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Cover photo by Dr. Nat White.
Equine Veterinary Education is a refereed educational journal designed to keep the practicing veterinarian up to date with developments in equine medicine and related sciences. Case reports are accompanied by invited reviews, both invited and submitted, to further the professional development of its members, and to provide resources and leadership for the benefit of the equine industry.


Communications regarding editorial matters should be addressed to: The Editor, Equine Veterinary Education, Mulberry House, 31 Market Street, Fordham, Ely, Cambridgeshire CB7 5LQ, UK. Telephone: 44 (0) 1638 720250, Fax: 44 (0) 1638 721868, Email: sue@evj.co.uk.

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Published monthly. Deadlines are the seventh of the preceding month.

Address advertising inquiries to Dana Kirkland (859) 233-0147 / dkirkland@aaep.org

AEP 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.
Wellness: Self-compassion on ‘Tulsa Time’

By Aimee Eggleston, DVM

Highlights:

The veterinary profession is at risk for mental and addictive ill health.

Self-compassion helps strengthen emotional resilience.

Practitioners must care for themselves as much as they care for animals and their owners.

In August 2003, a pudgy, sweet 8-week-old black lab named Tulsa easily inserted herself into my life. For most of the next 13 years, Tulsa was my daily and steadfast work companion. Sitting beside me in the cab of my truck after many an equine euthanasia, she would soothe my frayed nerves; sometimes her thick coat would catch my tears. Last November, Tulsa lost her life at the hands of an aggressive cancer. I held Tulsa on that day as my veterinarian friend tended to her. I petted Tulsa’s silky ears and, for the last time, her coat caught my tears as she passed from this world.

I buried Tulsa’s ashes next to the forsythia bush in my backyard. As I dug the hole, burying the shovel in the dirt over and over, I embraced the anger that came over me. As I gently placed the box containing Tulsa’s ashes in the ground, I embraced my grief for the loss of my beloved pet. I placed the marker over the freshly disturbed dirt; flowers next to the marker. When I was done, tears flowed down on Tulsa yet again.

I describe this moment in my life because it is an experience that many of us share. For myself and many of my veterinarian friends, our pets have been some of the toughest and most important strands of the tapestry of our lives—as we navigated veterinary school, internships, residencies and our first jobs. We love them hard and with abandon; they are our silent advisers and counselors, the constants in our lives. To recognize their loss as anything less than the loss of a family member is to deny ourselves the very thing we share freely every day with clients and patients: compassion.

I also share the story of my loss of Tulsa because her connection to euthanasia, both at the end of her life and as my work companion of so many years, locates this issue of compassion within our veterinary profession. In our veterinary practices, we regularly see the pain etched on an owner’s face when confronted with the loss of their pet. At these moments our compassion is given freely and without judgment. But do we as a veterinary profession turn that compassion inward? What does it mean to practice self-compassion and why is it important?

Self-compassion is made up of three components: self-kindness, common humanity and mindfulness. Self-kindness: in times of perceived or realized failure, inadequacy and general suffering, be kind and empathetic with yourself. Common humanity: realize that your struggles are part of a shared experience. Mindfulness: evaluate your life experiences “as is,” without judgment or the suppression of feelings. This three-pronged approach is self-compassion.¹

For our veterinary profession, the issue of self-compassion is an important one. Our profession is “at-risk” for mental and addictive ill health. Veterinarians have a higher suicide rate, and report characteristics consistent with anxiety disorders and depression in higher percentages, versus the general population; and veterinarians present with “at-risk drinking” at significant rates and may show increased use of narcotics. As individuals and as a profession, we cannot allow ourselves to have compassion for our patients and clients but not for ourselves. The consequences are too great.

Authors of a recent article in the Journal of Veterinary Medical Education found that veterinary students with higher non-judgmental, non-reactive mindfulness, and self-compassion had higher resiliency scores.² This finding indicates that self-compassion is an element in strengthening emotional resilience within our profession. Instilling principles of self-compassion and mindfulness into our professional training and professional lives recognizes that mental health, addiction and well-being are issues of importance within our profession.

We should not be a profession that only cares deeply for animals and their owners. We must be a profession that shows self-compassion, that develops and nurtures grounded, resilient, self-aware individuals connected to their own well-being. Tulsa, taught me much about being a veterinarian. Her life and companionship served as an important reminder of the place pets occupy in our lives, teaching me how to care for the humans that love my animal patients deeply. Tulsa’s death serves as a regular reminder to care for myself.

continued on page IV
The AAEP recently formed a Wellness Subcommittee to address issues like self-compassion, mental health and addiction, financial wellness and many others. More information about this initiative can be found at aaep.org/wellness. Issues of wellness will also occupy a significant place at the AAEP Convention in San Antonio; the “Lifestyle/Wellness” sessions will be held Saturday, November 18. Your AAEP is committed to these issues.

Dr. Eggleston owns Eggleston Equine, LLC in Woodstock, Conn., and is a member of the AAEP’s Wellness Subcommittee.

References

Wellness, continued from page III

Three elected to AAEP board of directors

Following a month-long vote by the membership that concluded September 22, Drs. Duane Chappell, Foster Northrop and Deborah Spike-Pierce have been elected to three-year terms on the AAEP board of directors. Each will be installed at the November 20 President’s Luncheon at the 63rd Annual Convention in San Antonio, Texas.

Dr. Duane Chappell, DVM
Dr. Chappell is equine technical service and pharmacovigilance veterinarian with Merck Animal Health.

Since receiving his veterinary degree from Purdue University in 1984, Dr. Chappell has served in many different facets of the industry. After eight years as an associate at mixed animal practices in the Midwest and seven years as a solo practitioner in Indiana, Dr. Chappell became co-owner of Northside Veterinary Clinic in Taylorville, Ill. He later served as resident veterinarian at Richland Ranch Quarter Horse farm and senior clinician/practice manager at Auburn Veterinary Service, both in Auburn, Ill., before spending four years as assistant professor in the veterinary technology program at Morehead State University in Morehead, Ky.

Dr. Chappell serves on the Foundation Advisory Council and chairs its student scholarship program. He previously served on the Dentistry and Infectious Disease committees. He is also chair-elect of the Christian Veterinary Mission and serves on its advisory board.

Dr. Foster Northrop, DVM
Dr. Northrop is founding owner of Northrop Equine PLLC, an ambulatory racehorse and sport horse practice based in Louisville, Ky.

After graduating from the University of Georgia in 1989, Dr. Northrop joined John R. Steele & Associates in Vernon, N.Y., where he provided care to Standardbred racehorses and show horses of all disciplines. In 1992, he relocated to Louisville and spent 16 years practicing on Thoroughbred racehorses and sport horses with Dr. Mark Cheney before starting his own practice in 2008.

Dr. Northrop is a member and former vice chair of the AAEP’s Racing Committee, his contributions to which were recognized when he received the AAEP President’s Award in 2010. He also serves on the Kentucky Horse Racing Commission and on the Thoroughbred Aftercare Alliance board of advisors. He previously served as a member of the Grayson-Jockey Club Research Foundation’s Research Advisory Committee.

Dr. Deborah Spike-Pierce, DVM, MBA
Dr. Spike-Pierce is an ambulatory practitioner and shareholder at Rood and Riddle Equine Hospital who will discontinue clinical practice in 2018 to assume practice management responsibilities for the Lexington, Ky.-based practice.

Upon graduating from Michigan State University in 1993, Dr. Spike-Pierce practiced for a year at a Standardbred racetrack before interning at Rood and Riddle. She spent three years as an assistant to Dr. Larry Bramlage, primarily in equine lameness and imaging, before embarking on her ambulatory practice in Thoroughbred yearlings and sales work in 1998.

Dr. Spike-Pierce is a member of the AAEP’s Leadership Development Committee and previously served as chair of the Student Relations Committee and on the Membership Development Committee and Welfare and Public Policy Advisory Council. She is a former president of both the Kentucky Association of Equine Practitioners and the Kentucky Veterinary Medical Association, and currently serves as president of the KVMA Foundation and as a member of the KVMA Legislative Committee.

EQUINE VETERINARY EDUCATION / AE / NOVEMBER 2017
Hawaii beckons with golden sand, warm sun and science of critical care
Register for AAEP’s Resort Symposium in Maui by January 5

Acquire practical emergency medicine solutions along with memories that will last a lifetime at the AAEP’s 20th Annual Resort Symposium, which will be held January 29-31, 2018, in Hawaii at the Four Seasons Resort Maui at Wailea.

Indulge your mind during half-day educational sessions that will boost your ability to administer critical care when confronted with neonatal, respiratory, colic, ophthalmic and other emergencies. Each day’s sessions will conclude with interactive case discussions with the presenters—Drs. Ben Buchanan, Diana Hassel and Pam Wilkins.

Following sessions, indulge your body by soaking up plenty of vitamin D at the beach, the pool or with colleagues on optional group excursions that include a waterfall hike and swim, a sunset sail and a whale watch sail.

The Resort Symposium offers 15 CE credits. The deadline to register for this unique education-vacation opportunity is January 5. To register or view the complete educational program for the meeting, visit aaep.org/meetings/20th-annual-resort-symposium.

Thanks to Boehringer Ingelheim and IDEXX for their sponsorship of the 20th Annual Resort Symposium.

Touch Point: Electronic examination wellness form available for use with clients

The examination is the cornerstone of the veterinarian-client-patient relationship. Our patient may not talk, but they can “speak” to us through a well-performed exam. When done properly, the examination transforms information into insight and builds trust between the owner or trainer and veterinarian.

If we think of the examination solely as a clinical tool, however, we miss the opportunity to create stronger relationships with our clients. Our clients value how we treat them just as much as they value our medical treatment of their horses.

The AAEP surveyed over 6,000 horse owners and trainers and found that relationship factors are as important to client satisfaction as your ability to provide veterinary care.

Your clients want you to:
• Take your time with their horse during each exam or visit.
• Explain your diagnosis and treatment recommendations in terminology they can understand.
• Demonstrate sincere compassion for the horse.
• Value your client’s opinion.

As a practitioner, you can use the examination to deliver all of these important client satisfaction drivers, plus excellent veterinary care. The principles of a relationship-focused examination can be performed during any type of examination, in any type of equine practice.

New for you is a wellness examination form template that can be modified for your use with your clients. Visit “The Examination” section of touch.aaep.org to download the electronic wellness template and other resources to help you provide a client-focused examination.
AAEP members and others are invited to submit papers for consideration for presentation during the AAEP’s 64th Annual Convention in San Francisco, Calif., Dec. 1–5, 2018. Eligible for consideration are scientific papers, “how-to” papers, review papers, 250-word abstracts and The Business of Practice papers.

**Submitting your paper**
- All papers must be submitted at [http://aaep2018.abstractcentral.com](http://aaep2018.abstractcentral.com) by March 15, 2018, 3:00 p.m. ET. The system will shut down after this time.
- All papers must adhere to the instructions for authors, available at the same website.
- Be sure to familiarize yourself with the submission process well in advance of the deadline. You can set up your profile with paper and author information in advance and then upload your paper when it is complete.
- Since the review process is blinded, make sure your paper does not include author or institution names.

**A few key points**
- Products and equipment must be identified by chemical or generic names or descriptions and footnoted.
- Due to the length and complexity of the process, all deadlines are strictly enforced.
- Submission of a paper represents a commitment to present this paper at the meeting if it is selected.
- Selected papers will be printed in the 2018 AAEP Proceedings and presented at the 2018 Annual Convention. The presenting author will receive complimentary registration and a travel allowance.

**Ethical Considerations**
- 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 submitted paper or with companies that manufacture or sell competing products.
- If your presentation references the use of a compounded pharmaceutical, ensure that you are familiar with the FDA guidelines on the use of compounded pharmaceuticals and that the product you reference is in compliance.
- All AAEP abstracts submitted for presentation should cite levels of evidence-based medicine.

**Types of papers accepted**
*All paper presentations are limited to 15 minutes plus 5 minutes for Q&A.*

**Scientific papers** should be a minimum of 600 words. Special attention will be given by the Educational Programs Committee to material with practical content or new information.

**How-to papers** should describe and explain a technique or procedure used in veterinary medicine or the equine industry. The technique should be relatively new or not widely understood or used in practice. There is no word limit for how-to papers.

**Review papers** should update the membership on a new subject or gather information that may be conflicting. Although a review paper does not necessarily contain original data, it is anticipated that the presenter will have considerable experience in the field.

**Abstracts ≤ 250 words** may be submitted by authors who intend to publish in a refereed journal. An abstract conforming to the AAEP instructions for authors must also be submitted (for review purposes only) to allow the reviewers to assess the experimental design, materials and methods, statistical analyses, and results (with graphs, tables, charts, etc.) and to discuss the results as they pertain to interpretation and conclusions.

**The Business of Practice papers** may cover any business management topic that can help the practitioner and their practice achieve more success and improve profitability. The theme for 2018 is “Practice Culture for Profitability.”

**Need help submitting a paper?**
As an aid to private practitioners, first-time authors or members seeking guidance with their submission, AAEP offers a mentorship program in which experienced presenters are available to provide advice and direction. However, mentors are not responsible for rewriting or selecting material.

Contact Carey Ross, scientific publications coordinator, at cross@aaep.org for a list of available mentors or with questions concerning the annual convention and educational paper submission.
National Equine Health Plan published
Valuable resource will help curtail risk of disease spread

By Dr. Nat White and Bailey McCallum

Highlights:

Goals of the plan support the health and economic viability of the U.S. equine industry.

Plan coordinates industry stakeholder preparedness to decrease risk of disease spread.

Veterinarians encouraged to share the document with their horse-owning clients.

The National Equine Health Plan (NEHP), created through a partnership with the USDA, state animal health officials, the AAEP and numerous American Horse Council member organizations, is now available at equinediseasecc.org/national-equine-health-plan.

The goals of the plan are to protect the health and welfare of the U.S. equine population, facilitate the continued interstate and international movement of horses and their products, ensure the availability of regulatory services and protect the economic continuity of business in the equine industry. Ultimately, the following goals support the larger mission of enhancing the health and economic viability of the U.S. equine industry.

1. Protect the health and welfare of horses in the U.S. and North America;
2. Facilitate the continued interstate and international movement of equine and associated products with science-based requirements and standards;
3. Protect the business continuity and economic viability of the equine industry;
4. Establish the role of the industry, federal and state authorities, and tribal governments in equine disease prevention and control and in natural disasters;
5. Ensure the availability of current diagnostic testing, inspection and certification services;
6. Create and maintain a communication system that provides timely and rapid dispersal of accurate information about disease outbreaks;
7. Provide guidelines for control, identification and containment of equine diseases; and
8. Support and make available educational programs for horse owners and industry representatives on disease identification and prevention.

The NEHP functions as a roadmap for coordinating horse owners and industry organizations with veterinarians and state and federal animal health officials to prevent, recognize, control and respond to diseases and environmental disasters. The plan facilitates horse industry preparedness, effective rapid communication and owner education, which make up the foundation for preventing diseases and disease spread. Links to information and resources are included in the NEHP document, including a list of “Roles and Responsibilities” for all industry stakeholders.

The Equine Disease Communication Center is a key element of the NEHP and provides critical communication of information during disease outbreaks. Additionally, the EDCC website, equinediseasecc.org, provides information about diseases, vaccination, biosecurity, state health regulations, state animal health official contact information and links to USDA-APHIS veterinary services.

Veterinary practitioners are the first to recognize and contain infectious diseases and play a key role in the NEHP. By integrating the roles of regulatory agencies with industry stakeholders, equine health and welfare are improved. This coordination of all stakeholders helps decrease the impact of infectious diseases on the horse economy. The NEHP provides immediate access to resources and communications needed to optimize disease mitigation and prevention.

The NEHP is a valuable information resource for AAEP members and their clients. It serves as a guide for regulations and responses needed to mitigate and prevent infectious diseases. Sharing this document will help educate horse owners about how veterinarians and state and federal officials work together to decrease the risk of disease spread. The equine industry as a whole can benefit from the awareness created by the NEHP.

Dr. Nat White and Bailey McCallum are director and communication manager, respectively, of the Equine Disease Communication Center.
Five receive Merck Animal Health/AAEP Foundation scholarships

Five veterinary students committed to careers in equine medicine have been selected to receive $5,000 scholarships through the Merck Animal Health/AAEP Foundation scholarship program.

Congratulations to the following recipients, who will receive their awards November 19 during the AAEP’s 63rd Annual Convention in San Antonio, Texas. Pictured from left to right:

Brittany Bazeley, University of California, Davis
Travis Miller, Tuskegee University
Rachel Pfeifle, Auburn University
Alexandra Scharf, University of Georgia
Kelsey Stoner, Washington State University

The Merck Animal Health/AAEP Foundation scholarship program rewards second- and third-year veterinary students for their academic excellence, leadership in their school and AAEP student chapter, and long-term goals in equine medicine.

Education, including scholarship assistance, joins research and horse benevolence as the three pillars of AAEP Foundation support of its mission to improve the welfare of the horse. Scholarship support remains as important as ever given the significant financial investment of a veterinary education.

For more information about AAEP Foundation scholarship programs or to give in support of AAEP Foundation’s educational or other equine welfare initiatives, visit aaepfoundation.org.

Mark it down! 2018 AAEP calendars make great gifts

Show your appreciation to clients and simultaneously support the welfare of the horse this holiday season with a gift of the 2018 AAEP Horse Sense Calendar.

The calendar features stunning equine photography each month along with anatomical diagrams and a chart on the back cover to help your clients keep track of important health dates. Calendars also are customized to include your practice’s contact information, which will help maintain top-of-mind-awareness with clients year-round.

In addition to the executive-style calendar, a small, adhesive “stick up” calendar imprinted with your practice’s contact information is available in quantities of 150 or more. This 3” by 2 ¼” calendar can be placed on refrigerators, filing cabinets or in vehicles. Each order comes with a complimentary display box.

A generous portion of every calendar order goes to the AAEP Foundation and is invested in projects and programs that benefit the welfare of the horse.

Order your copies at http://tinyurl.com/aaepfcal by November 1 to ensure holiday delivery. Or, if you are planning to attend the AAEP’s 63rd Annual Convention in San Antonio, you can place your order at the AAEP Foundation booth in the trade show. Calendars purchased at the convention will be shipped within two weeks to arrive before the holidays.
Surrounded by the breathtaking beauty of Maui, acquire the diagnostic and treatment techniques to respond to, manage and resolve respiratory, colic, neonatal, trauma and other emergencies from board-certified experts in private practice and academia:

Ben Buchanan, DVM, DACVIM, DACVECC
Diana Hassel, DVM, DACVS, DACVECC
Pam Wilkins, DVM, DACVIM, DACVECC

Register by Jan. 5 at aaep.org/meetings
Nutramax Laboratories Veterinary Sciences, Inc., trusted by veterinarians and consumers for over 20 years, has demonstrated our dedication to you, your clients, and your patients through our educational partnership with the American Association of Equine Practitioners. Cosequin®, our brand of joint health supplements, is the No. 1 veterinarian-recommended brand.*

At Nutramax Laboratories, Inc. we believe that products should be supported by research to ensure the best results for you and your patients. Our commitment is shown in our investigations into mechanisms of action for our product ingredients and our support of studies evaluating efficacy, bioavailability and safety at leading veterinary schools. We stand by the quality of our products and manufacture them following standards similar to those practiced by the U.S. pharmaceutical industry. Every lot of our products produced is tested by our quality control department to ensure your patients receive a safe, high-quality product containing the amounts of ingredients claimed on the label.

Developing products to improve the quality of life for your patients is our goal. Cosequin® and Dasuquin® joint health supplements are the No. 1 veterinarian-recommended brands for equine and small animal patients respectively. Proviable®-EQ is a probiotic digestive aid containing Saccharomyces yeast species to support normal gastrointestinal health and function. Welactin® Equine is an omega-3 fatty acid fish oil supplement going beyond skin and coat to support overall health and wellness.

To learn more about our products, contact Nutramax Laboratories at (888) 886-6442 or visit nutramaxlabs.com.

* Source: Survey conducted in February 2016 of equine veterinarians who recommended oral joint health supplements.

Source: Among veterinary brands. Survey conducted in February 2016 of small animal veterinarians who recommended oral joint health supplements.

**The AAEP welcomes new members and congratulates recent graduates**

**New Members:**
- Fernando Amitranos, MVZ, Madison, WI
- Victoria A. Bentley, DVM, Mexico, NY
- Mary Bumgarner, DVM, Weatherford, TX
- Marinella Ruegger de Freitas, DVM, Elgin, TX
- Susan C. Eades, DVM, DACVIM, College Station, TX
- Pablo Espinosa Mur, DVM, Davis, CA
- Andrew R.E. Jones, BVetMed, MS, Bonsall, CA
- Rick Katchuk, DVM, Red Deer, AB, Canada
- Candace Kendrick, DVM, Colorado City, TX
- Sarah J. Lopez, DVM, Los Lunas, NM
- Katherine Jane McDonald, DVM, Rocky View County, AB, Canada
- Danielle Miller-Boster, DVM, MS, Atkinson, IL
- Danielle Christine Opfenring Amavisca, DVM, Yuma, AZ
- Tomohiro Otsuka, DVM, Urakaka-Gun, Japan
- Paul M. Schultz, DVM, Fulton, MO
- Julie Storme, DVM, Fort Collins, CO
- Warren C. Wynn, DVM, Weatherford, TX

**Recent Graduates:**
- Casey Colwell, DVM, Nolensville, TN
- Melissa Curry, DVM, Martin, OH
- Samantha Dzierzak, DVM, Phoenix, AZ
- Katherine Ellis, DVM, Chino Hills, CA
- Bianca Fedele, DVM, BS, Versailles, KY
- Michael Forrester, DVM, Bryan, TX
- Lyndsey Hayden, DVM, Wallingford, CT
- Caitlin Hutcheson, DVM, Woodbine, MD
- Jacqueline Jewell, DVM, Vashon, WA
- Alaina Kringen, DVM, Orient, SD
- Abigail McElroy, DVM, Lansing, MI
- Katherine Mundy, DVM, Ottawa, ON, Canada
- Thomas Dalton Pate, DVM, West Point, MS
- Kaela Paternoster, DVM, South Glastonbury, CT
- Ben Reed, DVM, Exshaw, AB, Canada
- Cara Rosenbaum, DVM, Newtown, CT
- Katherine Roybal, DVM, Mesilla Park, NM
- Joy Sizemore, DVM, Washington Court House, OH
- Allison Sproat, DVM, Aldergrove, Sk, Canada
- Heather Trainor, DVM, Amesbury, MA
- Amelia Tyler, DVM, Browns Summit, NC
- Drew Upright, DVM, Winter Park, FL
- Lindsay Wolcott, DVM, Califon, NJ
Dr. James K. Boutcher, the primary veterinarian for 1977 Triple Crown winner Seattle Slew, passed away September 22 at his home in Versailles, Ky. He was 76.

Dr. Boutcher received his veterinary degree from Auburn University in 1970 and devoted 40 years of his life caring for horses throughout Kentucky and as far away as Japan. According to bloodhorse.com, Dr. Boutcher was the veterinarian who recommended to Harold Snowden Jr. that he geld an ill-tempered Thoroughbred colt named John Henry, who would go on to racing stardom before retiring in 1985 as the world’s leading money earner at $6,591,860.

An AAEP member since 1974, Dr. Boutcher was a former president of the Kentucky Association of Equine Practitioners and a longtime member of the AVMA, Kentucky Veterinary Medical Association, Florida Veterinary Medical Association and several Thoroughbred organizations.
AAEP Meetings and Continuing Education

November 17-21, 2017
63rd Annual Convention
Henry B. Gonzalez Convention Center
San Antonio, Texas

January 29-31, 2018
20th Annual Resort Symposium
Four Seasons Resort Maui at Wailea
Maui, Hawaii

For more information, contact the AAEP office at (859) 233-0147 or (800) 443-0177 or online at aaep.org.

Membership Benefits

Embrace volunteer opportunities to serve the profession and industry

“One of the most important AAEP member benefits is my opportunity to volunteer as one of the voices and leaders for the health and welfare of the horse. Many of us can serve as a speaker at special events, as a mentor to an equine veterinary student or assist with one of the AAEP’s many owner education programs. Moreover, I am proud to be an AAEP member who can enhance and influence the professional development of my veterinary colleagues and aspiring veterinary students. Thank you AAEP!” —Rachel Cezar, DVM, Waldorf, Md.

Do you want to contribute to your profession in meaningful ways beyond your daily practice? If so, the AAEP wants to hear from you.

A broad and diverse network of member volunteers is critical to fulfilling the AAEP’s mission to improve the health and welfare of the horse, further the professional development of its members and provide resources and leadership for the benefit of the equine industry.

Regardless of whether your volunteer interests lie in traditional board, committee and task force assignments or in more targeted opportunities such as new member ambassador or Ask the Vet expert, a completed volunteer interest form is necessary for consideration. When volunteer positions become available, the Leadership Development Committee queries the database of potential volunteers to identify candidates who match the desired training, experience and attributes.

To complete the form and become eligible for volunteer service, just log in to aaep.org and select “View My Member Profile” to complete the Volunteer Interest Form.

Buy or sell pre-owned practice equipment

“I have converted thousands of dollars of underused equipment into cash through the AAEP’s Equipment Marketplace.” —Susan Moreland, DVM, Farmington, N.M.

Looking for used veterinary equipment to save on the cost of purchasing new? Or perhaps you are upgrading and looking to sell your old equipment. No matter what side of the commerce equation you’re on, you can buy and sell pre-owned veterinary equipment in the AAEP’s online Equipment Marketplace.

Creating a listing is easy, and search capabilities allow you to narrow results to specific categories of equipment. From ultrasound machines and radiograph units to vet boxes and dental tools, you’ll find it all at aaep.org/dashboard/equipmentmarketplace.
You can rely on us for all your equine veterinary practice needs.

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Highlights of recent clinically relevant papers

**Headshaking prevalence**
In this study Sarah Ross and colleagues at the University of Bristol investigated the prevalence of headshaking within the equine population in the UK. A questionnaire on headshaking was distributed amongst horse owners. This also included ‘distractor’ questions on sarcoids and laminitis to reduce response bias. A total of 1014 questionnaires were completed. Forty-seven horses had shown headshaking signs in the previous 12 months, giving a prevalence of 4.6%. A further 1.6% had previously shown signs but not in the last year, giving a combined prevalence of 6.2%. Two horses had died/been euthanised due to headshaking. The majority of headshakers were less than 20 years old, with median age 12 years. There was no significant association between headshaking and either breed or sex.

May, April and March were the most commonly reported months for onset of headshaking signs. Overall, over 50% of horses first showed signs in these months. The majority of horses showed signs in summer (89.3%) and spring (76.8%) and 19.6% had signs in all four seasons. Sunshine and heat were associated with signs in 79.4 and 52.4% of cases, respectively. The most commonly reported clinical sign was vertical head movement (78.5%) followed by rubbing of the muzzle (38.5%). Of horses that were turned out, 26.4% showed signs at rest. Of those that were lunged or ridden, 53.2% showed signs during lunging and 95.1% during ridden exercise. On a grading system of 0 (no signs) to 3 (headshaking at rest), 50.8% were graded as 1/3, 30.2% were graded 2/3 and 19% at 3/3. The highest number of horses were graded 0/3 in autumn and winter.

Approximately one-third of headshaking horses were examined by a veterinarian. Of these, the most commonly performed diagnostic procedures were dental examination (83.3%) followed by aural and ocular examinations (50% each). Endoscopy was performed in 38.9% of cases and head radiographs in 22.2% of cases. The cause for headshaking was unknown in over half of cases (57.9%), allergy in 36.8% of cases and trigeminal-mediated headshaking was diagnosed in only one case. The reasons for this low level of veterinary intervention were not explored in this study and the authors postulate that owners may not recognise these clinical signs as manifestations of neuropathic pain. Alternatively, the high proportion of cases in which a comprehensive and costly investigation fails to identify a cause may deter owners from seeking veterinary assistance. Increased awareness of trigeminal-mediated headshaking (previously classed as idiopathic headshaking) may be required. In over 75% of cases, at least one treatment had been attempted, the most common being nose nets and eye masks. Nose nets resulted in resolution of signs in 19.5% of cases, but had no effect in 73.2% of cases.

**Tulathromycin treatment for bronchopneumonia**
This study by D. Rutenberg and colleagues in Germany and the USA investigated the efficacy of tulathromycin as a treatment of foals with mild to moderate bronchopneumonia. There is conflicting data on the efficacy of tulathromycin for the treatment of foals with bronchopneumonia. In this study the authors tested their hypothesis that tulathromycin is effective for the treatment of bronchopneumonia in foals and noninferior to the combination of azithromycin and rifampin.

A total of 240 foals on a farm endemic for infections caused by Rhodococcus equi were included. In a controlled, randomised, double-blinded clinical trial, foals with ultrasonographic pulmonary lesions (abscess score 10–15 cm) were allocated to three groups of 80 horses: 1) tulathromycin i.m. q. 7 days; 2) azithromycin-rifampin, orally q. 24 h; or 3) untreated controls. Physical examination and thoracic ultrasonography were performed by individuals blinded to treatment group. Foals that worsened were considered treatment failures and removed from the study.

The proportion of foals that recovered was significantly higher for foals treated with tulathromycin (70/79) or azithromycin-rifampin (76/80) compared to that of control foals (22/80). The difference in the percentage of efficacy of azithromycin-rifampin vs. tulathromycin was 6.4% (90% CI: -0.72 to 13.5%). Given that the confidence interval crossed the predetermined noninferiority limit of 10%, the null hypothesis that the response rate in the azithromycin-rifampin group is superior to that of the tulathromycin group could not be rejected. Resolution of ultrasonographic lesions occurred faster in foals treated with azithromycin-rifampin than in foals treated with tulathromycin.

Tulathromycin was effective for the treatment of bronchopneumonia in foals in this study but not as effective as the combination of azithromycin-rifampin.

**Parasite resistance**
In this study Cheryl Shea Porr and colleagues in the USA evaluated the efficacy of ivermectin (IVE) and moxidectin (MOX) on faecal egg counts (FECs) and egg reappearance period (ERP) in horses.

Faecal samples (n = 46) were collected and evaluated for parasite eggs using the Modified McMaster Fecal Egg Count technique. Eggs per gram (EPG) of faeces were recorded. Horses were randomly allocated based on prestudy FEC (low, <200 EPG; moderate, 200–500 EPG; high, >500 EPG), age (young, <15 years; old, >16 years), and housing (stall or pasture). Treatments included control (CON, no treatment, n = 10), IVE (n = 10), or MOX (n = 10). Faecal samples were collected and evaluated every 2 weeks for 12 weeks after treatment. Faecal egg count reduction tests were 100% for IVE and MOX, indicating that both anthelmintics were effective. However, parasite eggs began to appear in IVE horses in Week 6 and MOX horses in Week 8. Currently reported ERP for IVE and MOX are 6–8 weeks and 10–12 weeks, respectively, suggesting decreased efficacy of MOX. In pastured horses, MOX was more effective in reducing FEC than IVE (1.84 vs. 6.43 EPG, respectively). These data suggest that anthelmintic use improved internal parasite control and that MOX may have greater efficacy than IVE; however, the shorter ERP for MOX may indicate that MOX efficacy may be decreasing.

**Owner recognition of laminitis**
This study by Danica Pollard and colleagues in the UK aimed to ascertain whether owner-reported laminitis cases were confirmed as laminitis by the attending vet; and compare owner and veterinary reported information in these cases.
Data were obtained from vets and owners of 93 cases of laminitis seen by 25 veterinary practices in 2014 and 2015. Respondents recorded clinical signs, risk factors and underlying disease, with owners completing their responses independently from their vets. Welsh ponies and crosses were the most frequently represented breeds: 62% of cases were overweight. Of the 93 veterinary diagnosed cases, 51 (54.8%) were suspected to have laminitis by their owners. All 51 cases were confirmed to be laminitis by the attending vet. However, 45.2% of owners who did not suspect laminitis prior to vet diagnosis either did not know what the problem was or suspected a different condition including foot abscesses, bruised soles, navicular disease and colic.

Divergent growth rings were significantly more commonly reported by veterinary surgeons in cases where owners recognised laminitis, compared to cases unrecognised by owners. This suggests that owners of these horses either had previous experience of laminitis in their animal or used the presence of these rings as an indicator of laminitis. Difficulty in turning and weight shifting were more commonly reported by vets whereas increased hoof temperature and recumbency were more commonly reported by owners. Difficulty in turning was reported in more than 75% of cases, while the classic laminitic stance and divergent growth rings were much less common, highlighting a need to educate clients to recognise more subtle clinical signs.

Fewer owners reported equine metabolic syndrome compared to vets, but this difference was not apparent for PPID. The horse’s breed was more commonly reported as a risk factor in owner-recognised cases than in those not recognised by owners. Pony breeds were significantly more commonly reported in owner-recognised cases perhaps meaning that owners are not aware of the potential of laminitis in non-pony breeds. Fewer owners reported obesity compared to the vets, indicating that owners’ perceptions of their animal’s body condition score are not reliable. Further owner education is needed, particularly in areas such as the spectrum of possible clinical signs, equine metabolic syndrome and assessment of body condition.

### Large colon sand accumulations

In this study Isabelle Kilcoyne and colleagues in the USA determined the influence of radiographic quantification of sand accumulation on the medical vs. surgical management of large colon sand accumulations. Short- and long-term outcomes and complications were also compared.

Medical records and abdominal radiographs of 153 horses presented for colic over an 11-year period were reviewed. Breed, weight, amount of sand, presence of diarrhoea at presentation, treatment, and the development of complications were recorded.

The mean cross-sectional area of sand accumulation was 692.9 cm² (median 658.7 cm², 84.6–1780.7 cm²). Increased accumulation of gas on radiographs and abnormal transrectal examination findings were associated with an increased likelihood of surgery. The most common complication was the development of diarrhoea (20.3%) with only four (2.6%) horses positive for Salmonella spp. Horses had a favourable prognosis, with 94.8% of horses treated medically and 94.7% of those treated surgically surviving to discharge.

The authors concluded that increased accumulation of gas on radiographs and transrectal palpation of impaction or intestinal gas distension increase the likelihood of surgery. Both medical and surgical treatments carry a good prognosis. The sheer quantity of sand is not a factor when determining surgical intervention.

### Salmonellosis after colic surgery

In this retrospective case–control study Louise Southwood and colleagues in the USA compared long-term outcome of Salmonella-positive vs. Salmonella-negative horses discharged from hospital after colic surgery.

For each horse with positive culture for *Salmonella enterica* (SAL-POS, n = 59), at least two horses testing negative for *S. enterica* (SAL-NEG, n = 119) were enrolled. Owners were interviewed via phone at least 12 months after surgery regarding: 1) complications after discharge from the hospital; 2) duration of survival; and 3) return to prior or intended use. Association between immediate post-operative clinical variables such as Salmonella status and long-term measures of outcome was tested via ratios (odds ratio [OR]) and 95% confidence intervals. Data were analysed for survival using a Cox proportional hazards model and for return to use using multivariable logistic regression.

SAL-POS horses had a higher OR of surgical site infection (2.7 [1.1–6.9]) and weight loss (6.8 [1.8–26.1]). At the time of follow-up, there were 53/56 (95%) SAL-POS and 99/118 (84%) SAL-NEG horses alive. The final multivariable model for nonsurvival included post-operative colic (hazard ratio 7.6 [2.8–19.2]) and the interaction between Salmonella status and duration of rectal temperature >103°F post-operatively (SAL-POS 1.04 [1.01–1.07] and SAL-NEG 1.16 [1.06–1.25]). The majority of horses returned to their intended use regardless of their SAL-POS (38/50, 76%) or SAL-NEG (77/96, 80%) status.

Salmonella-positive horses that survive to discharge from the hospital after colic surgery have similar risks of long-term complications (colic/diarrhoea), survival, and return to function to Salmonella-negative horses.

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**References**


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Iatrogenic fracture of the premaxilla during standing exodontia

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Summary

This case report describes a 17-year-old horse that presented with a history of oral dysphagia secondary to buccal slab fractures and apical infection in two cheek teeth. During standing surgery to remove the diseased cheek teeth, the horse sustained a bilateral comminuted fracture of the premaxilla. An audible crack was followed by bilateral epistaxis. Subsequent radiography revealed minimal displacement at the fracture sites and the horse responded well to conservative management without the need for additional fixation techniques. One year later, the fracture appeared completely healed on repeat radiography. This is an uncommon complication of standing dental procedures necessitating the use of a speculum but is noteworthy and may be important to consider and inform owners in such aged cases.

Introduction

Idiopathic cheek teeth (CT) fractures are a relatively common finding in the equine oral cavity (Taylor and Dixon 2007). The most common fracture pattern is the lateral slab fracture of the maxillary CT, with the ‘09s being particularly over-represented (Dacre et al. 2007; Taylor and Dixon 2007; Dixon et al. 2008). In spite of pulp chamber exposure, in some cases, these fractures are discovered incidentally without evidence of clinical signs. In some instances the fracture fragments may be shed spontaneously and the sensitive pulp tissues become sealed from the fracture site with reparative (tertiary) dentine. This process helps to prevent deeper pulp and apical infection from occurring and allows the remaining tooth to erupt normally (Magloire et al. 1996; Dixon et al. 2000, 2008; Casey 2013). Clinical signs may be evident when fracture fragments attached to the gingiva cause stretching of the periodontal ligament and/or physically impinge on the adjacent buccal mucosa resulting in lacerations and ulceration. These instances both result in oral pain, which often manifests as dysphagia and/or quidding. However, a proportion of fractured CT become apically infected. Under such circumstances conservative treatment with antimicrobials may be effective in the early stages of disease (Dacre and Dixon 2004) but in others, and where apical infection is more severe and becomes chronic, antimicrobial therapy alone is often unsuccessful (Dixon et al. 2000; Tremaine 2004) and such teeth require extraction.

Exodontia may be performed under standing sedation or general anaesthesia (GA). Extraction of diseased cheek teeth per os in the sedated standing horse is an increasingly popular practice because it is associated with good success rates, reduced cost and fewer complications than tooth repulsion under GA (Dixon et al. 2005, 2008; Townsend et al. 2011). It is best performed while restrained in stocks under heavy sedation with regional local anaesthesia. With the proliferation in the variety of techniques and instrumentation available [e.g. minimally invasive transbuccal extraction (Stoll 2011, 2013; Langeneckert et al. 2015) and repulsion techniques (Coomer et al. 2011)] the vast majority of such teeth can be successfully removed without resorting to traditional repulsion of CT under GA, which carries complication rates of 32–70% (Pritchard et al. 1992; Tremaine and Dixon 2001; Earley 2012). It is usually a last resort in our practice and reserved for cases which are refractory to standing techniques.

Potential complications of exodontia include fracture or damage to adjacent teeth, alveolar bone fracture and sequestration, and iatrogenic mandibular fractures among others. Complications more specific to maxillary CT extractions include palatine artery laceration, sinusitis and oroantral fistulisation (Dixon et al. 2005; Earley 2012). Iatrogenic fracture of the rostral maxillae has only been reported once previously during the per os extraction of a maxillary CT; in this instance, with an almost identical fracture pattern, there was no epistaxis and the fracture was stabilised using an intraoral wire fixation device (Widmer et al. 2010).

This report describes a horse which sustained a premaxillary fracture during a procedure to remove two cheek teeth per os and the fracture was managed conservatively. This is a rarely reported complication of exodontia in the veterinary literature. The potential cause is speculative but may inform case selection for this procedure in older horses.

Case history

A 17-year-old Warmblood gelding was referred for investigation of oral dysphagia of several weeks duration. An equine dental technician had identified a cheek tooth fracture during a routine dental examination. The horse was able to eat grass, was slower than normal eating hard feed and struggled to eat hay without quidding. The owner reported that the horse was otherwise healthy.

Clinical findings and diagnosis

The horse was in good bodily condition and a general clinical examination was unremarkable. No abnormal head swellings, facial asymmetry or lymph node enlargement was evident. Oral examination with a full mouth speculum (stainless steel, 4 ratchet Haussman’s speculum)†, performed under standing sedation, revealed buccal slab fractures in cheek teeth 109 and 209. The fracture fragments were still attached to the gingiva, which were impinging on the buccal mucosa...
causing lacerations and ulceration and were considered to be the most likely cause of the dysphagia. Closer inspection using an oral endoscope indicated that both 109 and 209 were grossly abnormal and enlarged in both the buccopalatal and mesio-distal planes. A narrow rim of enamel bordered a single, deep food-stained caries to which all the pulps were exposed.

There was no evidence of sinusitis on lateral radiographs and oblique radiographs (lateral 30° dorsolatero-ventral projection) of both maxillary arcades revealed the 109 and 209 cheek teeth to have a generalised lucency and rostrocaudal enlargement, with a morphological change to the apices making the roots appear ‘clubbed’. A radiopacity consistent with sclerosis of the alveolar bone and a periapical radiolucency were also apparent (consistent with sclerosis of the alveolar bone and a periapical radiolucency were also apparent (Townsend et al. 2011). These findings are strongly associated with chronic apical infection (Townsend et al. 2011).

**Treatment**

After careful discussion with the owner a plan was made to remove the 109 and 209 teeth. The horse was placed in stocks with the head suspended by an overhead gantry and head cradle. An intravenous (i.v.) catheter (over the needle polyurethane) was placed and the horse sedated with a detomidine (Domosedan) bolus (6 µg/kg bwt). Multimodal analgesia (Cantwell and Robertson 2006) was provided in the form of morphine (0.3 mg/kg bwt i.m.) and i.v. flunixin meglumine (Cronyxin) (2.2 mg/kg bwt). A right maxillary nerve block (for extraction of the 109 in the first instance), using 10 mL 2% w/v mepivicaine (Intraepicaine) was administered with a 21 gauge, 50 mm needle placed aseptically, perpendicular to the skin just ventral to the zygomatic process and dorsal to the transverse facial vessels at the level of the caudal third of the orbit. The needle was inserted until penetration of the extraperiorbital fat body was appreciated and the local anaesthetic administered (Staszyk et al. 2008). Preoperative oxytetracycline (Engemycin) (5 mg/kg bwt) was administered i.v. Diazepam boluses were administered i.v. as required to reduce tongue tone and movement during the procedure (Coomer et al. 2011).

Sedation was maintained with a continuous rate infusion of detomidine (with a starting dose of 0.1 µg/kg bwt/min and modified as needed throughout the procedure). The horse was completely immobile and unresponsive to stimuli before the procedure commenced. The loose buccal fracture fragment was quickly and easily removed following elevation from its gingival attachment using a short, right-angled periodontal elevator, under oroscopic guidance. Owing to the abnormal interproximal spaces between 109 and adjacent cheek teeth, molar spreaders could not be easily employed. Using a variety of flat-bladed periodontal elevators (Ramzan) with different angles, depths and degree of curvature, the gingival attachments were broken down and the periodontal ligament disrupted. The application of conventional extraction forceps was not appropriate in this case. Elevation of the tooth under oroscopic guidance was as previously described (Ramzan et al. 2010); sometimes using two periodontal elevators simultaneously, placed on the buccal and palatal aspects of the tooth and with the repeated application of long-nosed extraction forceps to grasp the remaining portion of the erupted crown, oscillation resulted in steadily increasing movements of the tooth within the alveolus. This served to loosen the tooth sufficiently and the 109 was removed intact within 1 h.

The Hausman’s speculum remained open throughout the entire procedure but was closed for 15–20 min while the contralateral maxillary nerve block was placed in preparation for the extraction of the 209. The same approach to exodontia was applied to this tooth. Approximately 30 min into elevation and loosening of the 209 a loud cracking noise was heard. This was followed by significant bilateral epistaxis. The horse remained completely immobile during this sequence of events. The procedure was halted immediately and the speculum removed. Crepitus was palpable in the premaxillary region. Repeat radiographs (both lateral and oblique projections) revealed a bilateral comminuted fracture of the premaxilla with minimal displacement (Fig 2). The epistaxis abated spontaneously after 10 min; it seems likely that this was the result of disruption of the nasal septum.

Given that there was no evidence of premaxillary displacement and the horse appeared comfortable once the sedation had worn off and was eager and able to eat soaked feed, he was managed conservatively in the short term. After several days in hospital, he was discharged on a course of anti-inflammatory analgesic (phenylbutazone [Equipalazone], 2 mg/kg bwt per os twice a day) and antimicrobials (TMPS [Trimedazine], 4.2 mg/kg bwt per os twice a day) with instructions to remain on box rest and to be fed a complete soft diet (Fast Fibre) until re-examination.

**Clinical outcome**

The horse was re-examined 6 weeks later when he was reportedly eating well and had graduated from a complete soaked diet to one comprising part soaked and part forage. There was no sign of oral dysphagia during this transition and he had gained weight. Clinical examination was largely unremarkable with no evidence of maxillary swelling or asymmetry. No crepitus was evident and no pain response was elicited on palpation around the fracture site. Oral examination with a speculum was not performed at this time.

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**Fig 1**: Right latero-30°-dorsolatero-ventral radiograph of the maxillary arcade showing the presence of the diseased 109 cheek tooth.
stage. Radiography (lateral-lateral projection) of the interdental space revealed remodelling at the fracture sites consistent with fracture healing. The gelding was re-examined 6 weeks later at which point he was eating a normal range of feedstuffs without evidence of oral dysphagia. Further radiographs revealed continued healing at the fracture sites (Fig 3). Given his progress, reintroduction to light ridden work was suggested.

One year later the horse was eating normally and had returned to full ridden (bitted) work. No further routine dental work had been undertaken since the extraction of the 109 and the partially elevated 209 remained in situ. No other clinical signs consistent with apical infection (such as sinusitis) were evident. Left-sided facial swelling was noted only some of the time. The owner of the horse was understandably hesitant about further extraction owing to the risk of the premaxilla refracturing, particularly in the absence of clinical signs indicating that it might be causing a problem. Repeat radiography of the premaxilla demonstrated that the fracture had healed completely (Fig 4). Oblique radiographs of the left maxillary tooth roots showed periapical sclerosis, but no periapical radiolucency suggestive of an active apical infection associated with 209. Oral examination with a full mouth speculum was performed under heavy standing sedation. Large overgrowths (6–7 mm) were present on 409, opposing the gap left by exodontia of 109 and on 309, opposing the abnormal concave crown of 209. Sharp enamel points were evident on the buccal and lingual aspects of the maxillary and mandibular cheek teeth respectively. Extensive food packing between the overgrowth on 309 and the concave occlusal surface of 209 extended buccally displacing the cheek and it was this that accounted for the apparent transient facial swelling. The gingiva at the 109 exodontia site appeared healthy and there was no evidence of periodontal disease anywhere within the oral cavity. The sharp enamel points and overgrowths were subject to reduction using a combination of hand rasps and a motorised float. Reduction of the overgrowths was staged to reduce the risk of pulpar exposure (Bettiol and Dixon 2011).

Discussion
Cheek tooth extraction per os has become widespread within veterinary practice in recent years because it is associated with good success rates and minimal complications (Dixon et al. 2005, 2008; Townsend et al. 2011). Complications associated with standing exodontia are well described (Earley 2012) and iatrogenic fracture of the maxilla is a rare complication of equine dental surgery and has only been previously reported once previously during exodontia under standing sedation (Widmer et al. 2010). In the exodontia case previously reported and the one featured in

![Fig 2: Lateromedial radiograph showing the complete bilateral transverse fracture in the interdental space of the maxilla with minimal displacement. The rectangular radiodense opacity evident in the interdental space is a fragment of tooth displaced during the extraction of 209.](image1)

![Fig 3: Lateromedial (slightly obliqued rostrocaudally) radiographic projection taken 12 weeks post-operatively showing remodelling of the premaxillary fracture.](image2)

![Fig 4: Lateromedial radiographic projection taken 12 months post-operatively showing complete healing of the premaxillary fracture.](image3)
this report, the fracture was in the interdental region, well away from the teeth being extracted and was bilateral in nature. Factors common to these cases were the use of a full mouth speculum and advanced age.

Most of the standing exodontia procedures (MIT, MITSE, standing repulsion as well as per os extractions) employ the same full mouth speculae which are also used to hold the jaw open for routine dental examinations, although they are usually in place for significantly longer periods of time when performing dental surgery. However, speculae are rarely implicated as a potential primary cause of complications in such procedures. There are no published reports, to our knowledge, of direct injury to the bony structures of the skull from the application of full mouth speculae during routine dental correction; one case, described by Widmer et al. (2010), presented with epistaxis and a premolar fracture which was attributed to the delayed effect of using of a full mouth speculum for dental floating a few days previously.

A variety of full mouth speculae are available. The opening mechanism varies between models, but the ratchet type (used in the Hausmann model employed in this case) is the most widely used design. Inevitably some degree of force is required to ratchet open the speculum against considerable opposition from the masticatory muscles. Where the mechanism facilitates ease of opening it has been observed that speculae may be opened too wide causing harm to the TMJ, facial nerve and masticatory muscle (Simhofer and Schramel 2008). When a horse is heavily sedated and locally anaesthetised, it is easy to imagine how the mouth may be opened beyond its ‘normal’, comfortable limit. This may create abnormal stresses on the masticatory and support structures of the skull.

Many horses chew on the incisor plates of speculae during dental examinations and can generate immense force during the closing and power strokes of the masticatory cycle (Staszek et al. 2006; Huthmann et al. 2009). This activity decreases with sedation but is not always eliminated. In our experience, the addition of local anaesthesia usually stops movement in the majority of cases. Masticatory muscle tone (specifically the jaw closing musculature) although reduced is always maintained to some degree and will therefore exert some pressure on the incisor plates of an open speculum. It has been postulated that the full mouth speculum transmits powerful muscular forces through the incisor teeth to the maxilla and the mandible, with the maxilla being much weaker of the two (Widmer et al. 2010). This could result in fractures either when the force applied is very high (i.e. when chewing on the speculum or possibly where the speculum has been forced open) or in horses with weakened bone. This horse was relatively old (17 years), as in previous cases (Widmer et al. 2010), and it is possible that it had some degree of age-related change in bone microstructure which may have predisposed it to fracture (Furst et al. 2008), although reduced mineral density was not discernible with digital radiography.

Surgical repair was considered using one of a number of techniques available (Auer 2012). However, once the sedation and local anaesthesia had worn off, it became clear that the horse was comfortable, willing and able to eat soft feed. Radiographically, the fracture was closed, stable and minimally displaced. In view of these observations, a decision was made to manage this case conservatively in the short term and after careful discussion with the owner the horse was discharged from the hospital with instructions for him to be box rested at home and monitored closely for signs of oral dysphagia or obvious fracture destabilisation. Ultimately, this horse responded well to conservative management without the need for surgical fixation. This supports the observation by Townsend (2015) that trauma to the supporting bones around cheek teeth sustained during exodontia is remarkably well tolerated and even extensive fractures heal well.

While appropriate sedation, restraint and loco-regional analgesia are important in reducing the risk of this complication (through the reduction of abnormal high forces on the speculum), this case demonstrates that this does not eliminate the risk entirely and other as yet unidentified factors may have contributed to this outcome.

Authors’ declaration of interests

No conflicts of interest have been declared.

Ethical animal research

Not relevant to this case study.

Authorship

Both authors have contributed equally to all aspects of the study.

Manufacturers’ addresses

1Equine Blades Direct, Wedmore, Somerset, UK.
2Milacath, Erlanger, Kentucky, USA.
3Vetoquinol, Buckingham, Buckinghamshire, UK.
4Martindale Pharmaceuticals, Romford, Essex, UK.
5Bimeda, Anglesey, Wales, UK.
6Dechra, Stoke-on-Trent, Staffordshire, UK.
7MSD, Milton Keynes, Buckinghamshire, UK.
8CP Pharmaceuticals, Wrexham, Wales, UK.
9Inseminet Conceptis, Birmingham, UK.
10Allen & Page, Thetford, Norfolk, UK.

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Case Report

Computed tomographic diagnosis: Traumatic lingual process fracture and surgical resection

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Keywords: horse; computed tomography; temporohyoid osteoarthropathy; hyoid apparatus; head-shaking

Summary

A 9-year-old mare was presented with abnormal behaviour and head-shaking when ridden. Pathology of the hyoid apparatus was suspected and computed tomographic (CT) examination of the head was suggested. Computed tomographic images revealed a fractured lingual process (LP) of the basihyoid bone. Surgical resection of the fractured LP was performed. The mare recovered uneventfully and symptoms improved 10 weeks post-operatively.

Introduction

Fracture of the hyoid apparatus is a rare condition in horses (Chalmers et al. 2006a). The hyoid apparatus consists of an assembly of small bones. The single basihyoid bone (BH) and the LP, located on the midline, are bilaterally secured by the paired stylohyoid, ceratohyoid and thyrohyoid bones (Getty 1975; Divers et al. 2006; Cheetham et al. 2014) (Fig 1). This apparatus supports the tongue, larynx and pharynx and is able to move in a rostrocaudal direction at the stylohyoid-ceratohyoid joint (Blythe and Watrous 1997). The hyoid apparatus and its attaching muscles appear to play an important role in nasopharyngeal stability during exercise (Cheetham et al. 2014).

Fractures of the hyoid apparatus have only been reported in the stylohyoid bone. They are reported as a consequence of temporohyoid osteoarthropathy (THO) and in one case with osteomyelitis secondary to infection (Pease et al. 2004; Chalmers et al. 2006a). The clinical signs associated with THO can be both non-neurological and neurological depending on the progression of the disease and anatomical structures involved. Non-neurological signs can include refusing to take the bit and signs of discomfort such as head-shaking and ear rubbing. Neurological signs include facial nerve paralysis, vestibular signs and dysphagia (Walker et al. 2002; Divers et al. 2006; Palus et al. 2012). The diagnosis is based on clinical and neurological examination and diagnostic imaging.

Diagnostic imaging of the hyoid bone with conventional diagnostic modalities is limited due to the superimposition of multiple structures (Cornelisse et al. 2001; Hilton et al. 2009). Radiography of the head and guttural pouch endoscopy provide a good overview of the proximal stylohyoid and temporohyoid area. This can be helpful for the diagnosis of THO (Walker et al. 2002). Ultrasonographic examination of the larynx and intermandibular space has been described to image the ventral part of the hyoid apparatus, particularly the BH and LP (Archer 2014). Advanced imaging with CT provides high diagnostic value for evaluating the equine head including the hyoid apparatus and is the diagnostic imaging modality of choice (Hilton et al. 2009; Manso-Diaz et al. 2015). Thus exact diagnosis and surgical planning are possible. This report describes the clinical appearance, diagnostic imaging and subsequent surgical resection of a fractured LP.

Case history

A 9-year-old Warmblood mare, weighing 550 kg, was presented with a sudden onset of nonspecific behavioural problems, especially when being ridden. The rider reported the horse refusing to ‘take’ the bit and collect the neck properly with episodes of head-shaking when ridden. The horse had been examined for lameness and poor performance 4 weeks prior to referral. The referring veterinarian performed a thorough clinical, dental and lameness examination. A nonspecific pain reaction was detected when pressure was applied to the intermandibular area up to the temporomandibular joint area during examination. Dental examination of the oral cavity did not reveal substantial abnormalities. The referring veterinarian treated the horse with intra-articular corticosteroid injection of...
the temporomandibular joint and advised the bit should be changed.

The horse did not respond to the treatment. Fewer episodes of head-shaking were recognised when an unjointed bit was used. The referring veterinarian performed a repeated physical examination focused on the head and neck. Pain to pressure in the intermandibular area was still present. Also palpation and passive motion of the left facet joints C5–C7 provoked a slight pain reaction. Radiographs of the cervical spine and skull were obtained and a cyst-like lesion was subsequently thought to be in the proximal part of the left stylohyoid. The horse was treated for cervical neck pain with intra-articular corticosteroids injections to the left articular process joints of C5/C6 and C6/C7. The referring veterinarian referred the horse for further investigation of the head-shaking 6 weeks after treatment because of lack of response.

Case details

Initial examination confirmed a pain reaction to pressure over the entire area of the intermandibular space. Pain reaction and discomfort were elicited by squeezing the mandibles together. Examination of the horse in hand in walk and trot and on the lunge line in walk, trot and canter with a conventional head collar did not reveal any abnormalities. For the following ridden examination a bridle with a single broken bit was used. When the mare was bridled she demonstrated resistance by raising her head. During the ridden examination she showed moderate to severe permanent head flicking in a vertical direction in all paces when asked to flex the neck. Signs disappeared when the horse’s neck was in an elongated position and there was no traction on the reins. No other abnormalities were detected during the physical examination. Haematology was within normal limits. Complete dental and ophthalmological examinations were performed and remained unremarkable. Neurological examination did not indicate any deficits.

Videoendoscopic examination of the upper airway and guttural pouches did not show any abnormality. Lateral radiographs of the head were obtained and interpreted as normal (Fig 2). Computed tomography (CT) was planned to further investigate the mandibular region and hyoid apparatus.

Computed tomography findings

Two days following initial examination, a CT examination under general anaesthesia was performed.

An intravenous (i.v.) catheter was placed in the left jugular vein and xylazine (0.6 mg/kg bwt i.v.) and butorphanol (0.01 mg/kg bwt i.v.) administered as premedication. General anaesthesia was induced with ketamine (2.2 mg/kg bwt i.v.) and diazepam (0.03 mg/kg bwt i.v.). An endotracheal tube was placed and oxygen administered while maintaining general anaesthesia with a continuous rate infusion of 2 mL/kg bwt/h of an infusion containing 500 mL Guaifenesin 5%, 500 mg ketamine and 250 mg xylazine. The horse was positioned in dorsal recumbency for image acquisition.

Scans were acquired with a 4 detector row helical scanner (Philips CT Vision Mx8000, 120 kV, 150 mAs, 512 × 512 matrix, 256 × 256 mm field of view, soft tissue and bone algorithms)\(^1\).

Isovolumetric images were available in transverse, sagittal and frontal planes with a slice thickness of 1 mm. Evaluation of the images revealed a minimally displaced fracture of the LP with irregular fracture margins (Fig 3). Within the fracture gap several radiodense fracture fragments of various sizes were detected best revealed in transverse plane (Fig 4). The adjacent soft tissue was evaluated using soft tissue algorithms. In transverse plane the attachment of the omohyoideus and genioglossus muscles around the fractured area of the LP presented with inhomogeneous structure and slightly hypoattenuating density.

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A diagnosis of a comminuted, mildly displaced fracture of the LP with involvement of the muscle attachments was made. Further assessment of the images for surgical planning was obtained using 3D reconstruction.

Surgical procedure
The fractured LP was considered the cause of the symptoms therefore a surgical resection of the LP was planned under general anaesthesia.

Preparation and medical treatment
Surgery was performed under general anaesthesia in dorsal recumbency. Preoperatively the mare was administered procaine penicillin (10,000 iu/kg bwt i.m.) and flunixin meglumine (1.1 mg/kg bwt i.v.). Xylazine (0.6 mg/kg bwt i.v.) and butorphanol (0.01 mg/kg bwt i.v.) were used as preanaesthetic medication. General anaesthesia was induced with ketamine (2.2 mg/kg bwt i.v) and diazepam (0.03 mg/kg bwt i.v.). General anaesthesia was maintained after endotracheal intubation with isoflurane delivered in oxygen using a circle breathing system. Throughout the anaesthetic period isotonic saline solution (4 mL/kg bwt/h) was administered i.v.

The ventral cervical and intermandibular area was clipped beginning rostral to the mandibular lymph nodes caudal to beyond the cricoid cartilage following routine preparation of the surgical field for aseptic surgery.

Surgical resection of the LP
A 10 cm skin incision was made starting 1 cm rostral to the LP extending over the BH. The omohyoid and sternohyoid muscles were bluntly separated to reach the LP. Muscle fibres were detached from the LP to expose the fracture site and articulation with the BH. The LP was disarticulated from the BH using curved Mayo scissors and grasped with a Backhaus towel clamp (Fig 5). The LP was freed from remaining muscle fibres of the geniohyoid, omohyoid and sternohyoid using blunt dissection until its total excision. The omohyoid and sternohyoid muscles and subcutaneous tissue were reapposed using absorbable suture in a simple continuous pattern. The skin was closed using nonabsorbable suture in a simple interrupting matter. Specimens of bone and soft tissues were submitted for histopathological examination.

Post-operative care
The mare was treated with procaine penicillin (10,000 iu/kg bwt i.m. once a day) and flunixin meglumine (1.1 mg/kg bwt i.v. once a day) for the following 3 days. Subsequently, flunixin meglumine (1.1 mg/kg bwt per os once a day) was administered orally for 3 days. Daily examinations included checks for tongue movement and the intake of food and water. No abnormalities were detected during chewing and swallowing. The surgical side was examined for local swelling.

Outcome
The mare recovered uneventfully after surgery and was discharged after 6 days. Owner and referring veterinarian were instructed to handwalk the horse for 6 weeks followed by gradual return to normal routine. The use of a soft bit for example a synthetic bit during the first 4 weeks of recovery was suggested.

After 10 weeks the horse was presented for re-examination. Palpation of the intermandibular area did not provoke a painful or defence reaction anymore. A slight swelling of the surgical area was palpated. Ultrasonographic examination of the swelling revealed a 1.5 x 1 cm sized accumulation of hypoechoic fluid adjacent to the subcutis. Palpation of the surgical area was unremarkable. During ridden examination a short episode of head-shaking was observed after the horse was forced to flex the neck. However, the horse stopped the head-shaking after this short episode. It was subsequently recommended to gradually increase the workload and have the submandibular swelling rechecked on a regular basis by the referring veterinarian.

A telephone consultation with the owner 5 months post-operatively revealed that the horse was back in full training without any signs of head-shaking regardless of the bit that was used.

Histopathological examination revealed an irregular arrangement of cartilage and lamellar bone tissue within the medullary cavity and compact bone of the LP. This was
sugges
tive of reactive new bone formation most likely as a re
sult of a fracture.

Discussion
To the authors’ knowledge, fracture of the LP in horses has not been previously reported. Fracture of the hyoid apparatus has been reported in horses, usually involving the stylohyoid bone. Stylohyoid fractures are linked to THO (Walker et al. 2002; Divers et al. 2006; Palus et al. 2012). Affected horses show enlargement and eventual ankylosis of the temporohyoid joint that results in decreased range of motion of the hyoid apparatus with possible fracture of the stylohyoid or petrous temporal bone (Pease et al. 2004; Divers et al. 2006; Bras et al. 2012). In one case stylohyoid fracture was described in the absence of temporohyoid disease. The underlying cause was assumed to be osteomyelitis secondary to infection after periaryngeal injection (Chalmers et al. 2006a). Osteomyelitis of the stylohyoid secondary to infectious pharyngeal abscesses has also been recently described in calves (Nuss et al. 2015).

In the present case, no signs of infection of the LP and adjacent tissue were detected. Histopathological examination revealed new bone formation as a result of the fractured LP. Hyoid bone fractures in man are commonly caused by direct blunt or penetrating trauma and in rare cases by indirect trauma or bone stress (Gupta et al. 1995). Main symptoms in hyoid fractures in man are pain in the throat (Gupta et al. 1995). The exact cause for the LP fracture in the current case remains unknown. A sudden blunt trauma to the tongue like in man seems to be the most probable cause. Injury of the LP due to previous surgery in this area or endotracheal intubation could be ruled out in the current case.

The mare’s behavioural problems and reluctance to take the bit improved after surgical excision of the LP. Suggested cause was flexion of the neck and pressure of the bit to the tongue. It was recently described that the bit contacts the apex of the tongue and triggers its retraction (Cook 2014). When the apex is retracted and a closed jaw compresses the body, the root of the tongue will expand (Cook 2014). In our case it is considered that the expansion of the root of the tongue by contraction of the genioglossus was pulling on the LP and evoking a defence reaction when ridden. Furthermore, the sternohyoid and omohyoid muscle function as accessory respiratory muscles and increase their function during exercise by caudal traction of the hyoid apparatus (Holcombe and Ducharme 2007; Derksen 2012). Therefore, we assume that in the presented case rostrocaudal traction of the fractured LP and BH by attaching muscles triggered pain and thereby head-shaking and behavioural problems when ridden. Symptoms improved when an unjointed synthetic bit was used and the pressure to the tongue’s apex was decreased. In conclusion the suggested cause of pain in this case is the interaction of the bit with the root of the tongue.

During clinical examination the patient showed unspecific pain reaction to pressure in the intermandibular and periauricular region. Pain was also elicited by squeezing the mandibles together. In previous reports this manipulation is suggested to trigger movement of the BH (Divers et al. 2006; Palus et al. 2012). In our case, movement of the BH could have caused pain by movement of the fractured LP.

Computed tomographic examination of the head is widely used, especially for disorders of the sinonasal region, teeth, temporomandibular joint and temporohyoidal region (Puchalski 2007). It has also been used to evaluate the hyoid apparatus in the horse (Chalmers et al. 2001). Evaluation of the ventral hyoid area with conventional radiography is limited due to the complex anatomy of this area (Chalmers et al. 2001; Barakzai and Weaver 2005; Hilton et al. 2009). Endoscopic examination of the guttural pouches allows evaluation just of the proximal part to the stylohyoid bone (Hilton et al. 2009). Ultrasonographic examination of the larynx and ventral hyoid apparatus has been recently described (Chalmers et al. 2006a,b). Structures of the hyoid that can be visualised include parts of the BH including the LP, portions of the ceratohyoid and thyrohyoid bones and base of the tongue (Chalmers et al. 2006a,b; Archer 2014). In the reported case ultrasonographic examination of the ventral hyoid apparatus was not performed. An ultrasonographic image of the LP could therefore have suggested irregular bone margins and new bone formation and assisted in formulating a diagnosis. However, CT examination not only allowed for an exact diagnosis but also facilitated preoperative planning and ruled out additional bone-related diseases as THO or additional soft tissue lesions.

To date surgical excision of the LP has not been described. The complete disarticulation and resection was achievable similar to the ceratohyoidec
tomy in horses with THO. The risk of injury is low due to a median incision in the ventral laryngeal area and fewer nerves and vessels in this area compared with the paramedian approach of the ceratohyoidec
tomy (Divers et al. 2006). The resection of the LP in comparison to the ceratohyoidec
tomy needs only the disarticulation of the LP-BH articulation. The mare’s recovery after surgery was rapid and uneventful.

Improvement of clinical symptoms after surgery confirmed the assumption that fracture of the LP was the underlying cause of symptoms. Clinical symptoms started approximately 5 weeks prior to referral with sudden onset. Post-surgical examination after 10 weeks revealed a marked improvement of symptoms with a residual head-shaking at the beginning of riding. After 5 months the owner reported that the horse was back in full training and not showing episodes of head-shaking or behavioural problems. The progression of symptoms before as well as improvement after surgery supports the assumption that the fracture of the LP was more likely the underlying cause of symptoms rather than idiopathic head-shaking.

This is the first time an entity of the hyoid apparatus other than THO and osteomyelitis of the stylohyoid bone has been reported as a cause of head-shaking in a horse. However, THO is the most common disease of the hyoid apparatus and should be first ruled out. Radiography, endoscopy and ultrasonography of the hyoid apparatus can be very helpful diagnostics. However, advanced imaging modalities and CT in particular may be needed to reach an exact diagnosis in this particular area. The mare’s recovery after surgical intervention was good as well as long-term follow-up. Resection of the LP should be considered in similar cases.
Authors’ declaration of interests

No conflicts of interest have been declared.

Ethical animal research

This was a clinical case presented to the hospital. No research was performed. Owner informed consent was obtained.

Acknowledgements

We would like to thank R. Pellmann for referring the case and initial treatment provided.

Authorship

B. Rohwerder contributed to diagnostic imaging, surgical procedure, case report design and preparation of the manuscript. A. Buss contributed to diagnostic imaging (CT) and K.J. Boening contributed to the surgical procedure. The manuscript has been read and approved by all authors.

Manufacturers’ addresses

1Philips GmbH Market DACH Healthcare, Hamburg, Germany.
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Case Report

Treatment and ophthalmic sequelae in a horse with facial cellulitis and orbital abscess

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Keywords: horse; orbital abscess; facial cellulitis; optic nerve atrophy; tooth extraction; deep facial venous sinus

Summary

A 22-year-old Quarter Horse gelding with a history of dental (107) extraction 2 weeks previously was presented with facial cellulitis and an orbital abscess. The abscess was drained surgically dorsally and ventrally to the zygomatic arch and the horse was treated with parenteral antimicrobial and anti-inflammatory drugs. The affected eye was protected with a temporary tarsorrhaphy and treated via a subpalpebral catheter. Six months after presentation, the horse was in excellent general condition and returned to previous level of exercise but showed blindness on the affected side. Pallor of the optic disc and severe attenuation of the retinal vessels, suggestive of optic disc atrophy, were present.

Introduction

An orbital abscess is defined as the accumulation of purulent material within the tissues surrounding the eye. Orbital abscesses may be the consequence of a foreign body penetrating the conjunctiva, a septic embolus or may develop secondary to an ocular infection or an infection in the adjacent cavities (oral cavity or paranasal sinuses). An untreated or poorly responsive panuveitis may progress to endophthalmitis and form an orbital abscess. Streptococcus equi ssp. Zooepidemicus (Van den Top et al. 2007), Actinomyces, Habronema spp. and Cryptococcus may cause orbital cellulitis (Scott et al. 1974; Roberts et al. 1981; Gilger 2010). Phycomycosis (Conidiobolus and Basidiobolus) has been reported (Vallefuoco et al. 2014) to spread from the guttural pouch or the nasopharynx into the orbit (Gilger 2010). Facial cellulitis may be primary (Farstvedt et al. 2004; Castagnetti et al. 2008), a consequence of penetrating foreign bodies, or follow dental, periodontal or oral infections.

In this report, we describe the case of a horse presented with orbital abscess and facial cellulitis. A thrombus in the deep facial venous sinus was diagnosed sonographically. The abscess was drained dorsally and ventrally to the zygomatic arch and through the masseter muscle.

Case history

A 22-year-old Quarter Horse gelding was presented to the Swiss Institute for Equine Medicine of the University of Bern due to facial and orbital inflammation. The horse had developed difficulty masticating 2 weeks prior to presentation. At that time, the third right maxillary premolar (Triadan 107) was extracted orally due to sagittal tooth fracture and signs of periodontal disease. An intraorbital nerve block was performed at the time of cheek tooth extraction. The horse was not treated with antimicrobials initially. Four days prior to referral, facial swelling developed and the horse was treated with trimethoprim sulfonamide, 30 mg/kg bwt per os q. 12 h [Rota-TS Oral bioser], and phenylbutazone, 2 mg/kg bwt per os q. 24 h [Equipalazone Paste], unsuccessfully.

Clinical findings

At presentation, the gelding had normal vital parameters and showed diffuse swelling of the right facial and orbital regions. There was severe prolapse of the third eyelid, chemosis, exophthalmos and epiphora (Fig 1). The right menace, dazzle, direct and consensual pupillary reflexes were absent. The cornea presented a horizontal line of oedema due to the incomplete closure of the eyelids, and was positive to fluorescein staining. An examination of the oral cavity showed the filling of the alveolus of the extracted tooth with granulation tissue. An endoscopic examination of the nasal passages, pharynx and guttural pouches was unremarkable.

Diagnostic tests

Radiographic examination of the orbit, paranasal sinuses and tooth roots showed periobital soft tissue swelling containing multifocal fine radiolucencies. No signs of sinusitis or osseous changes could be observed (Fig 2). An ultrasonographic examination of the orbit and facial region revealed pronounced soft tissue swelling and echoic fluid containing multiple small (1–3 mm) hyperechoic foci casting a dirty acoustic shadow suggestive of the presence of fluid of high cellularity and gas in the retrobulbar regions. Heteroechoic material distended the deep facial venous sinus (Fig 3a). A fine needle aspiration of the retrobulbar tissue yielded malodorous brown fluid. Bacterial culture grew Streptococcus plurianimalium sensitive to common antimicrobials used in equine medicine. Complete blood count and chemistry profile showed hypoalbuminaemia (27 g/L; reference range 32.2–39.9 g/L) and an elevation of serum amyloid A (2321 μg/ml; reference range <8 μg/mL).
Outcome

The horse was treated with penicillin G, 30,000 i.u/kg bwt i.v. q. 6 h (Penicillin Natrium Streuli ad us. vet.),\textsuperscript{3} gentamicin, 6.5 mg/kg bwt i.v. q. 24 h (Pargenta-50 ad us. vet.),\textsuperscript{4} metronidazole, 25 mg/kg bwt per os q. 12 h (Metronidazol Ph.Eur),\textsuperscript{5} and flunixin meglumine, 1.1 mg/kg bwt i.v. q. 12 h (Flunikine).\textsuperscript{6} After 7 days of i.v. antimicrobials, treatment was continued with trimethoprim sulfonamide, 30 mg/kg bwt per os q. 12 h, metronidazole, 25 mg/kg bwt per os q. 12 h, and phenylbutazone, 2 mg/kg bwt per os q. 24 h, for 5 more days.

A 3 cm incision was made caudal to the orbit and dorsal to the right zygomatic arch so that a second incision was created dorsal to the zygomatic arch to allow through and through lavage. A third 2 cm vertical incision was created ventrally under sonographic guidance 2 days later to provide drainage to a second area of fluid accumulation. Incisions were merged and a Penrose drain placed between them. The Penrose drain was left in place for 5 days and the draining tract was rinsed daily with diluted povidone–iodine solution.

The eye was treated with ice packed bandages and tobramycin ophthalmic ointment q. 6 h (Tobrex, ocular ointment).\textsuperscript{7} The cornea developed 2 superficial ulcers and the third eyelid prolapse persisted. A temporary tarsorrhaphy was performed with a 2-0 nonabsorbable monofilament synthetic suture (Prolene)\textsuperscript{8}, and a subpalpebral lavage (SPL) system was placed through the upper eyelid (Fig 4) and kept for 7 days (Ocular Lavage Kit).\textsuperscript{9} Autologous serum was given q. 4 h via the SPL system and tobramycin (Tobrex, ocular solution)\textsuperscript{7} therapy continued.

The facial swelling chemosis and third eyelid prolapse resolved progressively and were no longer present 12 days after presentation. Corneal ulcers resolved 14 days after presentation. The horse was discharged 14 days after presentation and the eye remained nonvisual.

A recheck 6 months after initial presentation showed that the horse was in good general condition and was being used as a pleasure riding horse (Fig 4). The right direct pupillary light reflex, menace and dazzle reflexes were absent on the right side. Optic nerve atrophy, visible as pallor of the optic disc and attenuation of the circumferential blood vessels was observed (Fig 5). Ultrasonographic examination showed resolution of the deep facial venous sinus thrombosis (Fig 3b).

Discussion

Defining the structures affected in cases of orbital swelling is relevant to clinical management and recognition of potential complications. Eye and adnexa should be examined to rule out primary (e.g. bacterial conjunctivitis) or secondary lesions (e.g. corneal ulcers secondary to improper lid closure).
Exophthalmos is defined as an anterior displacement of a normal-sized globe within the orbit and is usually the consequence of an inflammatory or invasive process posterior to the globe (orbita and/or peri orbital sinuses such as conchofrontal, caudal maxillary and sphenopalatine sinuses) (Plummer 2007; Gilger 2010). Differential diagnoses in this situation include bacterial infection, retrobulbar oedema and/or haematoma, retrobulbar dermoid cysts, and neoplasias such as adenoma, adenocarcinoma, basal cell carcinoma, fibroma, haemangiosarcoma, lymphoma, mastocytoma, melanoma, nerve sheath tumours, neuroendocrine tumours, plasmacytoma or squamous cell carcinoma. In cases of periorbital swelling without exophthalmos, a space occupying lesion behind the eye is less likely and differential diagnoses include bacterial cellulitis and/or abscessation, lacrimal gland abscesses, trauma, granuloma, cutaneous leishmaniosis (J. Racine and C. Koch, personal communication 2016) and neoplasia such as sarcoïds and fibrosarcoma (Greenberg et al. 2011).

Damage due to compression, infection or inflammation were the likely causes of optic nerve atrophy in the case presented here. Optic nerve atrophy is encountered in diseases such as optic neuritis, equine recurrent uveitis, external compression, trauma, infarction, glaucoma, chemical intoxication, sphenopalatine sinus infection and neoplasia (Cutler et al. 2000; Barnett et al. 2008), and often goes together with retinal degeneration.

The orbital septum, a periosteal reflexion that extends from the bony orbit to the eyelids (Fig 6), is a relevant structure to the discussion of orbital cellulitis and abscessation. A postseptal abscess has the potential to lead to the loss of the affected eye and to infect meninges and encephalic structures. Development of an orbital abscess after dental infection is an uncommon but severe condition that may lead to loss of vision, meningitis or encephalitis in human patients (Zachariades et al. 2005). A case of fatal suppurative meningoencephalitis has been described in a horse concurrently affected with orbital cellulitis (Smith et al. 2004). Subperiosteal abscesses, another form of postseptal abscess, can occur in children as a consequence of a pansinusitis. The infection propagates from the ethmoidal sinus to the bone adjacent to the orbit to form a fluid pocket under the
periosteum (Zachariades et al. 2005). Similar propagation of infection from the paranasal sinuses to form subperiosteal postseptal orbital abscess have not been described in horses. Advanced imaging modalities, such as computed tomography and magnetic resonance imaging, can help to identify the extension of lesions in pathologies that affect the head of equids. Computed tomography can be performed standing or under general anaesthesia and it is generally more expensive. It is superior for the evaluation of soft tissues (retrobulbar mass, meningitis) or identification of foreign material (Van den Top et al. 2007; Greenberg et al. 2011).

The history of dental problems and progression of the disease makes retrograde seeding from a septic thrombus in the deep facial venous sinus or ascending infection along the infraorbital canal due to infraorbital nerve block plausible explanations for the cellulitis and abscess in the case reported here. We could speculate that the tooth extraction may have predisposed to thrombus formation in the deep facial sinus (Matsuda et al. 2010) and the valveless nature of this vessel may have facilitated stabilisation of the thrombus. Propagation of infection to the retrobulbar space may then have happened by retrograde seeding of infected thrombus particles or by development of a cellulitis around the thrombus that propagated in the direction of the retrobulbar space. There are three venous sinuses that lie below the masseter muscle and are connected to the facial vein (Fig 7). These valveless sinuses work like a pump pressed by the masseter muscle during mastication to drain blood to the maxillary vein and then to the external jugular vein. The sphenopalatine and the infraorbital veins are connected to the deep facial venous sinus. The infraorbital vein is fed by the rami dentales (dental branches) that collect blood from the alveoli of the maxilla. The vein runs through the infraorbital canal together with the infraorbital artery and then reaches the pterygopalatine fossa before joining the deep facial venous sinus. The infraorbital vein is fed by the rami dentales (dental branches) that collect blood from the alveoli of the maxilla. The vein runs through the infraorbital canal together with the infraorbital artery and then reaches the pterygopalatine fossa before joining the deep facial venous sinus. Besides its communication to the facial vein, the deep facial venous sinus is also directly connected to the ophthalmic plexus (Wissdorf et al. 2010; Barone 2012; Bach et al. 2014).

Meningoencephalitis is a severe disease with mortality rates from 96.4% (Toth et al. 2012) to 100% (Reilly et al. 1994; Smith et al. 2004; Bach et al. 2014) in horses. In cases where meningoencephalitis seemed to be secondary to bacterial infections in the region of the head (e.g., teeth infections, sinusitis, and orbital cellulitis), abscesses of the pituitary gland were observed in 4 of 7 (Smith et al. 2004) and 4 of 5 cases (Bach et al. 2014). In a case series of 4 horses with pituitary abscesses and basilar empyema, all horses had signs of upper airway infections (sinus or guttural pouch) before developing meningoencephalitis. Since the cavernous venous sinus surrounds the pituitary gland and is in direct contact with the ophthalmic plexus and the deep facial
venous sinus, this pathway may be an important haematogenous source of infection of the brain and meninges (Smith et al. 2004; Bach et al. 2014). In the case reported here, haematogenous infection may have followed this path and persisted in the periorbital region.

Treatment options for orbital cellulitis and abscessation include topical and/or systemic antimicrobials, anti-inflammatory drugs and drainage. Enucleation may be necessary if severe endophthalmitis is present or if the orbital infection does not resolve. Abscess drainage achieved by transoral orbitotomy or lateral orbitotomy are described techniques to treat orbital abscesses in dogs (Vallefuoco et al. 2014). In the case presented here the differences in anatomy, the fluid accumulation in the retrobulbar space and periorbital tissue and the sonographic visualisation of the abscessed areas dictated the approach. The supraorbital adipose tissue and the temporal part of the frontoscutularis muscle present caudal to the orbit and medial to the zygomatic arch provide a convenient access to the deeper orbital tissues analogous to the approach described for small animals. The auriculopalpebral nerve (Fig 7) should be avoided when using this approach. The dorsal location of the opening is a disadvantage of this approach. Temporary tarsorraphy and the use of SPL to aid the administration of ophthalmic therapy may be necessary to protect the cornea in cases of severe exophthalmos or chemosis (Gilger 2010).

The successful treatment of an equine patient with facial cellulitis and an orbital abscess is reported here. The pertinent anatomy and relationships of the orbital area are dissected and described and the diagnostic modalities available to explain underlying aetiopathophysiological mechanisms and guide therapy in cases of orbital abscesses discussed. Knowledge of the anatomy of the complex facial vessels may help in understanding propagation of infections in the equine head, particularly after routinely performed procedures such as dental extractions.

Authors’ declaration of interests
No conflicts of interest have been declared.

Ethical animal research
The horse of this case report was a client-owned animal. The owner gave his consent for publication. The horse was examined by a group of highly specialised veterinarians (surgery, ophthalmology and internal medicine) and treated with high standards of veterinary care. Client confidentiality
was maintained in this case report. No human can be identified on any of the pictures.

Acknowledgements
We would like to thank Thea Rhyner from the NPZ in Bern for referral of the case.

Authorship
Julien Racine provided clinical care and prepared the manuscript. S. Borer supervised the ophthalmic treatments and participated in preparation of the manuscript. M. Stoffel and M. Klopfenstein prepared the anatomic illustrations. M. Klopfenstein Bregger supervised the case and was involved in writing the relevant sections. C. Navas de Solis performed the ultrasonographic follow-up of the case and participated in preparation of the manuscript. M. Klopfenstein Bregger supervised the case and was involved in preparation of the manuscript. All authors approved the final manuscript.

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Clinical Commentary

Complications following oral extraction of cheek teeth: What’s next?

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The renewed interest in equine dentistry since the end of the 20th century has led to great scientific advancements in the knowledge of equine dental disease and supported the development and availability of modern and efficient instrumentation. Oral extraction performed in the standing horse using regional nerve anaesthesia, such as the maxillary or inferior alveolar nerve blocks, is regarded as being the gold standard for exodontics. This has significantly reduced the complication rate (Duncanson 2004), compared with formerly more popular techniques such as dental repulsion (Prichard et al. 1997; Debelian et al. 2006). However, the frequency of these conditions decreased drastically with the widespread use of antibiotics. Nevertheless, oral extraction must not be taken for granted as complications can still occur (Dixon et al. 2005, 2009). They can be grossly divided into two groups. The first group is encountered during the extraction procedure and often originate from more technical aspects such as tooth fracture during extraction, insufficient forceps fixation on the tooth’s crown to allow simple oral extraction, space limitations especially in the back of the mouth and in pony breeds, and damage to surrounding tissues/teeth due to improper instrument use. The second group arises from infectious problems responsible for persistent ensuing infections involving the dental alveolus and its surrounding structures such as the sinus cavities.

Invasive procedures of oral tissues such as root scaling, tooth extraction, endodontic treatment and periodontal surgery are known to cause transient bacteraemia in man (Debelian et al. 1994; Forner et al. 2006). Routine dental scaling and extraction have been proven to provoke transient bacteraemia in dogs and cats (Black et al. 1980; Harari et al. 1991, 1993; Nieves et al. 1997). It was recently demonstrated that transient bacteraemia of oral origin commonly occurs during dental extraction in horses with bacterial genera isolated from swab samples of extracted teeth largely corresponding with those identified in blood cultures (Kern et al. 2017). The significance of this temporary bacterial spread is, however, questioned as these bacteria are rapidly eliminated by the reticuloendothelial system in the healthy individual (Silver et al. 1975; Debelian et al. 1994). In man, predisposing conditions such as an immunosuppressive status or heart disease can induce potential dangerous complications including infective endocarditis, myocardiab or cerebral infarction and other nonoral diseases (Kilian 1982; Syjänen et al. 1989; Nord and Hemdahl 1990).

The development of facial cellulitis and an orbital abscess following oral extraction of a cheek tooth in a horse as described in the article in this issue by Racine et al. (2017) is an extremely exceptional complication. Two comparable cases have been described (Vlaminck et al. 2001; Bach et al. 2014). Following oral extraction of a sagittally fractured 109 cheek tooth, a 4-year-old Warmblood mare developed a retrograde necrotising thrombophlebitis of the deep facial venous sinus (Vlaminck et al. 2001). The mare had a concomitant secondary sinusitis treated by regular lavage through a rostral maxillary sinus trephination. Two days post-surgery, the mare became febrile and developed a diffuse, sensitive swelling of the right retrobulbar region with slight exophthalmus which further spread caudally to the temporomandibular joint and cranial aspect of the right parotid gland. Radiographic and ultrasonographic examinations were unsuccessful in identifying any underlying pathological process. Despite an intense medical treatment, 1 week after surgery her condition rapidly deteriorated (anorexia, depressed), accompanied by development of negative menace, dazzle and direct pupillary reflexes in the right eye and resulted in sudden shock, collapse and death. An autopsy revealed the presence of a purulent, foul-smelling content in the right deep facial venous sinus which could be traced all the way from the area of the 109 alveolus to the cavernous venous sinus surrounding the pituitary gland. Histology further identified a massive neutrophilic infiltration and the presence of numerous Gram-positive coccoid bacteria in and around the brain stem. The second case was described in a case series of five horses developing meningitis following sinus surgery (Bach et al. 2014). A 23-year-old Warmblood gelding was diagnosed with sinusitis secondary to apical disease of Triadan 109 and 110 cheek teeth. Treatment consisted of oral extraction of infected teeth and regular sinus flushing after trephination of the conchofrontal and caudomaxillary sinus. Within approximately 1 week after the start of treatment, the horse developed progressive neurological disease leading to recumbency, nystagmus, seizures and death. Necropsy and histological analysis of tissue samples confirmed meningoencephalitis, necrotising inflammation of the parotid gland and intracranial phlebitis and bacteremia.

In older human literature, such serious post-extraction complications have been reported without apparent associations with immunocompetence of these patients. They include cavernous sinus thrombosis (Wen-Der Yung et al. 1991), facial space abscesses (Bullock and Fleishman 1985) and brain abscesses (Kiser and Kending 1963; Hollin et al. 1970). However, the frequency of these conditions decreased drastically with the widespread use of antibiotics. The mechanism by which infection can spread from a dental origin as hypothesised in the report by Racine et al. (2017) is based on the induction of thrombus formation secondary to tooth extraction and retrograde seeding from the septic thrombus to more proximally located structures situated along the venous vascular system. This phenomenon
of thrombus formation in venous sinuses of the head is, although rare, a well known disease process in man (i.e. thrombosis of the cavernous sinus at the base of the brain). Multiple reports describe thrombosis of the cavernous sinus secondary to spreading infection in the nose, sinuses, or teeth (Ogundiya et al. 1989; Cannon et al. 2004; Desa and Green 2012). Three factors are necessary for a thrombus to form within a blood vessel, including damage to the intima, hypercoagulability of the blood and slowing or stasis of the bloodstream (Scarpellino et al. 1936). Acute inflammation may be all that is required to initiate integrity changes of the inner lining of blood vessels and activate coagulation. It has been suggested that this can be initiated by the surgical trauma of removal of the tooth (Ogundiya et al. 1989). The valveless nature and the wider diameter of the deeper venous sinuses of the horse’s head contribute to slowing of the bloodstream and facilitated spread of septic emboli. Necropsy findings from reports by Vlaminck et al. (2001) and Bach et al. (2014) confirm the likelihood of vascular spread of infection that might also have been responsible for the complications encountered in the report by Racine et al. (2017).

As Racine et al. (2017) mentioned, the iatrogenic route of infection through performance of a regional nerve block should also be considered as a possible cause of retrobulbar infection. Rerograde spread of infection through the intraorbital canal following an infraorbital nerve block, or direct inoculation of bacteria in the abundance of loose connective and fatty tissue of the pterygopalatine fossa when performing a maxillary nerve block are possible. Infiltration of these last tissues with blood due to inadvertent puncture of blood vessels further encourages bacterial growth. Strict aseptic preparation of puncture sites and the use of appropriate techniques should be able to minimise the likelihood of infection accordingly (Staszek et al. 2008).

The fact that the prophylactic use of antibiotics does not necessarily prevent development and propagation of infection was observed in the case report by Vlaminck et al. (2001). This horse was premedicated 12 h before surgery with benzylpenicillin 12,000 IU/kg (Penikel) intramuscularly and metronidazole (Flagyl) intravenously (Vlaminck et al. 2001). This horse was premedicated 12 h before surgery with benzylpenicillin 12,000 IU/kg (Peni-kel) intramuscularly and metronidazole (Flagyl) intravenously (Vlaminck et al. 2001). The use of readily available diagnostic imaging techniques such as ultrasonography can be very efficient in identifying and characterising unusual complications following tooth extraction such as spreading of infection in the soft tissues surrounding the eye. Rapid CT scan and/or MRI examination of the head of this mare might have added further valuable information on the location and severity of the infection in relation to structures such as the eye to explain the loss of vision and possibly aid in further directing treatment choices.

It can be concluded that the possibility of development of potentially life-threatening infectious complications following tooth extraction in horses should be considered as extremely rare, although awareness of their occurrence is imperative to be able to take the right decisions regarding diagnostics and treatment options when confronted with them. They emphasise that tooth extraction in horses should not be taken for granted.

Author’s declaration of interests

No conflicts of interest have been declared.

Manufacturers’ addresses

1Kela, Hoogstraten, Belgium.
2Sanofi Belgium, Machelen, Belgium.
3Zoetis, Louvain-la-Neuve, Belgium.

References


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Case Report

Septic peritonitis in a Percheron mare associated with Clostridium haemolyticum


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Keywords: horse; tetrastarch; hypoalbuminaemia; hypoalbuminaemia; oncotic pressure; ileus; colic

Summary
This report describes an 11-year-old Percheron mare that presented with signs of colic and was ultimately diagnosed with bacterial peritonitis of unknown origin. Bacterial culture of a peritoneal fluid sample isolated 2 Clostridial species, one of which was strongly suspected to be Clostridium haemolyticum. The horse was markedly hypoalbuminaemic at presentation, leading to the development of low oncotic pressure and ventral oedema. The mare was administered a low molecular weight/low molar substitution hydroxyethyl starch solution in conjunction with other therapies that resulted in marked improvement of clinical signs. The purpose of this report is to describe the clinical findings associated with equine peritonitis associated with C. haemolyticum, a rarely identified pathogen in the horse. Secondly, this report serves to describe the beneficial effects of tetrastarch administration in a clinical case with severe hypoalbuminaemia and ventral oedema.

Introduction
Peritonitis in the horse is typically a severe disease, with varied mortality rates depending on the cause (Hawkins et al. 1993; Golland et al. 1994; Matthews et al. 2001; Henderson et al. 2008). Horses that develop peritonitis following a rupture of the intestinal tract or abdominal surgery generally have very low survival rates, whereas multiple reports of horses with peritonitis caused by Actinobacillus equuli infection indicated survival rates of 100% (Golland et al. 1994; Matthews et al. 2001). A variety of bacteria have been isolated from abdominal fluid samples from horses with peritonitis. Gram-negative species comprise the majority of reported cases, likely due to origination of the bacteria in the gastrointestinal tract (Hawkins et al. 1993; Golland et al. 1994; Matthews et al. 2001; Patterson-Kane et al. 2001). To the authors' knowledge, survival of a horse infected with C. haemolyticum has not been previously documented. This report describes the treatment of a Percheron mare with septic peritonitis from which C. haemolyticum was isolated and treatment included administration of a novel hydroxyethyl starch (HES) solution.

Case details
An 11-year-old Percheron mare, weighing 689 kg, was presented to the Lloyd Veterinary Medical Centre (LVMC) at Iowa State University with a 3-day history of progressive lethargy and signs of colic. The mare had reportedly had serous nasal discharge 2 weeks prior to presentation, but no fever was reported. No medications were administered by the referring veterinarian prior to presentation. The horse had no previous history of illness, although the boarding facility had reported numerous Streptococcus equi subsp. equi outbreaks in recent years. The horse was initially examined by the referring veterinarian who identified marked tachycardia (90 beats/min) and dehydration; abdominal palpation per rectum identified no abnormalities. Nasogastric intubation yielded approximately 20 l of net gastric reflux, after which the heart rate was reassessed and remained at 90 beats/min. The horse was referred for further evaluation and medical management.

Upon presentation to the LVMC, the horse was quiet, but alert and responsive. Marked tachycardia was still present at 96 beats/min and increased respiratory rate (44 breaths/min) and effort were identified. Mucous membranes were pink and tacky, with capillary refill time of 3 s. The horse was estimated to be 5–7% dehydrated based on tachycardia and tacky mucous membranes. Moderate ventral oedema was also present. Rectal temperature was mildly elevated (41.1°C), despite administration of flunixin meglumine, and borborygmi were decreased in all abdominal quadrants. A colic examination was performed including nasogastric intubation, abdominal ultrasound and abdominocentesis. No net gastric reflux was obtained. Abdominal ultrasound identified loops of hypomotile, dilated small intestine with thickened walls (up to 10 mm with normal thickness being ≤4 mm) and hyperechoic material suggestive of digesta present in the small intestine; a moderate amount of anechoic free peritoneal fluid was also identified (Figs 1 and 2). Abdominocentesis yielded a turbid fluid sample that was red-tinged in colour. Total solids of the fluid, analysed by refractometry, were 79 g/l (normal: <20 g/l). Lactate was markedly increased in the peritoneal fluid (13 mmol/l, normal: <2.0 mmol/l); unfortunately, systemic lactate was not assessed, but hypovolaemia and decreased systemic perfusion likely contributed to increased peritoneal lactate. A peritoneal fluid sample was saved for cytological evaluation and bacterial culture and antimicrobial sensitivity the following morning.

Initial laboratory analysis at presentation was performed including complete blood count, packed cell volume (PCV) and total protein. Haemoconcentration secondary to dehydration was identified from the increased PCV (48%; rr: 34–45%). Complete blood count identified an inflammatory leucogram characterised by a mature neutrophilia (7.9 x 10^9 cells/l; rr: 2.1–6.7 x 10^9 cells/l) with occasional toxic neutrophils along with rare reactive lymphocytes and a mild thrombocytopenia (115 x 10^9 cells/l; rr: 130–300 x 10^9 cells/l). At this stage, septic peritonitis was suspected as the top differential diagnosis, although the cause was unknown. The
ILEUS was suspected to either be secondary to peritonitis, or due to concurrent duodenitis proximal jejunitis. The peritoneal fluid sample submitted for routine culture was set-up under aerobic and anaerobic conditions. No aerobic bacterial organisms were identified. Two strict anaerobic organisms were identified following 48 h of incubation. Both these organisms were identified as large Gram-positive rods consistent with Clostridium species. An aerotolerance test was done to confirm both organisms as strict anaerobes. A Bruker MALDI biotyper was utilised to attempt further speciation of the Clostridium species. One of the organisms did not have a reliable identification and the other was identified as Clostridium haemolyticum with a MALDI score of 1.766.

Treatment
Initial therapy included fluid resuscitation with i.v. bolus administration of 12 l lactated Ringer’s solution followed by a continuous infusion of 3-4 l/h. Intermittent gastric decompression was performed with a total of 19 l of net reflux recovered overnight. Flunixin meglumine (Prevail 1, 1.1 mg/kg bwt i.v. q. 12 h) was administered and broad spectrum antimicrobial therapy was instituted with procaine penicillin G (Procaine penicillin G 2, 22,000 iu/kg bwt i.m. q. 12 h) and gentamicin (Gentamicin 1, 6.6 mg/kg bwt i.v. q. 24 h).

The following morning the mare remained tachycardic (90 beats/min) and mucous membranes appeared congested. Cytological analysis of the peritoneal fluid identified 30.7 × 10⁹ cells/l, rr <5.0 × 10⁹ cells/l, of which 92% were neutrophils and 8% large mononuclear cells. Neutrophils were degenerate and numerous cells contained intracellular bacteria (small and large rods, small paired cocci) (Fig 3). Based on the elevated total nucleated cell count demonstrated within the abdominal fluid indicating septic peritonitis and the history of S. equi infection on the premises, a serum titre for S. equi M protein was analysed to rule out disseminated S. equi abscess; the result of this titre was moderately positive at 1:800, but not high enough to support a diagnosis of metastatic abscessation. Peritoneal fluid was also submitted for bacterial culture and antimicrobial susceptibility which yielded C. haemolyticum and an additional unspeciated Clostridial isolate. A 32 French thoracic trocar (Thoracic trocar catheter)³ was placed with a Heimlich valve to the right of ventral midline to facilitate lavage and continuous drainage of the abdominal effusion. Prior to lavage, 30 l of turbid red-tinged fluid was drained from the abdomen and 12 l lactated Ringer’s solution were infused and allowed to drain; this lavage was repeated twice daily until Day 4 of hospitalisation.

Complete blood count was repeated on Day 2 which identified a decreased WBC (5.84 × 10⁹ cells/l; rr: 5.0–11.0 × 10⁹ cells/l) and neutrophil count (2.51 × 10⁹ cells/l), presence of band neutrophils and persistent presence of Döhle bodies and basophilia within the neutrophils. Reactive lymphocytes were still present and serum biochemical analysis identified mild electrolyte derangements and mild azotaemia. Marked hypoalbuminaemia (18 g/l; rr: 33–46 g/l) and severely decreased oncotic pressure (COP) were also present (Fig 4).

Tachycardia persisted despite aggressive crystalloid therapy and peritoneal drainage. Due to concern for worsening hypoalbuminaemia and poor perfusion, colloid administration was pursued on the
afternoon of Day 2. A low molecular weight tetrastarch (TES) solution (Vetstarch) was administered as a 20 ml/kg bolus over 2 h. To document the duration of the effects of the colloid, COP was assessed at the completion of the bolus (0 h), then at 30 min, 1, 2, 3, 5, 12, 24 and 36 and 48 h (Fig 4). Albumin was re-evaluated at 24, 36 and 48 h after the TES infusion.

**Outcome**

Within 1 h of administration of the colloid, the mare’s heart rate decreased to 56 beats/min and urination was observed. The mare appeared brighter and displayed a strong appetite when feed was reintroduced, consisting of small handfuls of hay and pelleted feed every 3 h. Abdominal ultrasound the next day revealed normal thickness of the small intestinal walls (3 mm). Over the next week, the mare continued to improve. Vital parameters stabilised into a normal range by Day 3 and remained fairly constant until discharge on Day 7. The abdomen was lavaged twice daily until Day 4 at which point the gross appearance of the efflux fluid from the lavage was clear and the drain removed. Culture results were available on Day 6 and the antimicrobial therapy was changed from penicillin and gentamycin to metronidazole (Metronidazole, 15 mg/kg bwt q. 8 h for the next 4 weeks (q. 12 h) was prescribed; instructions for the owner to administer metronidazole for the next 4 weeks (q. 8 h for the next 2 weeks). Abdominocentesis was performed which identified a lymphocytosis, hyperproteinemia, mild anaemia and mild thrombocytopenia. Fibrinogen and neutrophil counts were within normal limits. Albumin and COP at this time were 0.28 g/l and 21.7 mmHg, respectively. Abdominocentesis was performed which identified a reduced cell count from the initial visit at 12.7 x 10^6 cells/l and protein of 68 g/l. An additional 3 weeks of metronidazole (15 mg/kg bwt per os q. 12 h) was prescribed; communication with the owner 2 weeks after cessation of antibiotics indicated that the mare had continued to gain weight and was bright and back in work.

**Discussion**

While numerous bacterial species including Clostridial agents have been isolated from peritoneal fluid of horses with peritonitis, to the authors’ knowledge this is the first documented case wherein _C. haemolyticum_ was isolated as the primary pathogen ante-mortem in equine peritonitis. Peritonitis in adult horses may arise secondary to intra-abdominal surgery, metastatic abscessation, neoplasia, trauma, parasite migration, abdominocentesis, complications of parturition, introduction of a foreign body into the abdomen, haematogenous spread of bacteria, or perforation of the gastrointestinal tract (Gay et al. 1980; Hawkins et al. 1993; Davis 2003). Despite these more obvious presentations, many cases of septic peritonitis are of unknown origin, as in this report (Hawkins et al. 1993; Elce 2006). The rare reports of equine disease associated with _C. haemolyticum_ and its counterpart _C. novyi_ type B have identified a 100% mortality rate, making notable the survival of the case described in this report (Dumaresq 1939; Hollingsworth 1978; Gay et al. 1980; Oaks et al. 1997).

Despite the MALDI score being lower than the recommended score of 2.0 for accurate speciation of bacterial isolates, several papers have found that a score of >1.7 was sufficient for speciation (Chekaoui et al. 2010; Fedorko et al. 2012; Fournier et al. 2012). Additionally, Chean et al. (2014) found that the MALDI was 100% accurate in identification of _C. botulinum_ spp. isolates consisting of 10 different _Clostridium_ species. The results of that study indicated that a MALDI score between 1.7 and 2.0 correctly speciated _Clostridium_ spp. isolates in greater than 88% of the cases (Chean et al. 2014). Further 16S rRNA sequencing could not be done in this case to verify the species as the isolates did not survive additional subculturing.

Clostridial infections in the horse are typically associated with gastrointestinal disease ( _C. difficile_, _C. perfringens_) (Baverud 2004; Diab et al. 2012), hepatic disease ( _C. piliforme_) (Borchers et al. 2006), neuromuscular disease ( _C. botulinum_, _C. tetani_) (Smith and George 2009; Johnson et al. 2015), or myositis (most commonly _C. perfringens_ and _C. sordelli_) (Adam and Southwood 2006). The causative agents of bacillary haemoglobinuria ( _C. novyi_ type D, also known as _C. haemolyticum_) and black disease ( _C. novyi_ type B) are well described pathogens in ruminants, but are seldom identified in the horse (Smith 2015). The pathogenesis of _C. novyi_ infection is initiated when spores ingested from the soil cross the intestinal mucosa and are transported to the liver. Disease is often associated with liver fluke infections in ruminants that lead to anaerobic environments within the liver and allow for germination and proliferation of the clostridial spores (_C. difficile_, _C. perfringens_) (Baverud 2004; Diab et al. 2012), hepatic disease ( _C. piliforme_) (Borchers et al. 2006), neuromuscular disease ( _C. botulinum_, _C. tetani_) (Smith and George 2009; Johnson et al. 2015), or myositis (most commonly _C. perfringens_ and _C. sordelli_) (Adam and Southwood 2006). The causative agents of bacillary haemoglobinuria ( _C. novyi_ type D, also known as _C. haemolyticum_) and black disease ( _C. novyi_ type B) are well described pathogens in ruminants, but are seldom identified in the horse (Smith 2015). The pathogenesis of _C. novyi_ infection is initiated when spores ingested from the soil cross the intestinal mucosa and are transported to the liver. Disease is often associated with liver fluke infections in ruminants that lead to anaerobic environments within the liver and allow for germination and proliferation of the clostridial spores (_C. difficile_, _C. perfringens_) (Baverud 2004; Diab et al. 2012), hepatic disease ( _C. piliforme_) (Borchers et al. 2006), neuromuscular disease ( _C. botulinum_, _C. tetani_) (Smith and George 2009; Johnson et al. 2015), or myositis (most commonly _C. perfringens_ and _C. sordelli_) (Adam and Southwood 2006). The causative agents of bacillary haemoglobinuria ( _C. novyi_ type D, also known as _C. haemolyticum_) and black disease ( _C. novyi_ type B) are well described pathogens in ruminants, but are seldom identified in the horse (Smith 2015). The pathogenesis of _C. novyi_ infection is initiated when spores ingested from the soil cross the intestinal mucosa and are transported to the liver. Disease is often associated with liver fluke infections in ruminants that lead to anaerobic environments within the liver and allow for germination and proliferation of the clostridial spores (_C. difficile_, _C. perfringens_) (Baverud 2004; Diab et al. 2012), hepatic disease ( _C. piliforme_) (Borchers et al. 2006), neuromuscular disease ( _C. botulinum_, _C. tetani_) (Smith and George 2009; Johnson et al. 2015), or myositis (most commonly _C. perfringens_ and _C. sordelli_) (Adam and Southwood 2006). The causative agents of bacillary haemoglobinuria ( _C. novyi_ type D, also known as _C. haemolyticum_) and black disease ( _C. novyi_ type B) are well described pathogens in ruminants, but are seldom identified in the horse (Smith 2015). The pathogenesis of _C. novyi_ infection is initiated when spores ingested from the soil cross the intestinal mucosa and are transported to the liver. Disease is often associated with liver fluke infections in ruminants that lead to anaerobic environments within the liver and allow for germination and proliferation of the clostridial spores (_C. difficile_, _C. perfringens_) (Baverud 2004; Diab et al. 2012), hepatic disease ( _C. piliforme_) (Borchers et al. 2006), neuromuscular disease ( _C. botulinum_, _C. tetani_) (Smith and George 2009; Johnson et al. 2015), or myositis (most commonly _C. perfringens_ and _C. sordelli_) (Adam and Southwood 2006). The causative agents of bacillary haemoglobinuria ( _C. novyi_ type D, also known as _C. haemolyticum_) and black disease ( _C. novyi_ type B) are well described pathogens in ruminants, but are seldom identified in the horse (Smith 2015).
bacillary haemaglobinuria have rarely been reported in the horse and of those documented cases all have died or been subjected to euthanasia as a result of the severity of the disease (Dumaresq 1939; Gay et al. 1980; Oaks et al. 1997; Davis 2003; Smith and George 2009). Interestingly, many of the clinical signs and clinicopathological changes associated with bacillary haemaglobinuria in ruminants were not observed in the case described in this report. Significant alterations in the erythron were not identified, nor were obvious coagulopathies or changes in the colour or character of the urine; the most significant clinicopathological changes identified in this case were consistent with endotoxaemia and third space loss of albumin. Additionally, there were no major alterations in the hepatic enzymes on the biochemical profile, consistent with the findings of the aforementioned case of black disease (Gay et al. 1980).

The origin of a Clostridial agent in the abdominal cavity was suspected to be either gastrointestinal or a result of hepatic parasite migration, although no definitive origin was identified. Two of the 5 previously reported cases of equine infection with C. novyi had hepatic lesions consistent with parasite migration evident at necropsy (Dumaresq 1939; Hollingsworth 1978); of these 5, 3 had evidence of peritonitis as in this case (Dumaresq 1939; Hollingsworth 1978; Gay et al. 1980; Oaks et al. 1997). While it can be speculated that parasite migration through the liver and subsequent hepatic infection with C. novyi may have been the origin of peritonitis in the case reported here, the deworming history of the horse was unknown. Lack of significant clinical findings on ultrasonographic imaging of the liver and evaluation of hepatic enzymes did not justify hepatic biopsy to identify a potential source for the peritonitis.

Ileus is a commonly reported occurrence in horses with septic peritonitis as a result of mechanical irritation of the intestinal serosa. Diffuse irritation and inflammation of the gastrointestinal serosa can activate spinal sympathetic inhibitory reflexes and lead to ileus (Lomax et al. 2010). Ileus is also thought to develop in peritonitis as a protective mechanism against spread of infection via peristalsis (Ragetly et al. 2012). Initially, this horse was suspected of having duodenitis proximal jejunitis in conjunction with septic peritonitis. While this remains possible, the short duration of ileus and rapid resolution of oedema within the intestinal walls made the peritonitis a more probable cause for ileus.

Medical management of peritonitis generally involves abdominal lavage and drainage, antimicrobial therapy, cardiovascular support and control of endotoxaemia and pain (Davis 2003; Dart and Bischofberger 2011). While the inciting cause (i.e. surgery, metastatic abscess, idiopathic) can dictate some aspects of treatment, these standards are generally approached regardless of cause. Interestingly, in the 3 previously reported cases of peritonitis resulting from C. novyi infection, the horse declined rapidly and either died or was subjected to euthanasia despite attempts at treatment (Dumaresq 1939; Hollingsworth 1978; Gay et al. 1980; Oaks et al. 1997). In this particular case, the positive outcome may have stemmed from earlier detection of clinical signs and more aggressive supportive therapy in the early stages of disease. Granted, with many years between these cases, it is likely that advancement in veterinary medicine as a field contributed to the survival of the case described here. Aggressive colloidal support was necessary to restore appropriate cardiovascular function and decrease oedema formation, in addition to antimicrobial therapy, use of nonsteroidal anti-inflammatory drugs and drainage and lavage of the peritoneal cavity. Additionally, the case reported here was displayed comparatively mild signs when compared with those previously reported in the literature.

The use of synthetic colloids in hospitalised horses is a common practice; however, reports utilising the newest hydroxethyl starch (HES) solution in clinical cases have not yet been published. Tetrastarch (TES, Vetstarch) recently became available on the veterinary market and has been shown to have fewer side effects than higher molecular weight hydroxethyl starch products in man and animals (Langeron et al. 2001; Boldt 2009; Ertmer et al. 2010; Mizzi et al. 2011). The effect of a synthetic hydroxethyl starch solution on COP is determined by the number of oncotically active particles present in the solution. Therefore, solutions with a lower mean molecular weight have a larger number of smaller colloid molecules, thus exerting more oncotic pressure in the vasculature (Langeron et al. 2001). The rate at which HES molecules are degraded is determined by the molar substitution ratio, which describes the number of hydroxyl molecules substituted with a hydroxyethyl molecule and thus made resistant to degradation by amylase (Boldt 2009). Solutions such as TES, that have a lower mean molecular weight (130 kDa) and molar substitution ratio (0.4) than other HES products such as hetastarch (mean molecular weight 670 kDa, molar substitution ratio 0.7), have been purported to have a greater effect on COP, but fewer side effects such as tissue accumulation, effects on coagulation and acute kidney injury (Langeron et al. 2001; Boldt 2009).

Following administration of the bolus of TES in this report, the horse’s COP remained increased above baseline for the next 48 h, despite minimal increases in serum albumin. Previous reports have documented different duration of effect of TES on COP in healthy animals, ranging from 6 to 48 h. The long lasting effect on COP in the case described in this report suggests that critically ill animals may have reduced clearance of synthetic colloids (Epstein et al. 2014; Vlijgen et al. 2014). The rapid improvement in clinical signs (heart rate, demeanour) immediately following administration of TES to this horse suggested that colloidal support was an important component of therapy in this case and contributed significantly to the positive outcome; however, in light of other treatments administered concurrently, this can not be definitively stated.

The most frequently reported adverse effects of synthetic colloid use in man include acute kidney injury and induction of coagulopathies, although reports of adverse effects in veterinary species are rare (Langeron et al. 2001; Kozek-Langenecker 2005). Studies in horses have identified minor alterations in platelet function and coagulation, but have been unable to identify significant effects on renal function (Jones et al. 1997; Schusser et al. 2007; Blong et al. 2013; Epstein et al. 2014; Vlijgen et al. 2014; Hepworth-Warren et al. 2015). Although induction of coagulopathy was a concern in this case, the administration of TES was pursued due to the severity of hypoalbuminaemia and evidence of hypovolaemia that persisted despite fluid resuscitation with crystalloids. Administration of equine plasma may have been a safer therapeutic option, but was cost prohibitive due to the large size of the horse. A volume of equine plasma at the current cost at LVMC equivalent to the volume of TES administered in this case would have cost greater than $2500 (£1649) for a single dose, nearly triple the cost of TES.
Additionally, TES is hyperoncotic (COP 36 mmHg) compared with equine plasma (ir: 18–25 mmHg), making it even more cost effective (Mitra and Khandelwal 2009). As recent work has suggested that TES is cleared more rapidly from circulation and has fewer adverse effects, this was selected over the more traditionally used hetastarch (Boldt 2009; Epstein et al. 2014). No overt clinical signs of a coagulopathy were observed in the case described here, but a limitation was the absence of monitoring in regards to clotting times. However, recent studies have shown that the main alterations induced by HES solutions involve platelet function, analysis of which was not available (Blong et al. 2013).

This report serves to document the survival of a horse with peritonitis most likely caused by C. haemolyticum, but also documents the clinical response of an adult horse to administration of a low molecular weight/low molar substitution hydroxyethyl starch solution in a horse with severe hypoalbuminaemia and accompanying peripheral oedema. Despite the controversy that surrounds the utilisation of synthetic colloids as a resuscitative solution, in the case described here, tetrastarch provided a more economically feasible option than equine plasma and significant clinical improvement was noted directly following administration. While additional therapies were administered over the course of the mare’s hospitalisation that undoubtedly contributed to the positive outcome, the temporal association between administration of tetrastarch and improvement of clinical signs (decreased heart rate, urination) suggest that these changes were at least in part due to the administration of the colloid. Due to the clinical nature of this case, the sole effect of TES administration could not be assessed without risking the horse’s wellbeing, but this case suggests that studies are warranted to investigate the effect of TES administration in hypoproteinaemic animals with peripheral oedema.

Furthermore, the effects of tetrastarch administration on endotoxaemia remain unknown, thus the authors suggest caution in the use of potentially caused by synthetic colloids.

Authors’ declaration of interests

No conflicts of interest have been declared.

Ethical animal research

This manuscript is a case report describing the management of a case admitted to the Lloyd Veterinary Medical Centre. The client consented to additional blood sampling for COP monitoring following administration of tetrastarch. Anonymity was maintained throughout the manuscript so as not to identify the owner or the horse.

Source of funding

Funding provided through authors research accounts.

Authorship

K.L. Hepworth-Warren was involved in case management, data collection and interpretation and preparation of the manuscript. B.L. Hay Kraus was involved in data interpretation and preparation of the manuscript. D.M. Wong was involved in data interpretation and preparation of the manuscript. A.C. Knoll was involved in bacteriological identification and manuscript preparation. G.L. Metcalf was involved in case management. All authors gave their final approval of the manuscript.

Manufacturers’ addresses

1Vetone, Nova-Tech, Inc, Grand Island, Nebraska, USA.
2Vetone, Norbrook Laboratories, Newry, Northern Ireland, UK.
3Teleflex Medical, Athlone, Ireland.
4Abbott Animal Health, Chicago, Illinois, USA.
5Teva Pharmaceuticals USA, Sellersville, Pennsylvania, USA.

References


Continued from page 587


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*Only provide to foals 4-6 hours after foal first nurses the mare. **Weese J, Cote N, deGannes R. Evaluation of in vitro properties of di-tri-octahedra smectite on clostridial toxins and growth. Equine Vet J 2003;35:638-641
Case Report

Balloon dilation to treat oesophageal strictures in five foals

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Keywords: horse; endoscopy; oesophageal dilatation; intralesional corticosteroid therapy

Summary

Oesophageal stricture is a rare disorder in foals. The following manuscript describes the successful treatment of oesophageal strictures with balloon dilation in 5 foals aged 3 weeks to 4 months. Three of the foals presented due to recurrent oesophageal obstruction, one due to dysphagia, and one due to weight loss, pytalism and bruxism. The strictures were diagnosed via endoscopy. In 2 cases, the use of intraoesophageal corticosteroid injection was performed to reduce the incidence of oesophageal stricture reoccurrence. In all cases, a satisfactory outcome with resolution of clinical signs was achieved.

Introduction

Oesophageal disorders in foals are uncommon in comparison to other diseases of the gastrointestinal tract, and include impaction with a foreign body or food, perforations, diverticula and strictures (Craig and Todhunter 1987). Oesophageal strictures may be congenital or acquired (Tillotson et al. 2003). Congenital strictures will normally become apparent by the time a foal begins to ingest solid food, and may present as milk regurgitation or as an initial episode of oesophageal obstruction (Knottenbelt et al. 1992; Bezdekova et al. 2015). Foals may also develop acquired strictures, with gastric ulceration and reflux oesophagitis thought to be associated in many cases in neonatal foals (Murray et al. 1988). Other aetologies of acquired oesophageal strictures are similar to mature horses and include previous oesophageal obstruction with secondary mucosal ulceration, and nasogastric tube trauma (Craig and Todhunter 1987; Stick 2012). This manuscript will describe the use of endoscopic balloon dilation to treat oesophageal strictures in 5 foals.

Case report

Foal A

History

A 3-month-old Thoroughbred filly presented to Hagyard Equine Medical Institute for evaluation of weight loss, pytalism and bruxism of 5 days’ duration. The foal had been receiving antimicrobial treatment for suspected Rhodococcus equi pneumonia for several weeks prior to admission.

Clinical findings

On presentation (Day 1), the foal was quiet and alert, with a body condition score of 4/9 (Henneke et al. 1983). Continuous bruxism and pytalism were noted. Vital parameters were within normal limits (heart rate 44 beats/min, respiratory rate 24 breaths/min and temperature 38.2°C). The remainder of the physical examination was unremarkable.

Fig 1

Diagnostic procedures

Oesophagoscopy was performed using a 2 m long, 11 mm diameter video enteroscope. Approximately 125 cm distal to the nares, passage of the endoscope was impeded by a circumferential narrowing of the oesophagus that prevented the endoscope entering the stomach. Contrast radiography of the oesophagus using barium sulfate paste was performed. This revealed distinct narrowing of the oesophagus at the level of the cardia, with no apparent involvement of the muscularis. Based on history and clinical findings, a presumptive diagnosis of mucosal oesophageal stricture was made. A tracheal wash was also performed due to the foal’s history of pneumonia; culture of the aspirate yielded growth of Escherichia coli and Staphylococcus aureus.

Treatment and outcome

Balloon dilation of the stricture was performed under standing sedation with romifidine (Sedivet; 0.08 mg/kg bwt i.v.) and butorphanol (Torbugesic; 0.02 mg/kg bwt i.v.). The endoscope was passed into the oesophagus until immediately proximal to the stricture; a wire-guided, Controlled Radial Expansion (CRE) balloon catheter was advanced through the biopsy channel of the endoscope to the stricture site. The balloon was then inflated to its maximal diameter using an inflation system and maintained at 6 atmospheres for 60 s to dilate the stricture. This was repeated 3 times until excoriation of the mucosa was present, following which the balloon was retracted. Following dilation, the endoscope could be easily advanced into the stomach where extensive grade 3 ulceration was present. Antimicrobial therapy with ceftiofur (Naxcel; 3 mg/kg bwt i.v. q. 24 h) and amikacin (Amikyclate-V; 25 mg/kg bwt i.v. q. 24 h for 10 days) was initiated based on culture and sensitivity results of the tracheal wash sample.

Following balloon dilation, the foal was muzzled and allowed to nurse every 2 h for progressively longer intervals over the first 48 h. Following this, the foal was allowed access to ad libitum nursing and pasture turnout. Medical therapy consisted of ranitidine (6.6 mg/kg bwt per os q. 8 h), omeprazole (Gastrogard; 2 mg/kg bwt per os q. 24 h) and sucralfate (20 mg/kg bwt per os q. 6 h) for 14 days. Following balloon dilation, the foal could be slowly introduced back to hay, following which she could be slowly introduced back to hay.
No further episodes of oesophageal obstruction occurred. Three years after balloon dilation was performed, the filly is in race training and has raced twice.

**Foal B**

**History**

A 4-month-old Thoroughbred colt foal presented with a history of recurrent oesophageal obstruction of 2 weeks’ duration, the most recent episode occurring immediately prior to admission. Treatment with gentamicin sulfate (6.6 mg/kg bwt i.v. q. 24 h), sulfamethoxazole and trimethoprim (30 mg/kg bwt per os q. 12 h), omeprazole (Gastrogard; 2 mg/kg bwt per os q. 24 h), sucralfate (20 mg/kg bwt per os q. 6 h) and flunixin meglumine (Banamine; 1.1 mg/kg bwt per os q. 24 h) had been initiated by the referring veterinarian 3 days prior to referral.

**Clinical findings**

On admission (Day 1), the foal was bright and alert, with normal vital parameters (heart rate 56 beats/min; respiratory rate 24 breaths/min; temperature 38.4°C). A bilateral mucoid nasal discharge was present. The remainder of the physical examination was unremarkable.

**Diagnostic procedures**

Endoscopy of the oesophagus using a 2 m long, 11 mm diameter enteroscope noted a circumferential narrowing within the thoracic portion of the oesophagus, 75 cm distal to the nares. The oesophagus appeared dilated proximal to the stricture when assessed by endoscopy, but no ulceration of the oesophagus or stomach was noted. A diagnosis of oesophageal stricture and secondary megaoesophagus was made. No abnormalities were detected on thoracic ultrasonography.

**Treatment and outcome**

Oesophageal dilation was performed under standing sedation with xylazine hydrochloride (Anased; 1 mg/kg bwt i.v.) and butorphanol tartrate (Torbugesic; 0.03 mg/kg bwt i.v.), using the same technique as described for Foal A. The CRE wire-guided balloon catheter was inflated once to 6 atmospheres (20 mm) for 60 s. Repeat dilation was not performed since excoriation of the oesophageal mucosa was present following the first dilation (Fig 3). On Day 2, repeat oesophagoscopy demonstrated marked improvement in the diameter of the stricture; therefore the foal was discharged with instructions to be maintained on a diet of grazing and nursing only. Although there was no evidence of pneumonia on clinical or ultrasonographic examination, due to the high risk of aspiration pneumonia, treatment with gentamicin sulfate (6.6 mg/kg bwt i.v. q. 24 h for 5 days) and sulfamethoxazole and trimethoprim (30 mg/kg bwt per os q. 12 h for 7 days) was continued. Omeprazole (Gastrogard; 2 mg/kg bwt per os q. 24 h for 7 days), sucralfate (20 mg/kg bwt per os q. 6 h for 7 days) and flunixin meglumine (Banamine; 1.1 mg/kg bwt per os q. 24 h for 3 days) were also continued.

Following discharge, weekly endoscopic evaluations were performed to assess the stricture. Although the stricture diameter was noted to have improved compared to presentation, the oesophagus at the stricture site was still only approximately 50% of normal oesophageal diameter. The megaoesophagus was still visible at each endoscopic assessment. Balloon dilation was repeated 3 times over the
following 4 weeks. The largest inflation diameter achievable with the CRE balloon catheter was 20 mm, therefore a 15 mm diameter nasogastric tube was inserted into the oesophagus alongside the balloon catheter to produce wider dilation. The balloon catheter was then inflated to 6 atmospheres (20 mm) for 3 repetitions of 60 s, until excoriation of the oesophageal mucosa was present. At the third balloon dilation procedure (fourth in total), the decision was made to use intraoesophageal corticosteroid therapy due to the continual recurrence of the stricture. Following balloon dilation, a 25-gauge sclerotherapy injection catheter, with 5 mm needle-projection, was preloaded with 6 mg (1 ml) of triamcinolone acetonide (Vetalog)\(^3\). The catheter was introduced through the endoscope biopsy channel and advanced in small increments until it emerged from the endoscope at the level of the stricture site. The needle was extended and directed at an oblique angle into the oesophageal mucosa at the level of the stricture. Triamcinolone acetonide was injected in a 4-quadrant approach, with 0.25 ml (1.5 mg) aliquots injected at 12, 3, 6 and 9 o’clock positions. Five weeks after initial presentation, the foal suffered an acute episode of oesophageal obstruction having accidentally been fed a dry pelleted feed. Subsequently, the foal developed bronchopneumonia presumed secondary to aspiration of feed particles. Treatment was initiated with an extended-release formula of cefitiofur crystalline, (Excede\(^4\); 6.6 mg/kg bwt i.m., 2 doses 4 days apart). Three weeks later, endoscopy was repeated and revealed that the stricture was now approximately 75% of normal oesophageal diameter; the dilation of the oesophagus proximal to the stricture was unchanged. Oesophageal dilation was repeated using a 35 mm RigiFlex Achalasia balloon dilator\(^5\). This was inserted alongside the endoscope to lie beside a 15 mm diameter nasogastric tube, which gave a dilation diameter of 50 mm. Intraoesophageal corticosteroid therapy was repeated at this time as previously described. Dilation with the RigiFlex Achalasia balloon alongside a 15 mm nasogastric tube was repeated 3 further times over the next 2 weeks. By 11 weeks after presentation, the oesophageal stricture was dilated to 85% of normal oesophageal diameter. One week later, the foal’s diet was adjusted to include hay alongside grazing and nursing. Follow-up evaluations demonstrated that the foal was gaining weight appropriately and was able to graze and eat hay without oesophageal obstruction. The animal was re-examined as a yearling at which time he was of appropriate size and weight for his age group, and no issues with recurrent oesophageal obstruction were reported. Endoscopic evaluation revealed that the stricture and proximal megaesophagus were unchanged from the previous examination. On long-term follow-up 3 years later, the colt is healthy and in race training and has raced 4 times.

**Foal C**

**History**

A 3-week-old Thoroughbred colt foal presented with a history of recurrent oesophageal obstruction over the previous 10 days.

**Clinical findings**

On admission, the foal was bright and alert. Heart rate (80 beats/min) and temperature (38.6°C) were within normal limits, but respiratory rate was increased (68 breaths/min).

**Diagnostic procedures**

Endoscopy of the oesophagus was performed under standing sedation with diazepam\(^1\) (0.15 mg/kg bwt i.v.), xylazine hydrochloride (Anased\(^1\); 1 mg/kg bwt i.v.) and butorphanol tartrate (Torbugesic\(^6\); 0.03 mg/kg bwt i.v.). A 1 m long, 9 mm diameter video endoscope\(^7\) was passed into the oesophagus and an oesophageal stricture was identified 60 cm caudal to the nares. The lumen of the oesophagus at the stricture site was approximately 5 mm in diameter, preventing passage of the endoscope into the stomach. Based on the narrow diameter of the oesophagus at the stricture site and the endoscopic appearance, the stricture was considered to be mucosal. On thoracic ultrasound examination, comet tail artefacts were visible radiating from the pleural surface of the lung bilaterally, consistent with mild bronchopneumonia presumed to have developed secondary to aspiration.

**Treatment and outcome**

Balloon dilation using a CRE balloon catheter\(^1\) was performed as described for Foal A. The balloon was inflated once to 6 atmospheres (20 mm) for 60 s, following which the oesophagus was completely dilated with excoriation of the oesophageal mucosa, and it was possible to advance the endoscope into the stomach. No gastric ulceration or other abnormalities of the oesophagus were noted. Treatment was initiated with cefitiofur (Naxcel\(^8\); 3 mg/kg bwt i.v. q. 12 h for 5 days) due to the presence of bronchopneumonia, along with sucralfate\(^9\) (20 mg/kg bwt per os q. 6 h for 7 days), and flunixin meglumine (Banamine\(^10\); 1.1 mg/kg bwt i.v. q. 12 h for 2 days). Following the balloon dilation procedure the foal was muzzled and allowed to nurse until the mare’s udder was empty, every 2 h for 24 h.

The foal was discharged from the hospital the day after the balloon dilation procedure, with instructions to be maintained on a diet of nursing and grazing only. Repeat endoscopy at 3 and 11 days post dilation revealed normal dilation of the oesophagus with no stricture visible. The animal was re-examined as a yearling. No further episodes of oesophageal obstruction had been reported and there was no evidence of oesophageal stricture on endoscopy. Four years after balloon dilation the colt is currently in race training; he has won or been placed in 14 of his 35 starts.

**Foal D**

**History**

A 10-week-old Standardbred colt foal presented for investigation of repeated episodes of oesophageal obstruction over the previous 2 weeks.

**Clinical findings**

On admission, the foal was depressed with ptyalism noted. Physical examination was otherwise unremarkable with vital parameters within normal limits (heart rate 60 beats/min, respiratory rate 20 breaths/min, temperature 38.4°C).

**Diagnostic procedures**

A 2 m long, 11 mm diameter enteroscope\(^1\) was advanced into the oesophagus. A distinct narrowing of the oesophagus was present 50 cm distal to the nares, which appeared to involve the mucosa only. A diagnosis of oesophageal stricture was made. The oesophageal diameter was estimated to be 5 mm at the stricture site, preventing advancement of the
endoscope into the stomach. Thoracic ultrasound examination revealed evidence of bronchopneumonia as evidenced by the presence of comet tail artefacts radiating from the pleural surface of the cranioventral lung fields bilaterally.

**Treatment and outcome**

Balloon dilation of the oesophageal stricture was carried out under standing sedation using xylazine hydrochloride (Anased\(^{11}\); 1 mg/kg bwt i.v.) and butorphanol tartrate (Torbugesic\(^{4}\); 0.03 mg/kg bwt i.v.). The balloon catheter was inflated to 6 atmospheres (20 mm) for 60 s for 3 repetitions, until excoration of the mucosa was present. Repeat endoscopy 24 h later (Day 2) revealed that the diameter of the oesophageal lumen at the level of the stricture was now approximately 15 mm diameter. The balloon catheter was inserted alongside a 15 mm diameter nasogastric stomach tube, and inflated to 6 atmospheres (20 mm) for 3 repetitions of 60 s, giving a maximal dilation of 35 mm. Treatment was initiated with sucralfate\(^{8}\) (20 mg/kg bwt per os q. 6 h for 7 days), ceftriaxone (Naxcel\(^{5}\); 3 mg/kg bwt i.v. q. 12 h for 5 days) and fluimixin meglumine (Banamine\(^{10}\); 1.1 mg/kg bwt i.v. q. 24 h for 2 days).

The foal was discharged 3 days after the initial balloon dilation procedure, with instructions for the foal to be allowed to graze and nurse ad libitum but receive no other feeding. Re-evaluation and repeat endoscopy were performed at 12 and 27 days post initial balloon dilation. Dilation was repeated at each examination using the same technique as Day 2. On Day 27, introesophageal corticosteroid injection was performed after dilation using the same technique as described for Foal A. Repeat endoscopy was performed on Day 53 after the initial balloon dilation procedure; the stricture site was fully dilated at this time and no issues with recurrent oesophageal obstruction were reported. The animal is now a 2-year-old and in race training.

**Foal E**

**History**

A one-month-old Thoroughbred filly foal presented for evaluation of dysphagia and fever. The foal had a prolonged history of autoimmune disorders (neonatal isoerythrolysis; immune-mediated neutropenia, thrombocytopenia and dermatitis) as a neonate, which required hospitalisation in an intensive care unit and treatment including antimicrobials and corticosteroids.

**Clinical findings**

On the day of admission the foal had become pyrexic and dysphagic. At presentation (Day 1), the foal was bright and alert but ptyalism was present. Heart rate (64 beats/min) was within normal limits, but respiratory rate (44 breaths/min) and temperature (39.4°C) were increased. Thoracic auscultation revealed increased bronchovesicular lung sounds bilaterally. Subsequent thoracic ultrasonography revealed comet tail artefacts radiating from the pleural surface of the lungs bilaterally, consistent with bronchopneumonia. Treatment was initiated with potassium G penicillin\(^{14}\) (25,000 iu/kg bwt i.v. q. 6 h), gentamicin sulfate\(^{9}\) (6.6 mg/kg bwt i.v. q. 24 h), fluimixin meglumine (Banamine\(^{10}\); 1.1 mg/kg bwt i.v. q.12 h) and sucralfate\(^{8}\) (20 mg/kg bwt per os q. 6 h) and progressive improvement in the foal's condition and ultrasonographic findings were observed. On Day 5, the foal was noted to be coughing and hypersalivating with a bilateral serous nasal discharge, and a firm mass could be palpated in the oesophagus immediately distal to the larynx, indicative of an oesophageal obstruction. N-butyłscopolamine (Buscopan\(^{5}\); 0.1 mg/kg bwt i.v.) was administered and the oesophageal obstruction resolved without need for further intervention.

**Diagnostic procedures**

Endoscopy was performed following resolution of the oesophageal obstruction using a 2 m long, 11 mm diameter endoscope\(^{7}\). This revealed an oesophageal stricture 60 cm distal to the nares with marked narrowing of the lumen preventing passage of the endoscope past the stricture. Subjectively, the stricture appeared to involve the mucosal layer only.

**Treatment and outcome**

Balloon dilation of the oesophagus was performed under standing sedation with xylazine (Anased\(^{11}\); 1 mg/kg bwt i.v.) and butorphanol (Torbugesic\(^{4}\); 0.03 mg/kg bwt i.v.)., using the technique described for Foal A. The CRE wire-guided balloon catheter\(^{5}\) was inflated to 6 atmospheres (20 mm) 3 times for 60 s each time. Following this, it was possible to advance the endoscope past the stricture site into the stomach, which revealed no evidence of mucosal ulceration however hyperkeratosis was present, characteristic of grade 1 gastric ulcers (Sykes et al. 2015). Repeat endoscopy was performed 24 h later and revealed the initial stricture site had started to narrow again therefore balloon dilation was repeated at this site using the same technique. Endoscopy was repeated 24 h later and revealed satisfactory dilation of the oesophageal stricture.

The foal was discharged 2 days following the initial dilation procedure and maintained on a diet of nursing and grazing. Endoscopic re-evaluation of the oesophagus was performed at 5 and 19 days post discharge and revealed no evidence of recurrence of the oesophageal stricture. Subsequently, the foal was reintroduced back to her normal diet including hay, and tolerated this with no further episodes of oesophageal obstruction. The filly remains healthy on long-term follow-up one year later.

**Discussion**

Oesophageal strictures in horses are uncommon, and can have a poor prognosis for full recovery. Three types of oesophageal stricture have been classified: Type 1 (mural lesions), involving the adventitia and muscularis; Type 2 (oesophageal rings/webs), which involves the mucosa and submucosa only; and Type 3 (annular lesions), which involve all layers of the oesophagus (Borchers-Collyer et al. 2007; Waguespack et al. 2007). In all cases in this report, the strictures subjectively appeared to be mucosal (Type 2) based on endoscopic appearance; this presumption was supported by the positive response to balloon dilation, as mucosal oesophageal strictures are considered most amenable to treatment with balloon dilation (Hawkins 2012). However, because diagnosis of the strictures was based on clinical signs and endoscopic findings only, with only one animal undergoing contrast oesophagography, it is not possible to diagnose the type of stricture conclusively. Endoscopy is an important diagnostic modality in these cases as it also allows evaluation of the oesophageal mucosa and
increased the likelihood of development of gastric ulceration. In this report, although more acute episodes of oesophageal obstruction, external trauma to the oesophagus, nasogastric tube trauma or previous oesophageal surgery (Stick 2012), in foals, the incidence of acquired versus congenital strictures is not known. Often, diagnosis of a congenital stricture is made based on history and clinical signs, and an acquired stricture cannot be definitively excluded (Craig and Todhunter 1987; Berlin et al. 2015; Bezdekova et al. 2015). In man, congenital oesophageal strictures have been classified as membranous webs, tracheobronchial remnants and fibromuscular stenosis (Nihoul-Fekete et al. 1987; Feng and Kong 1999). Congenital strictures generally become apparent when the foal begins ingesting solid food, as in Foal C in this report, although more recent reports describe oesophageal strictures diagnosed in foals younger than 2 weeks who presented with milk regurgitation (Knottenbelt et al. 1992; Berlin et al. 2015; Bezdekova et al. 2015). Based on the history and the absence of abnormalities of the oesophageal or gastric mucosa, it is suspected that Foal C had a congenital stricture. This was suspected to be a membranous web-type stricture; however, it is difficult to differentiate between membranous web-type and fibromuscular stenosis based on endoscopic appearance alone, and both types of stricture are considered amenable to treatment using balloon dilation (Grabowski and Andrews 1996; Feng and Kong 1999). Foals A and E had evidence of gastric ulceration on gastroscopy. In human medicine, gastro-oesophageal reflux disease is one of the major causes of benign oesophageal strictures (Marks and Richter 1993). The finding of grade 3 gastric ulceration and the location of the stricture close to the cardia suggests that Foal A may have developed an acquired oesophageal stricture secondary to reflux oesophagitis (Murray et al. 1988). Foal E presented initially with dysphagia and did not experience an episode of oesophageal obstruction until Day 5 of hospitalisation. The foal’s history of severe autoimmune disease requiring hospitalisation as a neonate would have increased the likelihood of development of gastric ulceration and subsequent reflux oesophagitis (Furr et al. 1992). The finding of grade 1 ulceration on gastroscopy may support this hypothesis, although the location of the stricture more proximally in the oesophagus, away from the cardia, means that a congenital stricture cannot be ruled out. Foals B and D were older (4 months and 10 weeks, respectively) at presentation, and were clinically normal with no episodes of oesophageal obstruction until well after they began ingesting solid food. It is hypothesised that these foals experienced an initial episode of oesophageal obstruction that resulted in oesophageal mucosal ulceration and subsequent development of a stricture. Foal B was also diagnosed with megaoesophagus. This was considered to be obstruction-induced megaoesophagus rather than dysfunction-induced megaoesophagus, with dilation of the oesophagus proximal to the stricture presumed to have occurred secondary to the repeated episodes of oesophageal obstruction (Broekman and Kuiper 2002). Interestingly, the presence of megaoesophagus did not appear to negatively affect long-term outcome in Foal B, in comparison to the study by Prutton et al. (2015), which found that animals with concurrent megaoesophagus had a worse prognosis.

In human and companion animal medicine, balloon dilation is a favoured technique for the treatment of oesophageal strictures due to the decreased incidence of complications (Ogilvie et al. 1980; Wesdorp et al. 1982; Fraune et al. 2009). Endoscopic balloon dilation has been widely used in human medicine for the treatment of adults with oesophageal strictures (Ogilvie et al. 1980; Wesdorp et al. 1982), and studies have also confirmed the safety and efficacy of the procedure in children with oesophageal strictures (Lan et al. 2003; Althammari et al. 2011; Chang et al. 2011). Attempted balloon dilation of an equine oesophageal stricture was first reported by Knottenbelt et al. (1992), but the successful use of the technique was not described until Tillotson et al. (2003) who used the technique in a foal with a suspected congenital oesophageal stricture. Based on the findings in this and previous reports, the technique appears to be most effective in cases of acquired mucosal stricture, or congenital membranous web or fibromuscular strictures (Grabowski and Andrews 1996; Feng and Kong 1999; Hawkins 2012; Reichelt et al. 2012; Berlin et al. 2015). Although there are risks associated with balloon dilation, including oesophageal perforation and diverticulum formation (Melendez et al. 1998), the complications associated with the procedure are considerably less than those associated with surgical management, which include mucosal tears, oesophageal perforations, diverticula formation, post operative stricture formation, perioesophageal infection, dehiscence and death (Todhunter et al. 1984; Craig and Todhunter 1987). The ability to treat strictures located in the thoracic portion of the oesophagus is also important, as these strictures are not amenable to surgical management due to the difficulty in accessing this part of the oesophagus (Nixon et al. 1983). Another advantage is the ability to perform the procedure using standing sedation rather than general anaesthesia, which is particularly important in foals where aspiration pneumonia is a concern.

Medical therapy and diet management were used in conjunction with balloon dilation in all cases. Rather than attempting natural bougienage with long-fibre feeding, the decision was made to allow ingestion only of milk and grass due to concerns regarding further oesophageal trauma. This method appeared to achieve good results with the only instance of repeat oesophageal obstruction after balloon dilation occurring in Foal B, following the accidental feeding of a dry pelleted food. Gastroprotectant therapy with sucralfate was used in all cases, along with H2 antagonists and/or proton pump inhibitors in Foals A and B. Sucralfate is a tissue protective agent, and, in human medicine, it has been shown to improve mucosal healing, increase mucus and prostaglandin production and decrease inflammatory response (Akman et al. 2000). In the limited literature on the use of sucralfate in equine gastric ulcers (Becht and Byars 1986; Geor et al. 1989; Orsini et al. 2003), its efficacy has not been proven, so its use in these cases was empirical, based...
on literature regarding the use of sucralfate in human medicine. The combination therapy of sucralfate, ranitidine and omeprazole used in Foal A was based on clinician preference rather than proven effect. Antimicrobial therapy was also implemented in all cases due to ultrasonographic evidence of bronchopneumonia presumed secondary to aspiration with the exception of Foal B, which had no ultrasonographic evidence of bronchopneumonia but was treated with antimicrobials due to the risk of developing aspiration pneumonia. Third-generation cephalosporins, as used in all cases except Foal E, are classified as critically important antimicrobials for human medicine by the World Health Organization. The decision to use these antimicrobials in the cases in this report was made based on ease of administration, culture results where available, and knowledge of the most common organisms isolated from the respiratory system according to the Annual Sensitivity Report of Hagyard Equine Medical Institute’s Laboratory.

Two of the foals in this report were treated with intraoesophageal corticosteroid injections in addition to balloon dilation. In human medicine, local injection of corticosteroids has been shown to improve outcome in cases of refractory oesophageal strictures (Zein et al. 1995; Kochhar et al. 1999; Kochhar and Makharia 2002; Altintas et al. 2004; Ramage et al. 2005). Ramage et al. [2005] demonstrated that intralesional corticosteroid injection reduced the requirement for repeat dilations, and where repeat dilation was necessary then the average time between dilation procedures was increased. The successful use of the technique has also been described in small animals (Fraune et al. 2009). The tendency of some oesophageal strictures to persist or recur has been attributed to the continuous inflammatory response to injury and the resultant collagen formation in the submucosa and muscular layers of the oesophagus, which leads to scar contracture. Triamcinolone acetonide interferes with collagen cross-linking and chronic scarring, thereby decreasing stricture formation (Kochhar and Makharia 2002; Ramage et al. 2005). Corticosteroids have also been shown to decrease the amount of fibrotic healing that occurs after dilation (Ashcraft and Holder 1969). The technique appears to be safe with very few complications reported in human literature. One report documented the occurrence of intramural injection at the corticosteroid injection sites in 2/7 patients [Zein et al. 1995], although several other studies report no complications [Kochhar and Makharia 2002; Altintas et al. 2004; Ramage et al. 2005]. Use of the technique in equine oesophageal strictures has been reported once, with 3 of the 4 treated horses surviving without recurrence of the stricture [Prutton et al. 2015].

Other therapeutic options to minimise stricture reoccurrence include pentoxifylline, mitomycin C and systemic corticosteroids. The use of systemic corticosteroids in preventing stricture formation/recurrence is controversial. Inflammation is known to cause scarring [Hunt et al. 1994], so theoretically systemic corticosteroid therapy administered at time of dilation should reduce inflammation and potentially decrease stricture recurrence. One human study demonstrated the successful use of systemic corticosteroids in treatment of 2 children with oesophageal strictures (one caustic-induced and one anastomotic) where intralesional corticosteroid therapy had failed [Morikawa et al. 2008]. However a meta-analysis of a variety of human studies that looked at the effect of systemic corticosteroid administration following ingestion of corrosive substances, found no difference in the development of strictures between those patients treated with systemic corticosteroids and those who did not receive them, and in some instances adverse effects were observed [Pelcová and Navrátil 2005]. Pentoxifylline is a xanthine derivative that has been shown to ameliorate fibrosis via inhibition of fibroblast proliferation and synthesis of glycosaminoglycan and collagen (Berman and Duncan 1989; Lai et al. 1994; Fang et al. 2003). In a study on corrosive oesophagitis in rats, pentoxifylline was shown to reduce stricture formation significantly compared to an untreated control group [Apaydın et al. 2001]. Mitomycin C is an antibiotic and chemotherapeutic agent that reduces collagen cross-linking and inhibits fibroblast proliferation. It has been shown to reduce scarring in experiments in rats, and is widely used as a topical agent to prevent post operative fibrosis following ophthalmic surgery [Majmudar et al. 2000; Türkyılmaz et al. 2005]. It has been shown to be beneficial in preventing stricture reoccurrence in children and may hold potential as a topical treatment to prevent recurrence of oesophageal strictures in equine patients [Weisse and Berent 2015].

This case report describes the diagnosis and successful treatment of oesophageal strictures in 5 foals using endoscopic balloon dilation. In 4 of the cases, the strictures appeared to be Type 2 (mucosal) strictures, thought to be acquired in origin, while one was a suspected congenital membranous web-type stricture. In all cases, the oesophageal stricture was successfully dilated to allow the foals to return to a normal diet and all the animals remain healthy at long-term follow-up with no impact on athletic career. This indicates that balloon dilation is a safe and effective method of treating mucosal oesophageal strictures in foals and carries a good prognosis for survival and athletic potential. Further investigation is required to determine the efficacy of the technique in other types of strictures. To the authors’ knowledge, this is the first time that intraoesophageal corticosteroid injection has been successfully utilised in foals with recurrent oesophageal strictures. This technique may be a useful adjunct to balloon dilation in cases of refractory oesophageal strictures in foals.

Authors’ declaration of interests
No conflicts of interest have been declared.

Ethical animal research
This was a retrospective case study, so approval from local or national bodies is not applicable. A high standard of veterinary care was maintained throughout in all cases. Clients gave consent for all procedures performed and treatments administered. Client confidentiality has been maintained throughout the manuscript including in images.

Source of funding
None.

Antimicrobial stewardship policy
Third-generation cephalosporins are classified as critically important Antimicrobials for Human medicine by the World
Health Organization. The decision to use these antimicrobials was made based on the site of infection and knowledge of the most common organisms isolated from the respiratory tract according to the Annual Sensitivity Report of Hagyard Equine Medical Institute’s Laboratory. These antimicrobials should not be considered first-line treatments and may not be appropriate in other cases of oesophageal structure in foals.

Authorship

H. Chidlow was involved in the treatment of some of the cases and was responsible for data collection and interpretation, and preparation of the manuscript. N. Slolis was involved in the treatment of all cases and assisted in manuscript preparation. E. Robbins was involved in manuscript preparation. All authors approved the final version of the manuscript.

Manufacturers’ addresses

1Olympus America Inc., Center Valley, Pennsylvania, USA.
2Bracco Diagnostics Inc., Monroe Township, New Jersey, USA.
3Bechinger Ingehim Pharmaceuticals, Inc., Ridgefield, Connecticut, USA.
4Zoetis, Florham Park, New Jersey, USA.
5Hospira, Inc., Lake Forest, Illinois, USA.
6Merck Animal Health, Summit, New Jersey, USA.
7Actavis Inc., Parsippany, New Jersey, USA.
8Covance Research Products, Inc., Madison, Wisconsin, USA.

References


Continued from page 635


Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher’s website:

Supplementary item 1: Abnormal head posture and merialation in a mature horse with confirmed West Nile virus encephalitis.

Supplementary item 2: General proprioceptive ataxia and upper motor neuron tetraparesis in a mature Quarter Horse stallion with a caudal cervical vertebral stenotic myelopathy confirmed on post mortem examination.

Supplementary item 3: Circling of the same horse described in Supplementary item 2.

Supplementary item 4: Same horse as described in Supplementary item 2 demonstrating floating of the thoracic limbs when walked with the head raised.

Supplementary item 5: Supplementary item 4 slowed to half the original speed.

Supplementary item 6: Clinically normal horse showing an appropriate single limb hop response of the right thoracic limb.

Supplementary item 7: Same horse as in Supplementary item 2 showing appropriate hopping response on the left thoracic limb.

Supplementary item 8: Right thoracic single limb hopping response in a 3-year-old Quarter Horse gelding with a C6-7 cervical vertebral stenotic myelopathy confirmed at post mortem examination. Compare the distance the thorax travels over the right thoracic limb to that of Supplementary item 9.

Supplementary item 9: Left thoracic single limb hop of the same horse as in Supplementary item 8. A significantly delayed postural reaction hop is present compared to the opposite limb and that expected of a clinically normal horse.
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Original Article

Ultrasound diagnosis of injuries of the cranial meniscotibial ligament of the medial meniscus

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Keywords: horse; hindlimb; stifle; meniscus; ultrasonography

Summary

Ultrasonographic examination of the femorotibial joint is now commonly performed for investigating hindlimb lamenesses. Different types of meniscal injuries have been described using this technique. In our practice, the cranial meniscotibial ligament of the medial meniscus (CrMTL-MM) is the most frequently affected soft tissue structure of the stifle. This article describes the technique to image this ligament, normal images and the ultrasonographic manifestations of desmopathies and enthesopathies of the CrMTL-MM.

Introduction

Ultrasonographic examination of the femorotibial joint is now commonly performed for investigating hindlimb lamenesses. During the last two decades, meniscal injuries have been diagnosed and documented using ultrasonography and diagnostic arthroscopy (Denoix et al. 1994; Denoix and Lacombe 1996; Walmsley 2003, 2005; Walmsley et al. 2003; Nelson et al. 2016; Adrian et al. 2017) and normal as well as abnormal ultrasonographic images of the equine menisci have been previously described (Denoix 1996, 1998, 2003; Denoix and Lacombe 1996; Flynn and Whitcomb 2002; Hoegaerts et al. 2005; Werpy 2007; Denoix and Coudry 2004; Hoegaerts and Saunders 2004; Whitcomb 2012) and normal as well as abnormal ultrasonographic images of the equine menisci have been previously described (Denoix 1996, 1998, 2003; Denoix and Lacombe 1996; Flynn and Whitcomb 2002; Hoegaerts et al. 2005; Werpy 2007; Denoix and Coudry 2004; Hoegaerts and Saunders 2004; Whitcomb 2012) and normal as well as abnormal ultrasonographic images of the equine menisci have been previously described (Denoix 1996, 1998, 2003; Denoix and Lacombe 1996; Flynn and Whitcomb 2002; Hoegaerts et al. 2005; Werpy 2007; Denoix and Coudry 2004; Hoegaerts and Saunders 2004; Whitcomb 2012) and normal as well as abnormal ultrasonographic images of the equine menisci have been previously described (Denoix 1996, 1998, 2003; Denoix and Lacombe 1996; Flynn and Whitcomb 2002; Hoegaerts et al. 2005; Werpy 2007; Denoix and Coudry 2004; Hoegaerts and Saunders 2004; Whitcomb 2012) and normal as well as abnormal ultrasonographic images of the equine menisci have been previously described (Denoix 1996, 1998, 2003; Denoix and Lacombe 1996; Flynn and Whitcomb 2002; Hoegaerts et al. 2005; Werpy 2007; Denoix and Coudry 2004; Hoegaerts and Saunders 2004; Whitcomb 2012) and normal as well as abnormal ultrasonographic images of the equine menisci have been previously described (Denoix 1996, 1998, 2003; Denoix and Lacombe 1996; Flynn and Whitcomb 2002; Hoegaerts et al. 2005; Werpy 2007; Denoix and Coudry 2004; Hoegaerts and Saunders 2004; Whitcomb 2012) and normal as well as abnormal ultrasonographic images of the equine menisci have been previously described (Denoix 1996, 1998, 2003; Denoix and Lacombe 1996; Flynn and Whitcomb 2002; Hoegaerts et al. 2005; Werpy 2007; Denoix and Coudry 2004; Hoegaerts and Saunders 2004; Whitcomb 2012).

Cranial meniscotibial ligament of the medial meniscus (CrMTL-MM) injuries have been found in sport and racehorses presenting with a lameness localised to the stifle based on physical findings (e.g. synovial fluid distension, pain to flexion etc.) and/or positive diagnostic analgesia of the medial femorotibial joint.

The purpose of this article is to describe the technique to image the CrMTL-MM, normal images of this structure and ultrasonographic manifestations of desmopathies and enthesopathies of the CrMTL-MM.

Basic anatomy

The CrMTL-MM is a strong and short ligament attaching the cranial horn of the medial meniscus to the cranial intercondylar area of the tibial plateau close to the most cranial fibres of the cranial cruciate ligament (Barone 2000). Its insertion surface is slightly more cranial than the insertion surface of the cranial meniscotibial ligament of the lateral meniscus (Fig 1).

Ultrasonographic technique to image the CrMTL-MM

Routinely, this ligament is imaged with a 7.5–10 MHz linear probe. As it is deep in the femorotibial joint space on the extended stifle, the best way to image the CrMTL-MM is to flex the stifle (Cauvin et al. 1996; Coudry and Denoix 2004; Hoegaerts et al. 2005; Whitcomb 2012) and put the probe medial to the intermediate patellar ligament close to the tibial tuberosity (Fig 2). To follow the orientation of the tibial plateau the probe is slightly oblique proximomedially-distolaterally. This probe positioning provides a longitudinal section of the CrMTL-MM. Care should be taken to screen carefully the distal part of this ligament close to the tibial plateau. A complementary image can be done using a transverse section of this ligament turning the probe 90°. Both stifles should be examined.

Normal ultrasonographic images

The CrMTL-MM is a strong echogenic structure made of parallel fasciculi inserting on an oblique bone surface of the...
cranial intercondylar area (Fig 3). Its dorsal surface is in contact with the heterogenous infrapatellar fat pad.

Ultrasonographic findings and lesions

Several types of CrMTL-MM injuries with different clinical significance can be found.

Chronic desmopathy and enthesopathy of the CrMTL-MM is the most clinically significant injury in our cases (Fig 4a). It
has been found only in lame horses. The ultrasonographic manifestations are a thickening of the CrMTL-MM presenting an irregular echogenicity, an irregular insertional bone surface combining bone surface irregularity or defect and enthesophytes and periligamentous fibrosis. These findings have been confirmed at post-mortem examination (Figs 4b,c). No other abnormalities of the medial meniscus were seen.

Desmopathy of the CrMTL-MM can be found alone (Fig 5); it is quite rare and is usually not correlated to any clinical manifestation.

Tears of the CrMTL-MM with fibrillation are often associated with synovial cyst herniation (Fig 6). They can be accompanied by mild to moderate synovial fluid distension of the medial recess of the medial femorotibial joint and a low grade lameness.

Subinsertional bone surface irregularity or defect of the cranial intercondylar area at the attachment of the CrMTL-MM is a common finding (Fig 7). If found alone, it can be asymptomatic in sport and racehorses.

Discussion

Lesions of the medial meniscus are common in horses presenting with a lameness localised to the stifle (Denoix and Lacombe 1996; Walmsley 2003, 2005; Walmsley et al. 2003; De Busscher et al. 2006; Nelson et al. 2016; Adrian et al. 2017). The CrMTL-MM is the most frequently injured part of the medial meniscus (Walmsley et al. 2003; Walmsley 2005; Denoix 2008); its injuries can easily be diagnosed using an adequate ultrasonographic technique and can be seen in every breed and discipline. As with many anatomical structures, it must be noted that asymptomatic ultrasonographic abnormalities such as mild to moderate desmopathy or enthesopathy can be identified. However, the presence of fluid distension (synovial cyst herniation), enthesophytes and/or periligamentous fibrosis is an indication of potential or true clinical significance. As mentioned above, a careful examination of the interface between the CrMTL-MM and corresponding surface of the insertional bone of the cranial intercondylar area is essential to diagnose enthesopathies.

A clear evaluation of the proximal surface of the CrMTL-MM can be performed with diagnostic arthroscopy (Walmsley 2003, 2005; Barrett et al. 2012; Adrian et al. 2017) and ultrasonography complements this technique in providing imaging of the internal structure of the ligament and bone insertion surface (Barrett et al. 2012). Increased radiopharmaceutical uptake over the cranial intercondylar
area of the tibia at nuclear scintigraphy has been identified in lame horses with CrMTL-MM enthesopathy. Cross-sectional imaging such as computed tomography (Bergman et al. 2007; Nelson et al. 2016) and magnetic resonance imaging (Judy 2009) have a great potential for diagnosing injuries of the CrMTL-MM and associated insertional bone, but these techniques must be performed under general anaesthesia.

Conclusion
The most common soft tissue injuries of the equine stifle are CrMTL-MM desmopathies and enthesopathies. Their diagnosis can be performed on the standing horse using ultrasonographic examination of the craniomedial aspect of the flexed stifle. The most clinically significant injuries are chronic desmopathies and enthesopathies with periligamentous fibrosis.

Authors’ declaration of interests
No conflicts of interest have been declared.

Acknowledgements
The authors thank the referring veterinarians for providing the cases.

Authorship
Both authors have equally contributed to the manuscript conception and approved the final version.

References


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Review Article

Equine peripheral and infundibular dental caries: A review and proposals for their investigation

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Keywords: horse; equine dentistry; dental caries; peripheral caries; infundibular caries

Summary
Several different theories on the aetiology of dental caries have been proposed, but it is generally accepted that it is primarily caused by acidogenic microorganisms converting fermentable carbohydrates to acids. There is still some discussion on whether caries is caused by specific microorganisms or a nonspecific mix of different microorganisms and on whether caries is a classical infection or is caused by dysregulation of the normal oral bacteria (dysbiosis). Two types of dental caries are recognised in horses, i.e. peripheral dental caries and infundibular dental caries, with peripheral caries appearing to be increasingly recognised. Little is known about the prevalence and severity of peripheral dental caries in the general equine population, or the risk factors and microorganisms involved in its aetiopathogenesis. Limited pathological studies have shown 2 types of cemental destruction in equine peripheral caries, and indicate that gross dental examination underestimates the severity of equine peripheral caries.

Introduction
Dental caries is defined as a demineralisation of the calcified (inorganic) dental tissues and a destruction of its organic component (Soames and Southam 2005). The aetiopathogenesis of equine dental caries (both infundibular and peripheral) is poorly understood, although some prevalence surveys and limited pathological and conventional bacteriological studies have been performed on these disorders.

In contrast, dental caries in man, and in brachydont (short-crowned teeth) animals (often as models for human dentistry) have been well studied and consequently this literature review is mainly based on brachydont dental studies. Although incisors also have infundibulae, infundibular caries has only been described in maxillary cheek teeth. Peripheral caries can affect all teeth, but is very rare in canine or incisor teeth.

Aetiology
Chemoparasitic or acidogenic theory
Miller (1889) was first to propose bacterial involvement in the development of dental caries in the acidogenic theory of caries. He postulated that dietary carbohydrates were fermented by oral microorganisms into acids, primarily lactic acid, but also acetic and propionic acid. These acids caused a drop in the pH of dental plaque and when it decreases below the critical level of 5.5, mineral ions are released from the hydroxyapatite crystals of enamel, initiating caries.

The same occurs in cementum at a less acidic level, i.e. at a pH level of 6.7 (Tanzer 1992), whereas the critical pH in dentine is about 6.0 (Vanuspong et al. 2002). The opposite effect also occurs, i.e. teeth become remineralised when the pH increases above the critical value (Soames and Southam 2005). However, these critical pH levels are not rigid, because the process of demineralisation/remineralisation also depends on the levels of hydroxy, phosphate and calcium ions in plaque fluid and saliva (Dawes 2003). The higher these hydroxy, phosphate and calcium levels are in the fluid surrounding the teeth, the lower the critical pH will be. Because the concentrations of these ions in saliva and plaque fluid can vary between individuals, the critical pH levels can also vary accordingly. With demineralisation of teeth, bacterial destruction of the now-exposed proteins and other organic components of dental tissues also occur (Soames and Southam 2005). Gottlieb (1944) postulated in the proteolytic theory of caries that proteolysis of the organic matrix precedes the disintegration of inorganic components in caries development, with their organic matrix, like enamel lamellae and rods forming a pathway for the invasion of microorganisms. Later, Martin et al. (1955) proposed a simultaneous microbial degradation of organic components (proteolysis) and dissolution of minerals of the tooth by the process of chelation in the proteolysis-chelation theory. They suggested that the breakdown products of organic components have chelating properties and dissolved the minerals in the tooth. Current evidence favours the acidogenic theory initially proposed by Miller (1889).

Dental erosion
Caries must be differentiated from dental erosion, which is caused by the direct action of acids on teeth by dissolving exposed calcified dental surfaces (cementum, enamel and/ or dentine). Dental erosion occurs over a larger dental area compared to caries and without the need for bacteria. An erosive (i.e. acidic) agent is a greater challenge for an exposed dental surface than a cariogenic substrate (i.e. fermentable carbohydrates) since an erosive agent usually contains no or low levels of calcium and phosphate, while the milieu of an acidic plaque is partly saturated with these ions. Moreover, the rate of demineralisation of an exposed dental surface due to an erosive substance is higher than that caused by a caries process, since in the former, calcium and peripheral...
and phosphate ions, which become dissolved are detached from the dental surface very quickly and immediately lost, whereas in caries, the dissolved minerals are transported away from the tooth more slowly partly because of the overlying plaque (Shellis 2013). In horses, widespread dental erosion has been recorded in horses fed abnormally acidic silage (haylage) (Dixon et al. 2010).

Prerequisites for development of caries

The prerequisites for a dental caries lesion to develop are: tooth, substrate, plaque and bacteria (Keyes 1960). Because the environment of the tooth surface beneath plaque is largely anaerobic, the subsequent anaerobic metabolism of carbohydrates by plaque bacteria will preferentially produce acids. Although factors such as location, composition and morphology of the tooth may also play a role in the development of caries, caries will not develop without the presence of acidogenic bacteria and substrate (monosaccharides, disaccharides or other fermentable carbohydrates).

Substrate

The pH changes that occur on exposed dental surfaces in response to ingestion of fermentable dietary carbohydrates are similar in teeth with and without caries. However, the initial pH in the plaque overlying teeth suffering from caries is lower and therefore the pH will remain under the critical level for a longer period than occurs beneath the pellicle of healthy teeth (Soames and Southam 2005). The frequency of fermentable carbohydrate intake is also important in the pH cycling of plaque (Fig 1). The more frequently that fermentable carbohydrates are ingested, the longer the plaque will be below the critical pH and thus will result in a tilting of the balance between demineralisation and remineralisation towards demineralisation (Ten Cate 2015). It has been suggested that frequent feeding of high levels of concentrates to horses, may predispose to peripheral caries (Dixon et al. 2010), which is supported by the acidogenic theory. Horses trickle feed for up to 18 h/day, primarily on forage, and if such forage contains simple carbohydrates, such as fructans that occur in young grass, there is great potential to maintain a critical pH in their oral cavity for prolonged periods.

Pellicle, plaque and bacteria

The normal thin biofilm adherent to the surface of the teeth is termed a pellicle (acquired pellicle), but if this biofilm becomes very thick and of abnormal composition, it is termed a plaque, the presence of which is one of the prerequisites for caries development. Normal pellicle formation starts within seconds of a tooth being exposed to saliva and plays an important role in oral lubrication, regulation of mineral homeostasis and host defence (Siquera et al. 2012). The pellicle is a thin (0.5–1 μm), largely proteinaceous layer, containing some carbohydrates and lipids that form on the surface of normal teeth. The sources of these compounds are salivary secretions, gingival crevicular fluid, oral epithelial cell and oral microbial products (Hannig and Joiner 2006; Siquera et al. 2012). Bacteria can adhere to acquired pellicle within 3 min of exposure of teeth to saliva (Hannig et al. 2007) and the proteins in pellicle have specific receptors for bacterial adhesins that facilitate this process (Douglas 1994; Hannig et al. 2007).

Plaque is an abnormal, thick biofilm that mainly consists of an organic matrix of salivary mucins (mucopolysaccharides, the major glycoprotein components of mucus) and extracellular polysaccharide polymers with attached microorganisms (Soames and Southam 2005). As the plaque biofilm matures, its microbial community becomes more complex (Fig 2). The rate of growth of dental plaque depends on the availability of nutrients, competition with other microorganisms, and environmental conditions within the biofilm (Chávez de Paz et al. 2008). Predilection sites for plaque to accumulate include mechanically protected areas (Buchalla 2013) and this would also appear to be the case in horses, as plaque is frequently found in cheek teeth diastemata (Cox et al. 2012). In man, the microbial community of the supragingival plaque differs from that of the subgingival plaque (Costalonga and Herzberg 2014). Erridge et al. (2012) used a thickness of 10 μm to distinguish pellicle from plaque in an equine dental peripheral caries study.

Fig 1: The pH cycling in human dental plaque depends on the frequency of fermentable carbohydrate intake: a) eating 3 times a day; b) eating 6 times a day; and c) eating 9 times a day. The arrows indicate the time of food intake. The broken red lines represent the critical pH under which demineralisation occurs and above which remineralisation can occur (adapted from Ten Cate 2015).
Supragingival plaque can have a structured architecture with polymer-containing channels (or ‘black holes’) connecting the dental surface with the oral cavity (Auschill et al. 2001; Marsh 2005). The microorganisms in this biofilm have an uneven spatial arrangement (Auschill et al. 2001), with the most viable bacteria present in the central part of the plaque and lining the channels where diffusion of nutrients takes place. Dead bacteria surrounding the viable bacteria were found closest to the tooth surface and the oral cavity and may function to protect the underlying, living microorganisms (Auschill et al. 2001).

Microbial community in plaque

The microbial community that forms in plaque has many advantages for the inhabiting microorganisms (Marsh 2005). Firstly, pioneer microbial colonisers create a microenvironment that is suitable for the attachment and growth of other microorganisms, a process termed coaggregation (Metwalli et al. 2013) (Fig 2). Secondly, molecules that cannot be broken down by individual species of bacteria can often be catabolised by the combination of microorganisms living in this community. Additionally, a pathogenic synergism may occasionally occur, causing the combination of organisms in the community to be more pathogenic than any of the individual microorganisms. Furthermore, the collaboration and gene transfer, which are likely to occur in a microbial plaque community, make them more resistant to antimicrobial therapeutics, environmental stress and host defences than oral bacteria living in isolation (Marsh 2005).

Another survival mechanism that oral bacteria are believed to use is a dormancy state during times of nutrient deprivation, when they enter a state of metabolic arrest without undergoing cell division or growth. During this dormant state, bacteria are less sensitive to antimicrobial agents, and also to changes in temperature and pH. When the bacteria later regain access to sufficient nutrients, they return to their higher metabolic rates (metabolic reactivation), with resumption of cell growth and division. A slow reactivation of nutrient-deprived Streptococcus anginosus and Lactobacillus salivarius in oral biofilms after the introduction of nutrients was suggested to also be part of their survival strategy (Chávez de Paz et al. 2008). An enhanced synthesis of certain proteins (that act as stress proteins) by some oral bacteria such as Streptococcus mutans may also help these bacteria survive suboptimal conditions (Svensäter et al. 2000).

Microorganisms involved in human dental caries

The cultivable microbiological flora in dental plaque varies between herbivorous, carnivorous and omnivorous mammalian species, while within the same dietary group, the microflora appears to be quite similar (Dent 1979). Using molecular bacteriological techniques, the Human Oral Microbiome Database now includes approximately 700 microbial species including bacteria and archaea that can be present in the human oral cavity in health and disease (Chen et al. 2010). Although Miller’s acidogenic theory has been generally accepted, there are different theories about which microorganisms are important in the development of dental caries.

Specific plaque hypothesis

In the specific plaque hypothesis, specific pathogenic microorganisms are proposed to cause caries. Lactobacilli were initially believed to be the most important bacteria in caries development because of their acidogenic and acidic characteristics, meaning that they can produce acid and survive in an acidic environment, respectively (Kligler and Gies 1915; Howe and Hatch 1917). However, Clarke (1924) discovered a further acidogenic and acidic bacterium: S. mutans, which additionally produces extracellular sticky glucans and intracellular polysaccharides. Extracellular sticky glucans enable bacteria to adhere to teeth and intracellular polysaccharides can be converted to...
acidic end-products, when dietary sugars are absent from the oral cavity (van Loveren et al. 2012).

A causal relationship between S. mutans and caries has been established in experiments with gnotobiotic rats (Fitzgerald et al. 1960; Gibbons et al. 1966) and conventional hamsters (Fitzgerald and Keyes 1960; Keyes 1960). These animals developed caries after exposure to caries-active conspecifics (Keyes 1960) or after their teeth were inoculated with ‘caries inducing streptococci’ (Fitzgerald and Keyes 1960) most of which fit the description of S. mutans (Edwardsson 1968; Guggenheim 1968). Following these studies, caries was classified as a transmissible infectious disease with S. mutans as the sole pathogen. More recently, other mutants streptococci such as Streptococcus sobrinus were classified as similar pathogens by some researchers, although these latter bacteria were less frequently identified in caries lesions, and when present, were in much smaller numbers than S. mutans (Shells 2013).

Nonspecific plaque hypothesis

However, caries has also been found in the absence of S. mutans (van Houte 1994; Kleinberg 2002). This finding has led to a shift from the specific plaque hypothesis that necessarily involves pathogenic mutants streptococci such as S. mutans, to the mixed/nonspecific plaque hypothesis (Kleinberg 2002; Kianoush et al. 2014). In this latter hypothesis, a wide range of acidogenic bacteria are proposed to be involved in the development and progression of caries, with the viridans streptococci including S. mutans, S. sobrinus, Streptococcus salivarius, Streptococcus sanguis and Streptococcus mitis believed to be the initial and main pathogens, followed by secondary invaders including Actinomyces, Bacteroides, spirochetes and lactobacilli (Maripandi et al. 2011). Other bacteria including Bifidobacterium, Propionibacterium, Veillonella, Selenomonas and Atopobium (Kianoush et al. 2014), Prevotella and Fusobacterium, can also be associated with caries (Maripandi et al. 2011).

Additionally, recent studies have shown that in addition to bacteria, high numbers of Candida albicans fungi (as yeast, filamentous cells or pseudofilaments) can be found in human dental plaque (Barbieri et al. 2007; Maripandi et al. 2011). Although C. albicans is normally a unicellular oral commensal, it can switch to a pathogenic invasive, multiple filamentous form to infect dental tissues. Moreover, C. albicans and S. mutans appear to interact with the presence of C. albicans enhancing the attachment of S. mutans to teeth and vice versa (Metwalli et al. 2013). S. mutans produces lactic acid which stimulates yeast growth and in turn, yeast growth decreases oxygen levels and produces growth factors for streptococci. The most common form in which C. albicans occurs with S. mutans is the yeast form with production of blastospores (Barbieri et al. 2007).

Ecological plaque hypothesis

Local ecological conditions are also important in the development of caries, as noted in the ecological plaque hypothesis. In this model, a biofilm is considered to consist of a normal resident bacterial community, i.e. a state of eubiosis, whereas caries reflects the presence of an abnormal oral bacterial community, i.e. a dysbiosis (Kidd 2005). A change in the local environment can result in an imbalance of plaque microflora causing dental demineralisation (Kidd 2005). Frequent access to dietary fermentable carbohydrates or a decreased clearance of carbohydrates by saliva, e.g. due to a lower saliva secretion rate, can lead to more acid being produced with subsequent demineralisation of tooth substance (Kidd 2005; Oisen 2006). A low pH is also beneficial for the growth of acidogenic and aciduric bacteria, thus enhancing the acidifying effect and predisposing the associated dental site to caries.

Substantial core model

The substantial core model was proposed after the finding that in a pH range of 4.5–7.8, approximately 60% of the bacteria taxa associated with dental caries (including Leptotrichia and Prevotella species and Streptococcus salivarius) can be found in carious dentine lesions regardless of the pH (Kianoush et al. 2014). A low diversity in microbiota was present in acidic conditions, whereas the microbial populations were more variable in pH neutral environments.

Microorganisms involved in equine dental caries

Little is known about the bacteria that are involved in equine dental caries, although a recent conventional and molecular bacteriological study revealed the presence of a newly discovered bacterial species, i.e. Streptococcus devriesei in cheek teeth infundibular caries lesions (Lundström et al. 2007). Baker (1979) reported that the healthy equine oral cavity often had high numbers of streptococci and micrococci, with low numbers of Lactobacillus spp., Fusobacterium spp. and colliforms and intermediate numbers of anaerobes. Veillonella spp. and hydrogen sulphide-producing bacteria. In equine periodontal disease a shift in cultivable bacteria occurred with progression of the disease, with a decrease in Gram-positive cocci and rods and an increase in Gram-negative aerobes, anaerobes and spirochetes (Baker 1979).

Equine cheek teeth infundibular caries

Different studies have described very diverse prevalences of equine (maxillary cheek tooth) infundibular caries, varying from 8% (Fitzgibbon et al. 2010) to 100% (Honma et al. 1962). This difference could possibly be explained by cemental hypoplasia being classified as infundibular caries by some authors, and also to age-related differences, as the high prevalence found by Honma et al. (1962) was in horses older than 12 years.

Using light microscopy and ultrastructural examinations, Klici et al. (1997) found infundibular caries in 24% (5/21) of maxillary cheek teeth: involving the centre of infundibular cementum in 4 of these teeth and its periphery in one. Most (63%; 10/16) other maxillary cheek teeth contained one or 2 small central infundibular channels (termed vascular channels) filled with shrunken connective tissue. In recently erupted teeth, many lateral branches of the central vascular channels extended into the infundibular cementum, reducing in size towards its periphery before terminating adjacent to the cementoenamel junction.

The presence of areas of cemental hypoplasia in the vascular channels seems to predispose to the development of localised central infundibular caries. It has also been proposed that when areas of hypoplastic infundibular cement are exposed to the oral cavity with dental wear, food and oral microorganisms enter these defects and
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predispose to the development of more severe infundibular caries (Baker 1974; Kilic et al. 1997). This is supported by the finding that the maxillary 09s are usually most severely affected by infundibular cemental hypoplasia and also with infundibular caries (Windley et al. 2009; Fitzgibbon et al. 2010). However, a recent clinical survey in donkeys found the 06s to be most commonly affected by infundibular caries (Rodrigues et al. 2013). Infundibular caries may lead to apical infection if caries proceeds through infundibular enamel and the adjacent dentine and pulp become affected (Dacre et al. 2008), or to a pathological dental fracture (most often sagittal – termed caries-related infundibular fractures) (Dixon et al. 2014) as a result of mechanical weakening of the tooth in advanced caries (Dixon 2002; Dacre et al. 2007).

The system that is most commonly used for grading equine infundibular (Table 1) and peripheral caries (Table 2) is the modification of the Honma et al. (1962) system described by Dacre (2005).

**Equine dental peripheral caries**

Peripheral caries can affect all of the dental calcified tissues (cementum, enamel and dentine), and so the terms peripheral caries or peripheral dental caries are preferable to the previously used term of peripheral cemental caries (Dixon et al. 2010). This type of caries has some similarities to smooth surface caries in brachydont teeth. The prevalence of equine peripheral caries appears to be increasing in Europe (Gere and Dixon 2010). Wafa (1988) described a peripheral caries prevalence of 0.3% in 355 horse skulls in a post mortem study in Ireland and a 0.9% prevalence was found in dental surveys in Swedish horses by Lundström and Pettersson (1988, 1990). A more recent post mortem study on Swedish horses reported a prevalence of 6.1% (31/510) peripheral caries (Gere and Dixon 2010). A recent clinical survey of 800 donkeys from the Spanish-Portuguese border showed a similar peripheral caries prevalence of 5.9% (Rodrigues et al. 2013).

A post mortem study of 22 equine skulls showed dental plaque overlying the interdental tooth surfaces in most mandibular teeth that were examined (Cox et al. 2012). Erriage et al. (2012) found that 67% of (peripheral) equine cheek teeth caries lesions were covered by plaque, which was usually 10–1000 μm thick, sometimes with food adherent to the plaque or teeth. The clinical crown surfaces of all control teeth contained a pellicle (<10 μm in thickness) but no food material was histologically present in this pellicle. On gross examination, Gere and Dixon (2010) found food firmly attached to the sides of some cheek teeth affected by peripheral caries that may further have contributed to maintaining a localised caries-inducing environment on the surface of the teeth.

Older equids are more commonly affected by peripheral caries and additionally, caries lesions appear to be more severe in older animals (Dacre et al. 2008; Dixon et al. 2010; Gere and Dixon 2010). Peripheral caries most commonly affects the caudal cheek teeth (Triadan 09–11) (Gere and Dixon 2010).

**TABLE 1: Grading of infundibular caries using the modified Honma system (from Dacre 2005)**

<table>
<thead>
<tr>
<th>Grade of caries</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0</td>
<td>Normal tooth, i.e. no macroscopic infundibular caries visible. Discolouration, (without pitting) of peripheral cement, possibly of dietary origin, is present in some normal teeth</td>
</tr>
<tr>
<td>Grade 1</td>
<td>Only cementum is affected: lesions appear as superficial or focal pitting lesions or even as extensive cemental loss, although some cementum remains visible. Discolouration, (without pitting) of peripheral cement, possibly of dietary origin, is present in some normal teeth</td>
</tr>
<tr>
<td>Grade 2</td>
<td>Cementum and underlying enamel are affected</td>
</tr>
<tr>
<td>Grade 3</td>
<td>Only cementum affected: lesions appear as superficial or focal pitting lesions or even as extensive cemental loss, although some cementum remains visible. Discolouration, (without pitting) of peripheral cement, possibly of dietary origin, is present in some normal teeth</td>
</tr>
<tr>
<td>Grade 4</td>
<td>Dental integrity is affected (i.e. secondary dental fracture present)</td>
</tr>
</tbody>
</table>

**TABLE 2: Grading of peripheral caries according to the modified Honma system (from Dacre 2005)**

<table>
<thead>
<tr>
<th>Grade of caries</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0</td>
<td>Normal tooth, i.e. no macroscopic peripheral caries visible. Discolouration, (without pitting) of peripheral cement, possibly of dietary origin, is present in some normal teeth</td>
</tr>
<tr>
<td>Grade 1</td>
<td>Only cementum is affected: lesions appear as superficial or focal pitting lesions or even as extensive cemental loss, although some cementum remains visible. Discolouration, (without pitting) of peripheral cement, possibly of dietary origin, is present in some normal teeth</td>
</tr>
<tr>
<td>Grade 2</td>
<td>Cementum and underlying enamel are affected</td>
</tr>
<tr>
<td>Grade 3</td>
<td>Cementum, enamel and dentine are affected</td>
</tr>
<tr>
<td>Grade 4</td>
<td>Dental integrity is affected (i.e. secondary dental fracture present)</td>
</tr>
</tbody>
</table>
Dixon 2010). The main equine salivary ducts drain rostrally in the mouth, therefore the buffering effect of saliva may be less in the caudal aspect of the equine oral cavity (Gere and Dixon 2010).

The prevalence of diastemata in a Swedish post mortem study was significantly higher (64.5%) in horses with, than without peripheral caries (45.7%) and the 3 caudal cheek teeth were more commonly affected by both diastemata and peripheral caries (Gere and Dixon 2010). This is in contrast with Ramzan and Palmer’s (2011) clinical study of 108 horses where diastemata were predominantly observed within the mandibular cheek teeth quadrants and affected all interdental spaces, with no significant association found between the presence of diastemata and peripheral caries.

Periodontal disease can sometimes be found adjacent to areas affected by peripheral caries (Gere and Dixon 2010). However, Cox et al. (2012) showed that dental plaque often covered cemental ‘erosions’, but no statistically significant relationship could be found between the amount of plaque or degree of peripheral cemental erosions present and the presence and severity of periodontal disease. Rodrigues et al. (2013) found that periodontal disease was present in only 3.9% of peripheral caries cases. Concurrent infundibular caries was found in 13% (Erridge et al. 2012) and 32% (Gere and Dixon 2010) of teeth affected by peripheral caries.

Peripheral caries lesions that were macroscopically graded as grade 1.1 showed 2 distinct histological patterns (Erridge et al. 2012). In one type, layers of peripheral cementum became undermined by plaque and flaked off, as also occurs in human root (cemental) caries. In the second type, flask-like carious lesions filled with plaque were present. grade 1.2 lesions showed extensive histological loss of peripheral cementum and thus the underlying enamel of the clinical crown was exposed. Although enamel exposure was often observed around the entire circumference of the tooth, the enamel was unaffected, possibly because a much lower pH (5.5) is required for enamel to develop caries in contrast to cementum (pH 6.7). Because enamel is dissolved during the histological decalcification process, it histologically appears as an empty space between cementum and dentine. Whilst there was still a very small layer of intact cementum covering the enamel in grade 1.2 lesions, in grade 2 lesions, plaque was found within the enamel space indicating that the full cemental layer and the underlying enamel were now affected.

Although grade 3 lesions were not macroscopically found in Erridge et al.’s study, Gram staining showed bacteria within the dentinal tubules in 63% of sections with peripheral caries, demonstrating the involvement of dentine, thus showing that macroscopic grading of carious lesions underestimates their severity as compared to histopathological examination (Erridge et al. 2012). Additionally, Gram or Picrosirius Red staining revealed even higher grades of caries than was found with haematoxylin and eosin staining. It was remarkable that even teeth which macroscopically and microscopically (with haematoxylin and eosin staining) appeared to have grade 1.1 lesions, turned out in fact to have grade 3 lesions following Gram staining. This suggests that in equine (peripheral) cemental caries, just as occurs in human cemental (root) caries and donkey infundibular cemental caries, a simultaneous bacterial colonisation and demineralisation occurred (Frank 1990; du Toit et al. 2008), whereas in human enamel caries, demineralisation appeared to precede bacterial invasion (Frank 1990).

Areas for further research
As noted, there is a lack of objective information on the prevalence and severity of peripheral caries in many equine populations, including those in the UK. Additionally, little is known of the risk factors for the development or progression of peripheral caries. It was hypothesised that the feeding of haylage is a risk factor (Gere and Dixon 2010) but the more recent study of Rodrigues et al. (2013) showed a 5.9% peripheral caries prevalence in donkeys that never ate haylage. The feeding of high levels of concentrates (cereal based food) has also been hypothesised to be a risk factor (Dixon et al. 2010; Gere and Dixon 2010), but these areas need to be factually examined, before any objective guidelines on the prevention or control of equine peripheral caries can be given.

Little is known about the microorganisms involved in equine dental (infundibular or peripheral) caries. A comparative conventional bacteriological examination of swabs and scrapings of plaque from cavitous dental tissue and of pellicle from control horses would determine which cultivable bacteria are involved in this disease. Additionally, because approximately 50% of oral bacteria cannot be conventionally cultured (Siqueira and Rôças 2013), molecular bacteriology of the equine oral cavity in control and caries affected horses needs to be performed, for example using next generation sequencing of bacterial 16S RNA gene amplicons. The results of conventional and molecular bacteriology could then be correlated. Until accurate knowledge of the microorganisms involved in equine dental caries is known, the value of equine antiseptic mouthwashes currently used cannot be critically evaluated.

Authors’ declaration of interest
No conflicts of interest have been declared.

Ethical animal research
Ethical review not applicable for this review article.

Source of funding
Dewi Borkent is a postgraduate research student kindly funded by The Horse Trust.

Authorship
D. Borkent wrote this review article with help from P. Dixon.

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1. Publication Title
   Equine Veterinary Education

2. Publication Number
   [Redacted]

3. Filing Date
   9/28/17

4. Issue Frequency
   Monthly

5. Number of Issues Published Annually
   12

6. Annual Subscription Price
   $151.80

7. Complete Mailing Address of Known Office of Publication (Not printer) (Street, city, county, state, and ZIP code)
   AAEPP
   4033 Iron Works Parkway
   Lexington, KY 40511

8. Complete Mailing Address of Headquarters or General Business Office of Publisher (Not printer)
   Same

9. Full Names and Complete Mailing Addresses of Publisher, Editor, and Managing Editor (Do not leave blank)
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13. Publication Title
   Equine Veterinary Education

14. Issue Date for Circulation Data
   August 2017

15. Extent and Nature of Circulation
   Scientific journal mailed to equine veterinarians who are members of the AAEPP

   a. Total Number of Copies (Net press run)
      7,713
      7,952

   b. Paid Circulation
      (By Mail and Outside the Mail)
      (1) Paid Outside County Paid Subscriptions Stated on PS Form 3541 (include paid distribution above nominal rate, advertiser's proof copies, and exchange copies)
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      6,501
      (2) Paid Outside the Mail Including Sales Through Dealers and Carriers, Street Vendors, Counter Sales, and Other Paid Distribution Outside USPS
      0
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      (3) Paid Distribution by Other Classes of Mail Through the USPS (e.g., First Class Mail)
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      50
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   f. Total Distribution
      (Sum of (1) and (15c))
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      7,710

   g. Copies Not Distributed
      (See instructions to Publishers 84 (page 3))
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   i. Percent Paid
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   in the November issue of this publication.
   ☐ Publication not required.

18. Signature and Title of Editor, Publisher, Business Manager, or Owner
   David Feole, AAEPP
   4033 Iron Works Parkway
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   Date
   9/28/17
Evaluation of ataxia in the horse

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Keywords: horse; neurology; ataxia; examination

Summary
Ataxia in the horse is a common clinical presentation requiring a methodical approach for accurate neurolocalisation. Observed neurological deficiencies should support a localised single aetiology whenever possible. Relevant differential diagnoses should be considered based on the case history and localisation. Diagnostic strategies can then be applied relative to disease risk.

Introduction
Examination of the ataxic horse is commonplace in practice. Early descriptions seem to hold true today; 'The power to move with speed is entirely lost, nor is the ability to progress at a slower pace by any means assured' (Mayhew 1861). Although descriptions of equine ataxia have changed little, our recognition of common disease presentations has. This review will focus on clinical examination and neurolocalisation of ataxia in the horse.

A brief discussion of terminology is appropriate given the overlapping descriptors and the propensity to incorporate our recognition of common disease presentations has. This deficiency in generation of the gait (upper motor neuron – UMN) or the ability to support weight (lower motor neuron – LMN) (DeLahunty 2009). The word ‘ataxia’ derives from to a- (without) + taxis (order; modern Latin, Greek origin) and is defined medically as the loss of ability to coordinate movement. Specific to this review, ataxia will be discussed as a result of spinal cord injury and dysfunction (SCD). It is important to note other origins of ataxia that can lead to 3 manifestations of disease: vestibular, cerebellar and general proprioceptive. The following is a brief anatomical and clinical description of these disorders, in-depth descriptions of these disorders can be found in the referenced neuroanatomy texts (Furr and Reed 2008; Mayhew 2008; DeLahunty 2009; Smith 2009; Thomson et al. 2012).

Vestibular ataxia results from any disturbance of the vestibular system originating at the receptors (inner ear), along the course of axon or vestibular ganglion (petrosal portion of temporal bone) or the vestibular nuclei (brainstem). Vestibular ataxia is further subdivided into peripheral (PVD) and central vestibular dysfunction based on pathology location relative to the vestibular brainstem nuclei. The most common clinical equine presentation is unilateral PVD causing head tilt with the poll deviating towards the side of dysfunction (muzzle away), pathological nystagmus with contralateral fast phase (away from side of dysfunction) and ipsilateral listing of the trunk. Ventral deviation of the eye may be noted ipsilateral to the lesion location. If confined to a small stall, horses with PVD may fall or circle towards the affected side or assume a listing position against the stall wall on the affected side. The quality of circling should not be misinterpreted with that of a forebrain (prosencephalon) disturbance, which tends to be more propulsive and less influenced by the size of confinement. Ipsilateral facial neuropathy accompanies most cases of PVD due to its close approximation to the vestibular nerve within the internal acoustic meatus and petrosal portion of the temporal bone. Acquired ipsilateral deafness is a feature of PVD due to cochlear nerve injury along the same course of the vestibular nerve (Aleman et al. 2014). Temporohyoid osteoarthropathy is one of the most common clinical presentations of unilateral PVD and facial neuropathy in the mature horse. Important differentiating observations of central vestibular dysfunction are mentation changes, presence of other cranial nerve deficits (facial nerve exception), postural reaction deficits or UMN paresis.

Cerebellar ataxia in the mature horse is unusual and is much more common in the neonate and foal. Cerebellar ataxia may be developmental, as in cases of cerebellar atrophy and Dandy-Walker syndrome, or acquired from structural or inflammatory disturbances in the mature horse (Miller et al. 1985; Wong et al. 2007; Braull et al. 2011). Clinical observation shows distinct hypermetria of the thoracic and occasionally pelvic limbs, which may worsen with gait speed. Postural reaction tests (described below) may be normal to exaggerated. Intention tremors of the head may be observed during voluntary movement or when stationary due to disturbances of cerebellar influence on fine motor control. Absent menace response may be observed with loss of cerebellar integration of the evoked blink reflex. Appropriate mentation is commonly observed in cases of congenital cerebellar disease. Acquired cerebellar disease may be due to multifocal encephalopathy and have concurrent mentation changes, cranial nerve dysfunction, general proprioceptive ataxia and postural reaction deficits. As a general rule, the most severe symptoms are associated with the area of most significant disease, e.g. primary forebrain pathology will have profound forebrain dysfunction with the potential for secondary vestibular/cerebellar ataxia that might be recognised clinically.

The remainder of this review will address general proprioceptive ataxia due to SCD, or spinal ataxia. General proprioceptive ataxia can occur with any injury affecting the sensory (afferent) components of the central nervous system; however, most spinal cord injuries disturb both sensory and motor (afferent) components given their anatomic proximity. Peripheral nervous system afferent inputs gathered from peripheral receptors (i.e. Golgi tendon receptors, muscle spindles) ascend peripheral nerves and join with spinal nerves...
to enter the spinal cord. Cell bodies of these nerves are located in the spinal ganglion (dorsal root ganglion). Post ganglionic axons may terminate locally on interneurons for myotactic reflex activity or course cranially to the brainstem or cerebellum for conscious and subconscious proprioception. Efferent UMN responses descend from the forebrain and brainstem, under cerebellar influence, via ventrolateral tracts of the spinal cord, synapsing on LMN cell bodies. Large accumulations of LMNs (grey matter) are present in the cervical (C6–T2) and lumbosacral intimescences (L4–S2) representing the increased number of nerves for motor function of the limbs. The LMN has both central (spinal cord) and peripheral (axon to neuromuscular junction) nervous system components. Disturbances along the afferent or efferent (often both) central nervous system pathways within the spinal cord can result in general proprioceptive ataxia and UMN dysfunction, respectively. Clinical observation of general proprioceptive ataxia or UMN paresis may appear as a single entity; however, the ability to differentiate between these systems clinically is controversial, and is probably not necessary for SCD localisation. Therefore, the description of general proprioceptive ataxia due to SCD typically includes a description of motor function [tetra- or paraparesis]. Subjective grading of ataxia and UMN paresis is most appropriately suited for cervical SCD (C1–T2, Fig 1) (DeLahunta 2009).

History
A detailed history is essential to developing an appropriate problem list, disease differentials and diagnosis. Open questioning of the caretaker, while avoiding leading statements, can enhance a practitioner’s awareness of relative disease risk. ‘How long has your horse been ataxic?’ assumes the horse has a neurological dysfunction leading to ataxia and influences the perception of signs. The typical anamnesis begins with the case background, where age and breed specific risk factors can be taken into account. Other critical information gained is the onset, duration and progression of problems. Note clinical observations prior to interpretation when formulating a problem list. Documenting previous treatments is important given the propensity of most horse owners to medicate empirically prior to seeking veterinary care.

Examination
The principles of examination are based on a consistent, efficient and systematic approach. An example neurological examination chart is shown in Fig 1. Consistency is valued over template conformity. The practitioner should find an approach that is best suited and safe for the individual case. Observing the horse in its environment can yield information regarding gait disturbances, behaviour and mentation. Similar observations of a horse loose in a hospital box stall also suffice. Most examinations start at the head and work caudally. Head and neck posture are noted. Head pressing, head tilt or lateral flexion of the neck are suggestive of encephalopathy, vestibular/cerebellar and unilateral forebrain dysfunction, respectively. Cervical spinal cord injury can result in a head tilt due to acquired torticollis (McKelvey and Owen 1979; Van Bieren et al. 2004).

Most horses investigate their new surroundings, vocalise, urinate and defaecate, sometimes in that order, when placed in unfamiliar settings. Mentation derangements can be classified as alert and responsive, lethargic, obtunded, semicoma (stupor) or coma. Supplementary item 1 is an example of an obtunded horse diagnosed with West Nile virus encephalitis. Ataxia may originate from intracranial pathology, hence the importance of this observation and performing a complete cranial nerve examination prior to the locomotor assessment. Abnormal mentation changes or cranial nerve deficits could support multifocal disease or primary intracranial pathology. Extracranial sources of encephalopathology should be considered when formulating differential diagnoses. Additionally, myopathies, polyneuropathies and laminits can have similar presentations as SCD.

Identifying regions of muscle atrophy or asymmetry are supportive evidence of neurogenic atrophy from SCD. Rapid focal muscle loss is typical of neurogenic atrophy following LMN injury, while a slower course of disuse muscle atrophy is common with UMN disorders. Assessing cardiovascular and pulmonary function can exclude extracranial causes of weakness often misinterpreted as neurological dysfunction. Identify pre-existing diseases such as musculoskeletal abnormalities, evidence of laminits and arthropathies prior to the locomotor examination.

Determine the lateral range-of-motion of the head and neck. This can be performed by enticing the horse to voluntarily flex the neck laterally in either direction with food, or slow manual flexion. Articular cavitation or ‘cracking’ sounds may be heard when laterally flexing the neck of normal horses. Resistance to flexion or asymmetrical decreased range-of-motion may be observed with severe neck pain due to osteoarthrits of the articular processes, cervical myelopathies, neoplasia, or malformations and fractures of the cervical vertebrae. Extremely low head carriage, absent evidence of trauma, is suggestive of a central nervous system or neuromuscular disorder.

Cranial nerves, cervical and spinal reflexes and physical examination
Further reading on performing the cranial nerve examination can be found at the end of this review (Furr and Reed 2008; DeLahunta 2009; Smith 2009; Reed et al. 2010). Careful evaluation of cranial nerve function is critical to excluding intracranial pathology and identifying cervical SCD. The cervicofacial (cervicoauricular) reflex is an evoked facial motor response following stimulation of the cervical (C1–C3) dermatomes (Levine et al. 2007). A blunt instrument is tapped against the lateral skin between the jugular groove and crest of the neck from C3 cranial. The ipsilateral eye should be covered to limit visual influence. The observed response is contraction of the ipsilateral labial commissure (‘smile response’), caudal movement of the ipsilateral ear and blinking. Brainstem and/or cranial cervical spinal cord injury can result in an abnormal cervicofacial reflex (Rooney 1973). Cutaneous stimulation of the skin caudal to C3 will result inipsilateral movement of the neck which is a proposed local reflex arc (DeLahunta 2009). Caudal cervical SCD can interfere with this reflex, although the clinical sensitivity of this test is questionable (Levine et al. 2007; DeLahunta 2009).

The thoracolaryngeal reflex or ‘slap test’ is performed by slapping the lateral thorax caudal to the shoulder and palpating, or endoscopically observing, contralateral laryngeal adduction. Afferent sensory stimulus from the
Equine Neurologic Examination

Name ______________________ Age _____ Gender______
T° _____ Pulse ______/Rhythm _____ Resp _____/Character______
MM color ______ CRT ______s
Sensorium:
Alert and Responsive, Depressed, Lethargic, Obtunded, Semicoma (Stupor), Coma

Cranial Nerves:
<table>
<thead>
<tr>
<th>Normal</th>
<th>Abnormal</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head tilt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Facial symmetry</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pupils</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pupillary light response</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fundus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nystagmus/Strabismus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Palpebral reflex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Menace response</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jaw/lingual tone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phonation/swallowing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slap test</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Postural Reactions/Spinal Reflexes:

<table>
<thead>
<tr>
<th>Thoracic</th>
<th>Pelvic</th>
<th>Recumbent</th>
<th>Thoracic</th>
<th>Pelvic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left</td>
<td>Right</td>
<td>Left</td>
<td>Right</td>
<td></td>
</tr>
<tr>
<td>Limb placing</td>
<td>Patellar</td>
<td>XXXXXXXXXXX</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hopping</td>
<td></td>
<td>Withdrawal/pain</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Muscle tone/symmetry:
Adequate, atrophy (focal, regional, generalized), movement (fasciculations, tremors, myotonus, myoclonus)

Gait:

<table>
<thead>
<tr>
<th>Thoracic</th>
<th>Pelvic</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left</td>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td>Ataxia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paresis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Circling</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Backing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head elevated</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tail pull</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elevation change</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Hyperesthesia: _______________________________________________________

Problem list: _______________________________________________________

Localization: _______________________________________________________

DDx: _____________________________________________________________

Plan: _____________________________________________________________

Ataxia Grading*
0 - No deficits
1 – Deficits at normal gait, worsened by backing, turning, swaying, neck extension
2 – Deficits easily observed at normal gait and worsened by backing, turning, swaying, neck extension
3 – Deficits obvious when walking and tendency to buckle or fall when backing, turning or neck extension
4 – Stumbling, tripping and falling spontaneously
5 – Recumbent

Fig 1: Example equine neurological examination form (*from Veterinary Neuroanatomy and Clinical Neurology Eds: A. de Lahunta, E. Glass).

Thoracic wall ascends the contralateral cervical spinal cord to the medulla oblongata. Direct stimulation of the vagus nerve results in contralateral efferent recurrent laryngeal nerve stimulation and cricoarytenoideus lateralis contraction (Newton-Clarke et al. 1994). Spinal cord injury along the cranial thoracic and cervical segments can influence the thoracolaryngeal reflex; however the sensitivity of this reflex is generally low (Greet et al. 1980; Newton-Clarke et al. 1994).
The cutaneous trunci reflex can be triggered by tapping a blunt instrument along the dorsolateral dermis from the tuber coxae cranial to the shoulder. Afferent stimulus is carried via spinal nerves to spinal cord motor neurons cranial to the C8–T1 segments and the efferent response is carried via the lateral thoracic nerve to the cutaneous trunci muscle (DeLahunta 2009). The author typically performs this test from caudal to cranial with healthy caution given the unpredictable nature of any animal suddenly poked in the side, no less a horse. If the caudal portion of this test is intact, there is no need to carry on cranially; the reflex arc is intact, although segmental sensory loss is possible following spinal nerve/nerve root injury. Regional loss of this reflex supports thoracic SCD or brachial plexus/lateral thoracic nerve injury. This is not a reliable test in the recumbent horse. The perineal reflex and tail and anal tone are evaluated as the examination is carried caudally assessing the LMN reflex arcs of the cauda equina spinal nerves. Patellar and withdrawal reflexes are limited to the recumbent horse.

**Locomotor examination**

Examination for appropriate locomotion is performed by challenging the nervous system and observing the response. The inherent subjectivity leads to controversy when interpreting or comparing observations (Olsen et al. 2014). Spinal cord pathology is localised and interpreted in light of single etiology. This review is not all-encompassing; rather the following are basic guidelines to performing the locomotor examination that the author uses. Portions of the examination may be omitted for safety of horse and handler or due to limited additive value to neurolocalisation. Positioning of the limbs when a horse is at rest, while walking and when stopped is noted throughout the examination. Most horses with ataxia will show a wide base stance and fail to position their limbs squarely under the trunk when movement is stopped.

A slow walk on a solid even surface improves the consistency of neurological examination. Upper motor neuron SCD can be compensated for with speed in most quadrupeds as central pattern generators within local reflex arcs of the spinal cord maintain the gait (MacKay-Lyons 2002). The goal is to assess different general proprioception and efferent motor responses between the limbs, spinal cord and brain. Normal feedback mechanisms allow steady, refined and coordinated limb movement. Hence, slow and consistent examination is needed to differentiate neurological from orthopaedic pathology. If severe lameness is observed during the walk, further neurological testing will be influenced by compensatory mechanisms in the gait, stride and flight of the limbs. Re-examination is advised following regional or intra-articular anaesthesia. Minor lameness is prevalent in general practice and needs to be taken into consideration throughout the locomotor examination (Furr and Reed 2008).

Most neurological dysfunctions can be identified with the horse being led by hand. Ridden examinations can be used to exacerbate subtle clinical signs, but might not be essential for neurological evaluation. The appearance of a pacing gait in non-characteristic breeds is a significant change suggesting ataxia. Other supporting features of SCD (UMN) are a hypermetric gait and spasticity due to hyperexcitable extensor motor neurons (Edgerton and Roy 2010; Kurian et al. 2011).

**Line walking and backing**

Opinion differs on the best observation point when assessing the walk. The author prefers to observe the horse being led away from and towards the examiner followed by lateral observation if necessary. Hallmarks of SCD are abnormalities of hoof placement and arc, toe-dragging, coordination of limb movement, truncal sway and the rhythm of the gait (diagonal vs. pacing gait). Toe-dragging while backing is a compensatory sign of weakness and decreased proprioception without the ability to visually compensate for limb placement. In severe cases of SCD, horses might refuse backing, although novice horses may resist this from lack of training. Thoracic and pelvic hooves will interfere from lack of appropriate pelvic limb placing. Some normal horses will toe-drag when backing without obvious SCD, further supporting restraint when interpreting a single clinical observation. Observe the Supplementary item of a horse showing general proprioceptive ataxia in all limbs (Supplementary item 2).

**Circling**

Tight circling of a horse in lead is preferred over the typical wide circling employed during the lameness examination, which forces limb coordination and exaggerates neurological dysfunction. The pelvic limb or limbs will abduct widely in cases of SCD (Fig 2) with inconsistent hoof placement following the cranial swing phase of stride, referred to as circumduction, and a common sign of abnormal general proprioception. Spastic UMN paresis can contribute to exaggerated flexion of the limb during circumduction. Horses with severe SCD might be reluctant to circle and pivot on their pelvic or thoracic limbs with their neck or body rigidly fixed (Supplementary item 3). With severe deficits, crossing over of the thoracic limbs may occur. Severe orthopaedic disease might result in similar findings and repeating the circling following intra-articular anaesthesia is recommended.

**Tail pull**

The tail pull is probably the most subjective test of appropriate postural reactions. The intent is to pull the horse laterally via the tail during walking, either with the ipsilateral pelvic limb in the upper phase of stride or in weightbearing. Tail pulling can be constant or intermittent depending on examiner preference. Most horses will give to the tail pull with an ipsilateral sway of the pelvis for the first one or 2 strides, but should compensate to resist pulling in subsequent strides with the ipsilateral pelvic limb. Horses with SCD are much more prone to this compensatory sign of weakness and decreased proprioception without the ability to visually compensate for limb placement.

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Fig 2: Pelvic limb circumduction in a Quarter Horse filly with a C2–3 vertebral malformation resulting in compressive myelopathy.
pushing on the horse coordination. The same response can be evaluated by observation, since it is a measure of strength and by the individual pulling the tail rather than a distant may have no additive value. The tail pull test is best assessed during the previous locomotor examination, then the tail pull often short-strided and choppy in response to elevation of ground shows excessive lateral movement as the limb is in search of a ground contact point. The pelvic limb gait is described as floating. This is in contrast to the inconsistent hoof placement, hypermetria and severely delayed cranial phase of stride in a horse with a cervical SCD observed in Table 1: Differential diagnoses of common causes of ataxia in the horse

<table>
<thead>
<tr>
<th>Differential diagnosis</th>
<th>Age</th>
<th>Clinical course</th>
<th>Localization</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Structural</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malformation/malarticulation</td>
<td>&lt;2 years, any</td>
<td>Acute/chronic, progressive</td>
<td>C1–T2</td>
<td>UMN tetraparesis</td>
</tr>
<tr>
<td>CVSM</td>
<td>Young, adult</td>
<td>Chronic, progressive</td>
<td>C1–T2</td>
<td>UMN tetraparesis</td>
</tr>
<tr>
<td>Vertebral fracture/luxation</td>
<td>Any</td>
<td>Acute, nonprogressive</td>
<td>Any</td>
<td>UMN tetraparasiosis</td>
</tr>
<tr>
<td>Occipitoatlantoaxial</td>
<td>Young</td>
<td>Chronic, progressive</td>
<td>C1–2</td>
<td>UMN tetraparesis</td>
</tr>
<tr>
<td>malformation/luxation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Embolic myelopathy</td>
<td>Adult</td>
<td>Acute, nonprogressive</td>
<td>C1–T2, T3–L3</td>
<td>UMN tetraparasiosis</td>
</tr>
<tr>
<td>Infectious / Inflammatory</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post anaesthetic myelopathy</td>
<td>Adult</td>
<td>Acute, nonprogressive</td>
<td>T3–L3</td>
<td>UMN tetraparesiosis</td>
</tr>
<tr>
<td>Epidural haemorrhage</td>
<td>Any</td>
<td>Acute, nonprogressive</td>
<td>EHM – T3–L3 WNV, rabies – any</td>
<td>Any</td>
</tr>
<tr>
<td>Viral myelitis – EHM, WNV, rabies</td>
<td>Adult</td>
<td>Acute, progressive</td>
<td>Any</td>
<td>UMN tetraparesiosis</td>
</tr>
<tr>
<td>Bacterial/vungal myelitis</td>
<td>Any</td>
<td>Acute/chronic, progressive</td>
<td>Any</td>
<td>Any</td>
</tr>
<tr>
<td>Parasitic – EPM, Parelaphostrongylus tenuis, Halicthys tuberculatus spp., Hypoderma spp.</td>
<td>Adult</td>
<td>Chronic, progressive</td>
<td>Any</td>
<td>Any</td>
</tr>
<tr>
<td>Polyneuritis equi</td>
<td>Adult</td>
<td>Chronic, progressive</td>
<td>L4–S2</td>
<td>LMN paraparesis</td>
</tr>
<tr>
<td>Neoplasia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Discospondylitis/osteomyelitis</td>
<td>Any</td>
<td>Chronic, progressive</td>
<td>Any</td>
<td>Any</td>
</tr>
<tr>
<td>Primary, skeletal, haematopoietic, metastatic</td>
<td>Adult</td>
<td>Chronic, progressive</td>
<td>Any</td>
<td>Any</td>
</tr>
<tr>
<td>Degenerative</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDM/neuroaxonal dystrophy</td>
<td>Any</td>
<td>Chronic</td>
<td>Any</td>
<td>UMN tetraparesis, no ataxia</td>
</tr>
<tr>
<td>EMND/vitamin E deficiency</td>
<td>Adult</td>
<td>Chronic</td>
<td>Generalised</td>
<td>LMN tetraparesis, no ataxia</td>
</tr>
</tbody>
</table>

Descriptions of disease represent common presentations and do not account for all possible manifestations. UMN, upper motor neuron; LMN, lower motor neuron; CVSM, cervical vertebral stenotic myelopathy; EHM, equine herpesvirus myeloencephalopathy; WNV, West Nile virus; EPM, equine protozoal myeloencephalitis; EDM, equine degenerative myelopathy; EMND, equine motor neuron disease.

easier to pull ipsilaterally, have a delayed or absent compensatory resistance to the pull and occasionally interfere with contralateral limbs. Horses with severe UMN paresis may lose their footing or fall with moderate tail pulling. If the horse is showing significant signs of pelvic limb ataxia during the previous locomotor examination, then the tail pull may have no additive value. The tail pull test is best assessed by the individual pulling the tail rather than a distant observation, since it is a measure of strength and coordination. The same response can be evaluated by pushing on the horse’s shoulder and trunk at rest or when walking. Careful examination of the tail pull and truncal sway during rest and during movement can help differentiate UMN from LMN paresis. Reasonable strength at rest that worsens when walking supports UMN paresis (Furr and Reed 2008).

Head extension
Raising the head while walking removes visual compensation of thoracic limb placing and alters vestibular/proproprioeptive influence on gait, increasing the dependence on proprioception and motor integration. Mild thoracic limb hypermetria and delayed cranial phase of stride is expected in clinically normal horses while walking with the head raised, described as floating. This is in contrast to the inconsistent hoof placement, hypermetria and severely delayed cranial phase of stride in a horse with a cervical SCD observed in Supplementary item 4 and 5 [real time and slow-motion]. Observation of the thoracic hooves prior to contact with the ground shows excessive lateral movement as the limb is in search of a ground contact point. The pelvic limb gait is often short-strided and choppy in response to elevation of the head. Horses with SCD might assume a pacing gait while walking with the head in an elevated position, although this is not pathognomonic.

Curb testing
Altering ground height can test for compensatory mechanisms and adaptation of the proprioceptive and motor responses. Curbs of 15–25 cm work well. Barring any underlying lameness, most horses will place their limbs with intent on to the higher surface. Horses with a SCD may assume hypermetric pelvic limb movements when stepping on to an elevated surface followed by stumbling or falling when stepping down. The best observation to make is whether or not the horse is placing his hooves consistently and intentionally during this test. Any deviation or variability in hoof placement should raise suspicions of SCD.

Incline/decline walking
Walking a horse up and down an incline can further assist in identifying evidence of SCD. Neurological dysfunction is often exaggerated during the decline, but might be observed during the incline, with asymmetric abduction of the pelvic limbs, toe-catching and knuckling, falling, limb interference and reluctance to walk in a straight line downhill.

Postural reaction/hopping
Several tests can be used in the stationary horse to assess spinal cord function. The author prefers to perform these tests following the locomotor testing as they may be omitted depending on the severity of SCD present. Thoracic and pelvic limb placement tests are performed by placing a limb
crossed over, either cranial or caudal, the opposing limb. The time it takes for the abnormal limb position to be recognised and appropriately placed in a natural position is noted. Long delays or absent recognition of the crossed limb are abnormal. This suggests a delay in proprioception communication pathways anywhere from the receptor to the motor cortex of the brain. Abnormal limb placement can also be observed in horses with LMN dysfunction from peripheral nerve injuries. A well trained clinically normal horse may tolerate abnormal hoof placement.

Single limb hopping responses are valuable at identifying postural reaction deficits. Hopping can be performed last or omitted if substantial evidence of SCD is previously identified. The author finds the hopping response is a valuable means of investigating postural reaction deficits in the horse. Hopping is best performed on soft footing since some horses may fall if sufficient deficits are present. Perform the test by holding one thoracic limb off the ground while simultaneously pushing the cranial neck contralaterally. Use of a reference point (point-of-the-shoulder) is ideal to determine the extent of trunk movement prior to hopping on the contralateral limb. Repeat the test on the contralateral limb to compare symmetry. Horses with severe SCD may fail to generate a hop, while the test on the contralateral limb to compare symmetry. Following appropriate neurolocalisation, a group of reasonable differential diagnoses can be considered. Table 1 summarises the differential diagnoses of common causes of ataxia in the horse. Fig 3 is a neurolocalisation chart for a mature horse presenting with ataxia.

Cervical spinal cord segments C1–T2
Spinal cord dysfunction of the cervical spinal cord segments (C1–C5) results in UMN tetraparesis and general proprioceptive ataxia in all limbs. The severity of ataxia may appear more severe in the pelvic limbs and subtle in the thoracic limbs. Expected clinical observations include postural reaction and placing deficits of all limbs, truncal

Interpretation

Neurolocalisation
Assuming normal mentation and that a cranial nerve examination has sufficiently excluded intracranial pathology, neurolocalisation of SCD can be regionalised to the following spinal cord segments: cervical (C1–T2); thoracolumbar (T3–L3); and lumbosacral (L4–S2). Following appropriate neurolocalisation, a group of reasonable differential diagnoses can be considered. Table 1 summarises the differential diagnoses of common causes of ataxia in the horse. Fig 3 is a neurolocalisation chart for a mature horse presenting with ataxia.

Fig 3: Neurolocalisation chart for a mature horse presenting with ataxia. This depicts a rough template of differential diagnoses, however all disease variances are not accounted for. *Viral, bacterial, fungal and verminous encephalitis. **Anxious behaviour changes may occur with acute peripheral vestibular dysfunction. EPM, equine protozoal myeloencephalitis; EHM, equine herpesvirus myeloencephalopathy; CVA, cerebrovascular accident; CVSM, cervical vertebral stenotic myelopathy; EDM, equine degenerative myelopathy; NAD, neuroaxonal dystrophy; VFL, vertebral fracture-luxation; THO, temporohyoid osteoarthropathy; UMN, upper motor neuron. 

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sway when ambulating, a weak tail pull response, interfering when circling and inconsistent hoof placement on curbs and an incline/decline. The ataxia may have an exaggerated or hypermetric quality (spastic) in all limbs, especially in young horses. Disuse atrophy may be observed along the epaxial and gluteal muscles with chronic disease.

Caudal cervical SCD (C5–T2 segments) can result in LMN dysfunction of the thoracic limbs and UMN dysfunction of the pelvic limbs. Asymmetric thoracic limb LMN deficits can be misinterpreted as orthopaedic lameness. In these situations, the presence of pelvic limb ataxia and paraparesis support a neurogenic suspicion of thoracic limb dysfunction. However, if no other evidence of SCD is present, exclude orthopaedic disease first with any suspected asymmetric thoracic limb LMN deficits. The prevalence of orthopaedic disease affecting a single thoracic limb far supersedes that of neurogenic disease, especially if the horse is bearing weight. Single limb hopping tests can have value in cases of subtle SCD. Monoparesis as a sole clinical sign due to spinal cord injury is extremely rare and limited to focal myelopathies (embolism, protozoal myelitis, neoplasia etc.). Homer’s syndrome and unilateral body sweating can be observed with cervical SCD.

**Thoracolumbar spinal cord segments T3–L3**

Thoracolumbar SCD results in normal thoracic limb function and UMN paraparesis and pelvic limb general proprioceptive ataxia. Compared with cervical SCD, thoracolumbar UMN paraparesis and pelvic limb general proprioceptive ataxia. Specific observations include inconsistent pelvic limb hoof placement, interfering of the pelvic limbs, weak tail pull response and tripping or stumbling consistently on curb testing and when walked on a decline. These horses will often ‘weathervane’ when cirkled, showing little effort to coordinate the pelvic limbs. Urinary incontinence characterised by urine dribbling may be observed. Differentiating cervical from thoracolumbar SCD in the recumbent horse is challenging and may require sling support to identify postural reaction deficits. Upper motor neuron paraparesis should be differentiated from LMN paresis. Defining features separating UMN from LMN paresis should be differentiated from LMN paresis. Absence of the cutaneous truncal reflex (bilaterally suggests LMN pathology of C8–L3 spinal cord segments or associated spinal nerves) and intact extensor postural reactions (ability to support weight on the limb). Patellar hyperreflexia may be present in recumbent horses.

**Lumbosacral spinal cord segments L4–S2**

The lumbosacral intumescence and terminal spinal cord (conus medullaris) are located within the fourth lumbar and second sacral vertebrae in most mature horses. Thus SCDs cranial to the L4 vertebra cause general proprioceptive ataxia and UMN tetra-paraparesis. Injuries caudal to the L4 vertebra result in LMN deficits from dysfunction of the neuron cell bodies (spinal cord) or spinal nerves. LMN spinal reflex testing of the limbs has limited value in the ambulatory horse, but can be applied to recumbent horses, foals and Miniature Horses. The patellar reflex is the most repeatable LMN spinal reflex (DeLahunta 2009). Horses can show signifcant patellar hyporeflexia after prolonged periods of recumbency, not to be confused with areflexia, an indication of severe LMN dysfunction. Polyneuritis equi (cauda equina neuritis) affects the spinal nerves of the lumbosacral and coccygeal segments causing LMN deficits recognised as loss of cutaneous sensation, perineal reflex, weak tail/anal tone and urinary incontinence.

**Author’s declaration of interests**

No conflicts of interest have been declared.

**Ethical animal research**

Ethical review not applicable for this review article.

**Source of funding**

None.

**References**


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