred mares (year of retirement because of infertility during that breeding season) and averaged among the three stallions.

3. Results

Pregnancy rates and findings from breeding soundness examinations over a 5-year period of declining fertility for Stallions 1 through 3 are presented in Tables 1, 2, and 3, respectively. Fig. 1 provides results of the data normalized to the last year at stud and averaged among stallions.

Early in the course of progression of testicular dysfunction in these aging stallions, a period of decreasing sperm output occurred before a remarkable change in testicular size. The diminished sperm output resulted from lowered spermatogenic efficiency. Pregnancy rates often were not markedly affected early in this period. Before testicular size was noticeably decreased, sperm output, spermatogenic efficiency, and PR/C declined dramatically. Eventually, SPR declined as well. Although these

Table 1. Breeding Soundness Examination Findings for Stallion 1 Between 20 and 24 Years of Age

Age, y	Book	PR/C	SPR	TTV	DSO	Effic	%MN	%PM	Mean	COMP	Test	Est	LH	FSH	Inh	Test Res
19	79	55%	86%	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE
20	73	30%	67%	382	5.80	73%	43%	40%	NE	NE	NE	NE	NE	NE	NE	NE
21	63	31%	70%	380	3.48	43%	45%	30%	241	16%	NE	NE	NE	NE	NE	NE
22	53	21%	45%	312	2.09	32%	50%	25%	239	16%	650	275	2.0	2.1	NE	1300
23	40	25%	48%	308	2.06	32%	25%	15%	257	31%	784	156	11.0	10.2	NE	1126
24	20	7%	15%	260	1.74	32%	25%	15%	286	31%	650	156	NE	NE	NE	980

NE indicates not examined; Book, number of mares bred in season; PR/C, pregnancy rate per cycle, %; SPR, seasonal pregnancy rate, %; TTV, total testicular volume, cc; DSO, daily sperm output, $\times 10^9$; Effic, spermatogenic efficiency, %; %MN, percentage of morphologically normal sperm; %PM, percentage of progressively motile sperm, SCSA values; Mean, Mean-αt; COMP, %COMP-αt (percentage of cells outside the main population); Test, circulating testosterone concentration, pg/mL (normal range, 500–2000); Est, circulating total estrogens concentration, pg/mL (normal range, 150–400); LH, circulating luteinizing hormone concentration, ng/mL (normal range, 0.5–5.0); FSH, circulating follicle-stimulating hormone concentration, ng/mL (normal range, 0.5–15.0); Inh, circulating inhibin concentration, ng/mL (normal range, 2.2–3.4); Test Res, highest concentration of testosterone within 2 hours of GnRH administration, pg/mL (normal increase ≥100% within 2 hours).

changes were associated with a decrease in percentage of morphologically normal and progressively motile sperm in ejaculates, sperm chromatin structure assays did not reveal prominent changes in sperm DNA integrity. It was only late in the course of progression of testicular dysfunction (last 2 years of breeding) that sperm DNA integrity began to deteriorate. Baseline hormonal concentrations usually did not differ from reported normal ranges until after spermatogenic efficiency had declined to ≤40%, DSO approximated ≤2 billion, total number PMMN sperm at DSO was <500 million, and the average PR/C diminished to ≤33%. Sperm motility and morphology values typically but not always deteriorated from those found when each horse was still achieving normal pregnancy rates. Low total estrogen concentration was sometimes noted in the last 1 to 3 years of breeding (ie, two of three stallions). We also noted a low circulating concentration of testosterone in two stallions during this same time period (ie, 116–403 pg/mL), and sometimes testosterone did not achieve the lower limit of reported normal circulating concentration (ie, ≥500 pg/mL) within 2 hours of GnRH administration in one of three stallions. Total testicular volume during the last year at stud decreased to approximately 50% of the previous size when the stallions were several years younger. At this late stage of testicular dysfunction, variations from normal endocrine values were more likely to include low estrogen, testosterone and inhibin concentrations, and, in some instances, high FSH and LH concentrations.

4. Discussion

These findings support the premise that age-related testicular dysfunction follows a progressive pattern of deterioration. First, a decline in sperm output, including total number of normal motile sperm, is observed in ejaculates. This may be detected during pre-season breeding soundness examination but sometimes is found later during the breeding sea-

Table 2. Breeding Soundness Examination Findings for Stallion 2 Between 18 and 27 Years of Age

Age, y	Book	PR/C	SPR	TTV	DSO	Effic	%MN	%PM	Mean	COMP	Test	Est	LH	FSH	Inh	Test Res
17	88	70%	97%	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE
18	103	68%	88%	514	8.22	75%	47%	45%	232	18%	NE	NE	NE	NE	NE	NE
19	108	67%	85%	508	8.53	78%	45%	45%	250	20%	NE	NE	NE	NE	NE	NE
21	112	55%	84%	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE
22	92	52%	86%	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE
23	81	48%	83%	459	3.41	35%	49%	55%	234	9%	460	276	0.05	0.05	1.96	750
24	76	57%	88%	442	3.77	40%	43%	50%	231	15%	593	235	0.5	0.08	NE	645
25	78	34%	68%	360	1.73	23%	41%	30%	252	20%	274	231	1.8	0.8	4.19	494
26	61	19%	44%	346	1.50	21%	47%	25%	295	19%	NE	NE	NE	NE	NE	NE
27	26	0%	0%	299	1.00	17%	42%	25%	275	42%	116	128	2.8	0.8	3.99	370

NE indicates not examined; Book, number of mares bred in season; PR/C, pregnancy rate per cycle, %; SPR, seasonal pregnancy rate, %; TTV, total testicular volume, cc; DSO, daily sperm output, $\times 10^9$; Effic, spermatogenic efficiency, %; %MN, percentage of morphologically normal sperm; %PM, percentage of progressively motile sperm, SCSA values; Mean, Mean-αt; COMP, %COMP-αt (percentage of cells outside the main population); Test, circulating testosterone concentration, pg/mL (normal range, 500–2000); Est, circulating total estrogens concentration, pg/mL (normal range, 150–400); LH, circulating luteinizing hormone concentration, ng/mL (normal range, 0.5–5.0); FSH, circulating follicle-stimulating hormone concentration, ng/mL (normal range, 0.5–15.0); Inh, circulating inhibin concentration, ng/mL (normal range, 2.2–3.4); Test Res, highest concentration of testosterone within 2 hours of GnRH administration, pg/mL (normal increase ≥100% within 2 hours).

Table 3. Breeding Soundness Examination Findings for Stallion 3 Between 18 and 24 Years of Age

Age, y	Book	PR/C	SPR	TTV	DSO	Effic	%MN	%PM	Mean	COMP	Test	Est	LH	FSH	Inh	Test Res
17	94	67%	92%	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE
18	93	57%	84%	331	6.98	100%	42%	55%	NE	NE	NE	NE	NE	NE	NE	NE
19	79	49%	84%	330	6.20	93%	40%	50%	230	7%	NE	NE	NE	NE	NE	NE
20	93	54%	87%	331	5.84	87%	40%	50%	225	6%	NE	NE	NE	NE	NE	NE
21	57	55%	84%	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE
22	69	44%	80%	220	2.43	54%	43%	45%	233	16%	535	170	0.5	0.4	NE	NE
23	74	48%	80%	174	1.90	40%	40%	45%	217	21%	334	NE	13.1	23.4	1.77	NE
24	19	0%	0%	91	0.65	35%	25%	15%	278	31%	403	96	3.0	0.8	NE	610

NE indicates not examined; Book, number of mares bred in season; PR/C, pregnancy rate per cycle, %; SPR, seasonal pregnancy rate, %; TTV, total testicular volume, cc; DSO, daily sperm output, $\times 10^9$; Effic, spermatogenic efficiency, %; %MN, percentage of morphologically normal sperm; %PM, percentage of progressively motile sperm, SCSA values; Mean, Mean-αt; COMP, %COMP-αt (percentage of cells outside the main population); Test, circulating testosterone concentration, pg/mL (normal range, 500–2000); Est, circulating total estrogens concentration, pg/mL (normal range, 150–400); LH, circulating luteinizing hormone concentration, ng/mL (normal range, 0.5–5.0); FSH, circulating follicle-stimulating hormone concentration, ng/mL (normal range, 0.5–15.0); Inh, circulating inhibin concentration, ng/mL (normal range, 2.2–3.4); Test Res, highest concentration of testosterone within 2 hours of GnRH administration, pg/mL (normal increase $\geq$100% within 2 hours).

son, when pregnancy rates are noted to be lower than expected. As illustrated in Figure 1, the decline in output of normal motile sperm and the decline in spermatogenic efficiency precedes a decrease in testicular size. Decreased spermatogenic efficiency has been associated with an increased rate of germ cell degeneration during spermatogenesis.[9,10] As spermatogenic efficiency declines, a higher percentage of morphologically abnormal sperm and fewer motile sperm are often present in the ejaculate.[10] Thus, when testicular size began decreasing dramatically in the latter 2 to 3 years before forced retirement, sperm output dropped precipitously despite an apparent plateau in spermatogenic efficiency.

Our findings in these three stallions confirm that testicular dysfunction can become quite pronounced before sperm DNA integrity becomes severely compromised, as has been reported previously.[10] We also noted an increase in mean-αt in two stallions during their latter 2 years before forced retirement. Whereas the %COMP α-t measures the proportion of cells outside the main population of typically normal cells, an elevated mean-αt suggests that a greater proportion of sperm in the main population of the ejaculate has DNA of compromised integrity.

Others have described that aging stallions with more mild testicular dysfunction may not have significant changes in circulating hormone concentrations.[11–15] As testicular dysfunction worsens, endocrine changes appear. Roser[15] hypothesized that stallions with declining fertility usually first exhibit increasing circulating concentrations of FSH, followed later by decreasing concentrations of estradiol and inhibin, and, with further progression of testicular dysfunction, circulating concentrations of testosterone decline.[15] Besides determining baseline circulating concentrations of the above-mentioned hormones, Roser also recommends GnRH and human chorionic gonadotropin (hCG) stimulation tests to identify stal-

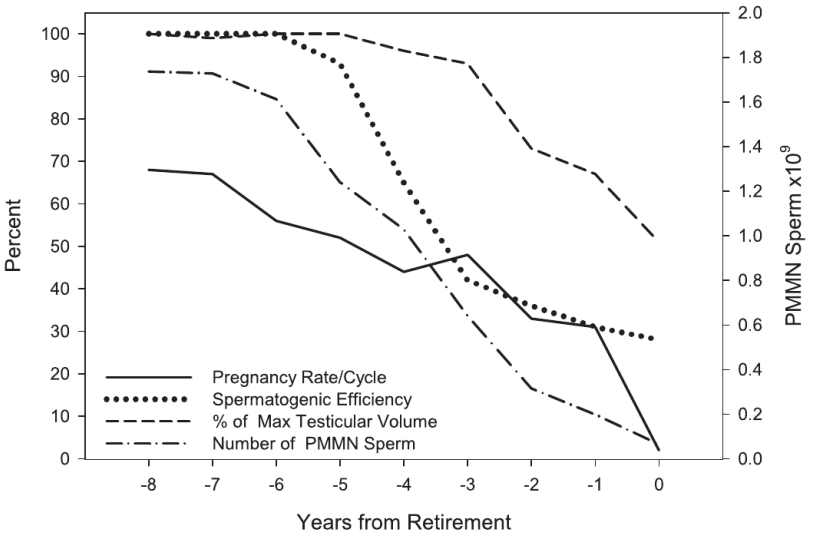


Fig. 1. Pregnancy rate per cycle (%), spermatogenic efficiency (%), percentage of initial (Max) testicular volume recorded at first examination, and total number of progressively motile morphologically normal (PMMN) sperm in ejaculates at daily sperm output (DSO) in three aging Thoroughbred stallions. Data are normalized to year of retirement because of infertility for each stallion and averaged among stallions.

lions with poor testosterone response.[15] Although global conclusions for all stallions cannot be drawn from only three stallions in this study, two were noted to have low concentrations of testosterone (or testosterone and estrogens) as the earliest detected variation from normal hormone concentrations.

Summary

Regular breeding soundness examinations in aging stallions are warranted to detect early changes that might predict onset of declining fertility. Management changes (eg, decreasing mating frequency, reinforcement breeding, mating closer to ovulation, and/or planned multiple matings per estrus, as previously described[2]) could then be instituted that might improve pregnancy rates during this period and until testicular dysfunction becomes so pronounced that a stallion must be retired for infertility.

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[a]Missouri-model artificial vagina, Nasco, Ft Atkinson, WI 53538.

[b]Disposable Nylon Mesh Gel Filters, Animal Reproduction Systems, Chino, CA 91710.

[c]Equine Densimeter, Animal Reproduction Systems, Chino, CA 91710.

[d]EZ Mixin-CST, Animal Reproduction Systems, Chino, CA 91710.

[e]INRA96, IMV Technologies, L'Aigle, France.

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