EQUINE VETERINARY EDUCATION/AMERICAN EDITION

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APRIL 2023

EQUINE VETERINARY EDUCATION

American Edition | April 2023

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IN THIS ISSUE:

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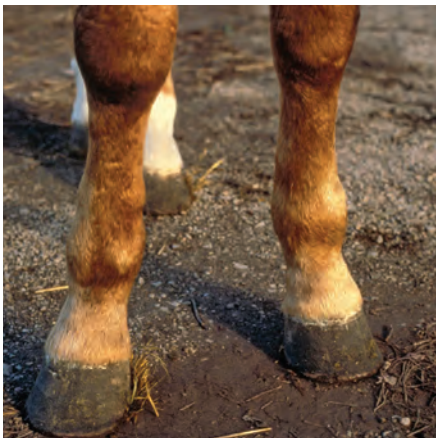

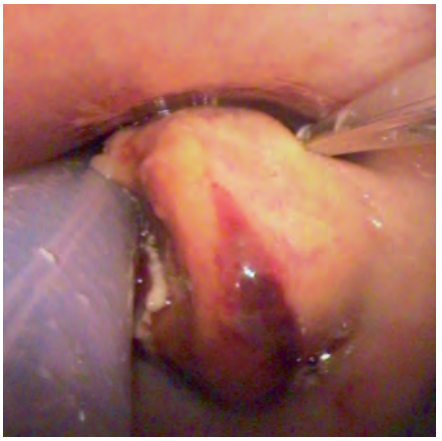

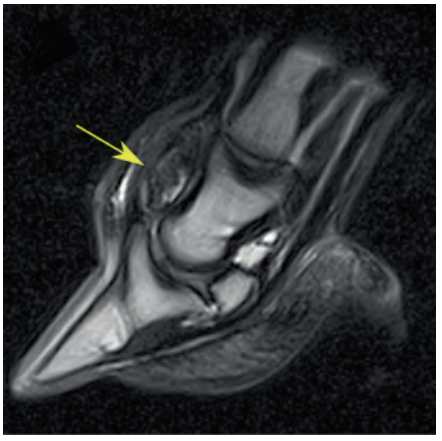

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Equine Veterinary Education is a refereed educational journal designed to keep the practicing veterinarian up to date with developments in equine medicine and surgery. Submitted case reports are accompanied by invited reviews of the subject (satellite articles) and clinical quizzes. Tutorial articles, both invited and submitted, provide in-depth coverage of issues in equine practice.

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Empowering our new colleagues: Ethical considerations for the modernization of equine internships

By Caitlin O'Shea, DVM, MS, DACVS-LA, CVA and Jackie Christakos, DVM

Dr. Caitlin O'Shea

Dr. Jackie Christakos

There is much ongoing discussion about attracting and retaining equine practitioners as well as the paucity of applications for equine internship programs. There are multiple reasons for this, including financial constraints and generational differences in learning styles and lifestyle goals. While the reasons for this trend are multifactorial, what new thought processes can practices embrace to help shift this trajectory?

Traditionally, many equine internships have not been run as structured programs, but rather as apprenticeships. These practices depend on individuals being eager to invest sweat equity and, in some cases, overwork themselves, emulating those who they worshiped as gods of equine practice. The model relies on learning by observation and absorption of information with little expectation of positive reinforcement of the apprentice. Many of us have experienced and accepted this style as typical; in today's world, however, it is both ineffective and questionably ethical.

Our new colleagues entering equine practice today have had very different cultural and formative experiences. These experiences, magnified by technology and a constant stream of information, have instilled unique strengths, including an inclination to challenge the status quo. Questioning the methods that may contribute to our profession's historically high level of burnout and mental health issues makes sense regardless of career stage.

In a recent American Association of Veterinary Medical Colleges (AAVMC) survey of veterinary interns and residents, organizational culture appeared to be a large contributor to the wellbeing of early-career veterinarians, along with the quality of mentorship offered amongst other factors.[1] Perhaps a starting goal is promoting a practice culture that encourages open modeling of our humanity. Mentors can commiserate that we struggle with imposter syndrome even once established in our careers. We wrestle with difficult cases but work through them using quality research and tapping of reputable sources. Mental health is a continuum that most of us are journeying through daily. Through the display of humility, humanity and honesty, we help give mentees and interns the skills and space to process and meet many challenges in the future, understanding adversity will continually arise but can be overcome.

AAEP TRANSFORMING EQUINE PRACTICE

Fair and humane working hours should be promoted as the new normal for all members of equine practice. AAVMC guidelines can be utilized as a reference for setting fair expectations for both practice and intern.[2] Sleep deprivation leads to medical errors and safety concerns, as well as promoting a poor environment for learning. It is the expectation that interns will often work longer hours than what is typically expected of an associate to maximize the learning experience over a short period of time. Consider the analogy of the prospect horse in training—all the potential in the world, but limited experience requiring greater repetition for optimum performance in a short time frame. More seasoned horses may perform at a high level with less time spent training, as they have years of previous experience and skill. However, appropriate scheduling for allowance of rest and recuperation is essential for both young horses and intern doctors to avoid a shortened career span. Regardless of the schedule adopted, the goal should be to match the needs of the practice and the intern doctor concurrently while maintaining reasonable terms for both.

Mentors must learn to better utilize interns, making them a professional part of our team from the beginning. Why should we support stress and unnecessary toiling if a better way of learning and gaining experience is possible? If we have fewer internship applicants and fewer interns, we can still run an efficient practice by changing our model. Consider that additional support staff and better use of existing skilled technical staff could be promoted as a viable alternative. This enables interns to place more emphasis on learning the art of medicine both practically and emotionally while leaning on staff who have ongoing knowledge of hospital operations and culture. Internships should above all be an educational experience.

Effective mentoring certainly goes both ways. Both mentor and mentee need to be willing to rise to the challenge and actively work to accept and incorporate feedback. Interns

continued on next page

AAEP ETHICAL PRACTICE Every Day-Every Time

Dr. O'Shea is an assistant clinical professor at Mississippi State University College of Veterinary Medicine and serves on the AAEP's Professional Conduct and Ethics Committee. Dr. Christakos is a partner at Littleton Equine Medical Center in Littleton, Colo., and serves on the AAEP board of directors and as co-chair of the AAEP's Internship subcommittee.

5 things to know about AAEP this month

1. Acquire updated treatment protocols for Equine Viral Arteritis with revised AAEP guidelines for this important systemic disease at aaep.org/document/equine-viral-arteritis.
2. Claim one of the remaining spots at Focus on Podiatry, June 22–24 in Lexington, Ky., and expand your repertoire with foot-related lameness. Register at aaep.org/meetings.
3. With its 2023 allotment, the UHVRC has provided over 53,000 doses of core vaccines for horses in need since its inception in 2008.
4. U.S. members: Administer compassionate service to horses without incurring financial stress by signing up for Vet Direct Safety Net at aaep.org/vet-direct-safety-net.
5. Researchers: Grant proposals for both the Young Investigators and the Innovation and Discovery Research Grants are due May 31. Learn more at foundationforthehorse.org.

Botulism, social license on tap for Virtual Round Tables in May

Log on for education and engagement as the AAEP's Virtual Wednesday Round Tables roll on in May, featuring a session devoted to practical takeaways from the outbreaks of botulism that were linked to the deaths of at least 20 horses in Louisiana and 28 more in other states this winter.

Each month's Round Tables feature one clinical and one non-clinical topic and are held on the second and fourth Wednesday of every month through October. The sessions are free for members; simply register in advance through AAEP Anywhere at aaepanywhere.org.

Following is the upcoming schedule of sessions through May:

April 26	Emergency Coverage
May 10	Lessons Learned from Recent Botulism Outbreaks
May 24	Social License to Operate

If unable to attend a live 90-minute Round Table, you can watch a recording of the session on-demand through AAEP Anywhere, the association's free-to-members online learning platform. On-demand sessions are available approximately 48 hours following the live session and include mentioned resources such as PowerPoint slides, images and more. CE credit is not offered for the Round Tables.

The AAEP thanks its Virtual Wednesday Round Table sponsors:

Empowering our new colleagues, continued

and mentees must communicate their experience clearly but respectfully. Prompt and appropriate communication when errors are made and/or insecurities arise are critical and part of the learning process. It is very difficult for mentors to effectively train and support the growth of intern doctors without open communication. On both sides, the tone of any communication can make a big difference in whether the message is heard and internalized or taken as undue criticism.

Athletes commonly seek a coach who will help them maximize their skills and thrive in a demanding environment. Interns and mentors should remember that, ideally, a similar dynamic exists in internship programs as well. Adaptive resilience training by design is not always comfortable, but progressively enables us to be more calm and effective dealing with stressful situations. Internship programs should be consciously structured to meet specific goals outlined at the beginning, and with regular check-in meetings to stay on track. Practices may consider utilization of competency-based training and education models to help facilitate true growth and more productive review processes.

Though some reading this article may have convinced themselves otherwise, the internship process wasn't entirely comfortable for many "back in the day." Consider all of those who have left equine practice over the years for similar stresses. Equine practice is inherently demanding but incredibly rewarding. Is our level of discomfort really a good measure of how dedicated we are? Let's start promoting a modern era of ethical, fair internship standards and continue to provide excellent training for more than just the toughest few.

References:
1 AAVMC Veterinary Resident and Intern Wellbeing Study, 2020
2 AAVMC Guidelines for Veterinary Intern & Resident Wellbeing, 2022

Nominate a difference maker for an AAEP award

Recognize professional excellence by nominating a colleague for a 2023 AAEP award. The nomination deadline is June 1, and winners will be announced and recognized during the President's Luncheon at the AAEP's 69th Annual Convention in San Diego, Calif., Nov. 29–Dec. 3.

Nominations are being accepted in the following categories:

AAEP Research Award
Distinguished Educator – Academic Award
Distinguished Educator – Mentor Award
Distinguished Life Member Award
Distinguished Service Award
George Stubbs Award
Sage Kester Beyond the Call Award
The Lavin Cup (The Equine Welfare Award)

Visit aaep.org/about-aaep/annual-awards for nomination forms as well as additional information about each award and the selection process. You may also request a nomination form from Sue Stivers at sstivers@aaep.org or (859) 233-0147.

Dr. Nat White accepts the 2022 Sage Kester Beyond the Call Award from Dr. Emma Read during the 68th Annual Convention in San Antonio, Texas.

Updated EVA Guidelines now available on AAEP website

The AAEP has updated its Equine Viral Arteritis (EVA) Guidelines to amend the treatment protocols for this infrequently diagnosed but important systemic disease that continues to impact trade by restricting the export of carrier stallions and virus infective germ plasm.

EVA is a contagious viral disease of equids in which widespread vasculitis may result in non-immune individuals, leading to fever, peripheral edema, pneumonia and abortion. Infection can vary from inapparent to fulminant clinical disease in young foals. Mortality is rare in otherwise healthy adult horses.

No specific antiviral treatment for EVA is currently available. Practitioners should observe the following treatment recommendations:

- Supportive therapies are indicated in moderate to severe cases of the disease, and specifically important in clinically affected stallions.
- Elimination of a carrier state is problematic. Because the virus is shed in semen, castration is the only reliable method for elimination of the career state.
- Non-surgical strategies such as the use of GnRH antagonist or anti-GnRH vaccines, may facilitate clearance in some stallions, but these methods are not fully validated and may have deleterious effects on libido and sperm production.

Dr. Peter Timoney

Dependent edema (or "stocking up") of lower extremities of both fore limbs of a horse acutely affected with EVA.

"Capable of being transmitted by respiratory and venereal routes, equine arteritis virus has the potential to cause extensive outbreaks of abortion, deaths in young foals, and the carrier state in stallions," said guidelines reviewer Dr. Peter Timoney, professor emeritus at the University of Kentucky's Maxwell H. Gluck Equine Research Center. "A safe and effective vaccine is available for immunizing stallions and non-pregnant mares."

View the updated EVA Guidelines or save them to your tablet or mobile device at aaep.org/document/equine-viral-arteritis.

ASSOCIATION

UHVRC awards 7,000 vaccine doses and microchips for horses in need

This Old Horse, a Hastings, Minn.-based provider of rescue, retirement and recovery support for older and special needs horses through their farms and foster network, is among 223 nonprofit aftercare facilities to receive complimentary vaccines and microchips through the Unwanted Horse Veterinary Relief Campaign (UHVRC).

"Bunny," a 27-year-old American Paint Horse who was a successful show horse and broodmare until her retirement, is among the equine residents at This Old Horse to benefit from Merck Animal Health vaccines and microchips through the UHVRC.

MERCK Animal Health

The longstanding partnership between Merck Animal Health and the AAEP provided 7,000 doses of both Prestige® 5 + WNV and EquiRab, to protect horses against Eastern and Western equine encephalomyelitis, equine rhinopneumonitis (EHV-1 and EHV-4), West Nile virus, equine influenza, tetanus and rabies; and 7,000 of the company's HomeAgain® TempScan® Microchips. Since inception of the UHVRC in 2008, Merck Animal Health has generously provided more than 53,000 doses of core vaccines valued at over $1 million.

"On any given day, we support more than 200 horses in our facilities, all of them requiring regular vaccination as part of their ongoing health maintenance," said Nancy Turner, founder and president of This Old Horse. "Microchipping plays a big role in our safety net program to assure that any of 'our' horses placed in adoptive homes can always find their way back home if their circumstances change. The generous award of vaccines and microchips will reduce our health and safety expenses for each horse, enabling the savings to be reallocated to help horses in other ways."

Dr. Dale Magnusson of Magnusson Veterinary Services in Hudson, Wisc., coordinated This Old Horse's application for free vaccines and microchips. The annual application deadline is Feb. 1. Learn more at aaep.org/horse-owners/unwanted-horse-veterinary-relief-campaign.

CONTINUING EDUCATION

Practical solutions to clinical challenges: 2023 AAEP CE opportunities

June 22–24, 2023

Focus on Podiatry

Spy Coast Farm • Lexington, Ky.

Sponsored by Antech & Sound, Contura Vet, Soft-Ride Deep-Gel Boots and Zoetis

Register at aaep.org/meetings

Nov. 29–Dec. 3, 2023

69TH ANNUAL CONVENTION

San Diego Convention Center
San Diego, Calif.

Registration opens summer 2023

Oct. 6–8, 2023

Foundational Skills in Equine Practice

Spy Coast Farm • Lexington, Ky.

Sponsored by Arenus Animal Health, CareCredit, Contura Vet, Dechra Veterinary Products and Merck Animal Health

Registration opens summer 2023

Every Day

AAEP Anywhere Online CE

aaepanywhere.org

Pick up an hour or two of free CE at your convenience. Log in with your AAEP website credentials.

New Practice Life episode offers first-hand look at veterinary care in Ukraine

Acquire an on-the-ground perspective of veterinary work in war-torn Ukraine and in other regions of the world during the February episode of the AAEP Practice Life podcast as co-hosts Dr. Mike Pownall and Dr. Jessica Dunbar chat with Dr. Gemma Campling, founder and director of the non-profit charity Worldwide Vets.

Discussing the emotional challenges of administering care in Ukraine, Dr. Campling said it's not restricted to operations in the southeast region of the country where there's active fighting and her team is often confined to garages and underground cellars due to missiles landing nearby.

"You can be in the quiet areas in the northwest where there's no real military action yet every few nights you hear these horrendous haunting [missile] sirens going off, and you lie in bed and you just think 'OK, I really hope the anti-missile technology's got this.' Most of the time it does, and nothing comes through," she said.

Among the topics discussed are the logistics of setting up operations in a country, including the unique challenges of doing so in Ukraine; the current situation on the ground in Ukraine for horses, other animals and their owners; how she and her team manage the emotional challenges of operating in a war zone; how the AAEP, The Foundation for the Horse and others have supported their efforts in Ukraine; and Worldwide Vets' plans for 2023 in Ukraine.

For those interested in volunteering with Worldwide Vets in any of the 10 countries on four continents in which they operate, or if interested in supporting their ongoing work through donation of funds or equipment, visit worldwidevets.com.

Download or listen to the 27-minute episode at podcast.aaep.org or on iTunes.

Dr. Gemma Campling discusses the work of Worldwide Vets in Ukraine during the Opening Session at the 2022 AAEP Annual Convention.

The AAEP Practice Life podcast is sponsored by Boehringer Ingelheim.

Boehringer Ingelheim

CONTINUING EDUCATION

Become the foot expert your clients expect

Register now for space-limited Focus on Podiatry

Fix the pesky foot problems that hinder the health and performance of your clients' horses by acquiring a strong foundation of podiatry principles and techniques at the AAEP's Focus on Podiatry, June 22–24 at Spy Coast Farm in Lexington, Ky.

Offering more than 20 CE credits and capped at just 60 participants for optimal instruction and skills acquisition, Focus on Podiatry will be led by a select team of veterinary podiatrists and farriers. Pertinent lectures reinforced by hands-on sessions will expand your working knowledge of the foot, increase understanding of anatomy and biomechanics of podiatry issues, and develop skills to interpret degree of damage and devise mechanical solutions that speed healing and restoration of function.

After daily sessions, enjoy evening social events at which you'll establish collegial relationships with the instructors and your fellow attendees.

The AAEP-member registration rate is $945, and all spots are filled on a first-come basis. Register for the meeting, book your hotel and view the educational program at aaep.org/meetings.

Focus on Podiatry is sponsored by:

contura vet · Soft-Ride · sound · zoetis

Apply for research funding by May 31

Application window open for young investigators & innovation grants programs

The Foundation for the Horse is accepting grant proposals from AAEP members until May 31 for both of its equine research grant programs. One awards up-and-coming researchers and the other funds established investigators; both programs seek to help pioneer medical advances in equine health.

Young Investigators Research Grants

Graduate students, fellows and residents are eligible for up to $20,000 in funding for the study of key diseases and disorders affecting equine health. Pressing research topics and areas of special interest include musculoskeletal, gastrointestinal and metabolic disease; laminitis; factors to improve racing safety; and development of new technologies (i.e., stall-side tests). However, research applications on any topic will be considered.

Pilot studies or preliminary studies that are part of a major study or which will lead to a major project are of particular interest; small standalone projects will also be considered. Since this program's inception in 2019, The Foundation has awarded $436,784 for 23 projects at nine universities in support of exceptional science conducted by 20 emerging researchers.

Additional information, including application instructions, requirements and selection criteria, is available at foundationforthehorse.org/graduate-student-fellow-resident-research-grants.

Innovation and Discovery Research Grants

Individuals with a background in equine research and a previous record of research publication are encouraged to submit proposals focused on areas of equine medicine that will improve the science needed to elevate the health and welfare of the horse. A minimum of two projects will each receive up to $50,000 in funding in 2023.

Dr. Lauren Smanik was among the awarded young investigators in 2022 for her project, "A pilot study on an experimental model for Palmar Osteochondral Disease (POD) in horses." Her award was sponsored by Thoroughbred Education and Research Foundation.

While all topics will be considered for funding, particular interests include musculoskeletal, gastrointestinal, respiratory and endocrine disease. Lameness, laminitis, colic and development of diagnostic technology are areas also considered important and in need of investigation. Selected investigators will be strongly encouraged to publish their results in a refereed journal and/or submit abstracts for presentation at the AAEP Annual Convention.

In 2022, The Foundation awarded $190,933 in support of four projects in the first year of the Innovation and Discovery Research Grants program, which was made possible by a generous gift from Mrs. Penelope Knight and her Coyote Rock Ranch.

Visit foundationforthehorse.org/innovation-discovery-research-grants for additional information, including application instructions, requirements and selection criteria.

A Community that Cares *For Horses*

Established to unite everyone who is dedicated to improving the health and well-being of horses, The Foundation for the Horse provides support for horses in need, relevant research and continued education for future equine veterinarians.

JOIN US TODAY! Your gift will ensure horses in need get the care they deserve.

The Foundation For The HORSE

Learn more and support our efforts at
foundationforthehorse.org

Dr. Jay Humburg, decorated and respected educator, dies at 89


Dr. Jay Humburg

Dr. Jay Humburg, beloved veterinary educator known as "The Legend" by his thousands of students during his nearly five decades in academia in the U.S., Caribbean and Africa, died Feb. 3 at the age of 89.

After receiving his veterinary degree from Kansas State University in 1957, Dr. Humburg practiced large animal medicine in Broken Bow, Neb., until 1967 when joining a Kansas State project in Nigeria to serve as a professor while helping build a veterinary school in the African nation. He returned to the U.S. in 1973 as a professor of large animal surgery and medicine at Auburn University, where he served until 2000. Dr. Humburg moved to the Island of Grenada, where he helped develop the veterinary school and was among the first faculty at St. George's University, from which he received the 2014 Distinguished Service Award for his outstanding contributions.

Dr. Humburg was an AAEP Honor Roll member and served on the Abstract Review, Equine Insurance and Scholarship committees between 1987 and 1994.

Dr. Alan Nixon, equine research hall of famer, dies at 67


Dr. Alan Nixon

Dr. Alan Nixon, 2009 inductee into the University of Kentucky Gluck Equine Research Hall of Fame and 2015 recipient of the American College of Veterinary Surgeons Founders Award for Lifetime Achievement, passed away March 1. He was 67.

Dr. Nixon received his veterinary degree in 1978 from the University of Sydney in his native Australia. Following an internship in Sydney, Dr. Nixon moved to the U.S., where he completed a residency in large animal surgery at Colorado State University leading to ACVS board certification.

After five years at University of Florida, Dr. Nixon moved to Cornell University where he provided service in clinical orthopedics and neurosurgery, and served as chief of surgery from 2002–2006. He served as director of the Comparative Orthopaedics Laboratory and JD Wheat Equine Sports Medicine Laboratory at Cornell, and he was the inaugural chief medical officer at the Cornell Ruffian Equine Specialists private practice hospital.

Dr. Nixon's long career in musculoskeletal research focused on targeted cell and gene therapy for joint disease. He authored over 200 manuscripts as well as two books: *Equine Fracture Repair* and *Diagnostic and Surgical Arthroscopy of the Horse*. In addition, he served multiple terms on the AAEP's Research Committee in the 1990s.

Dr. Nixon continued to work as an emeritus professor at Cornell following his retirement in 2020 and also served as an adjunct professor at the University of Florida.

Members in the News

John Eisele/CSU Photography

Dr. Yvette Nout-Lomas

Dr. Yvette Nout-Lomas selected for endowed professorship

Dr. Yvette Nout-Lomas, an associate professor of equine internal medicine at Colorado State University, has been selected as one of two inaugural holders of the Gordon and Joan Bishop Professorship, the first endowed professorship ever provided through the office of the provost.

Dr. Nout-Lomas received her veterinary degree from Utrecht University in the Netherlands and is board-certified in both veterinary internal medicine and veterinary emergency and critical care. She served on the AAEP's Educational Programs Committee from 2017–2019.

Dr. Christina Wilson

Dr. Christina Wilson named business partner of the year

Dr. Christina Wilson, owner of Equiheart Veterinary Services in Califon, N.J., has been named the Hunterdon County Vocational School District's 2022 Business Partner of the Year.

A veterinary graduate of the University of Illinois, Dr. Wilson is the equine and small ruminant veterinarian for the animal science program at the county's Polytech Career and Technical School and also serves on the program's advisory committee. She volunteers many hours sharing her knowledge of large animal care and providing hands-on learning opportunities for high school students interested in an animal husbandry career.

Dr. Barrie Grant

Dr. Barrie Grant appointed to California Veterinary Medical Board

California Gov. Gavin Newsome has appointed AAEP Honor Roll member and equine consulting practice owner Dr. Barrie Grant to the Veterinary Medical Board.

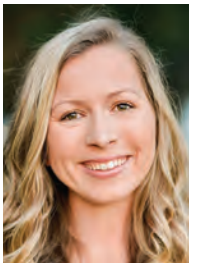
Recipient of the AAEP's Distinguished Educator – Mentor Award in 2013, Dr. Grant served as a professor of equine surgery at his alma mater Washington State University from 1974–1991, when he joined San Luis Rey Equine Hospital as an equine surgeon and became co-owner in 1995. He left in 2008 to start an equine consulting practice. Dr. Grant served on the AAEP board of directors from 1999–2001 and on the Educational Programs, Foundation Advisory, Research and Sports Medicine committees.

Promising equine researchers receive career development awards

Drs. Shune Kimura and Bethanie Cooper have been awarded $20,000 career development awards from Grayson-Jockey Club Research Foundation.

Dr. Shune Kimura

Dr. Kimura, a Ph.D. student at the University of Georgia and 2017 veterinary graduate of Tuskegee University, received the Storm Cat Career Development Award for his research entitled, "Metabolic Responses in Systemic Inflammatory Response Syndrome (SIRS) Impact Disease Severity, and Determine if Metformin Has Beneficial Anti-Inflammatory and Metabolic Effects in Equine SIRS."



Dr. Bethanie Cooper

Dr. Cooper, a Ph.D. student at North Carolina State University, from which she earned her veterinary degree in 2021, received the Elaine and Bertram Klein Career Development Award for her research entitled, "Myristoylated Alanine Rich-C-Kinase Substrate (MARCKS) Protein as a Therapeutic Target in Equine Asthma." Dr. Cooper was among the emerging researchers to receive Young Investigators Research Grants from The Foundation for the Horse in fall 2022.

In addition to Drs. Kimura and Cooper, five other AAEP members were among the primary investigators whose new equine research projects received grant funding for 2023:

Dr. Noah Cohen, Texas A&M University – *A VapA mRNA Vaccine for R. equi Pneumonia*

Dr. Carrie Finno, University of California, Davis – *Validation of Biomarkers for Equine Neurodegeneration*

Dr. Dale Kelley, Oklahoma State University – *Gallium Nitrate to Treat Bacterial Endometritis in Mares*

Dr. Lynn Pezzanite, Colorado State University – *Transcriptomic Response to Osteoarthritis*

Dr. Heidi Reesink, Cornell University – *Efficacy of Recombinant Equine Lubricin for Osteoarthritis*

AAEP Educational Partner Profile: Contura Vet

Contura Vet brings together bioengineering experience and veterinary expertise in the development of products for the treatment of osteoarthritis in animals.

Headquartered in London, Contura Vet develops and produces hydrogel products in our state-of-the-art, FDA- and GMP-approved facilities in Copenhagen, Denmark; the Contura Vet USA division was opened in Franklin, Tenn., in 2021.

Following success in human use with its unique, patented hydrogels, Contura International established Contura Vet with the development of Arthramid® Vet, a new class of injectable joint therapy for the treatment of arthritis in dogs and horses. Since 2009, Arthramid Vet has been used to treat non-infectious causes of joint lameness in animals, including both early and late stages of osteoarthritis (OA) and degenerative joint disease (DJD).

Arthramid Vet is a 2.5% iPAAG hydrogel that, over the past 10 years, has been safely and successfully used in over 90,000 horses to treat all stages of arthritis—from early-stage synovitis to late-stage osteoarthritis and degenerative joint disease.

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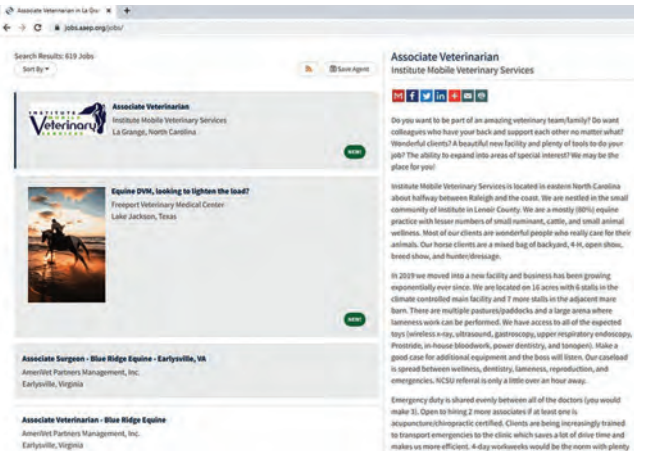

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[1] West Nile Virus Challenge Vaccine Efficacy, BI study number: V9 2009 WNV 12mo DOI

[2] Equine Influenza Challenge, BI study number: 01 V9 6mo DOI OH/03.

[3] Lack of Interference - Influenza Challenge, BI study number: 2012-001 Inf. Data on file at Boehringer Ingelheim.

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Equine Veterinary Education

RESEARCH HIGHLIGHTS

Highlights of recent clinically relevant papers

PERFORMANCE AFTER COLON SURGERY

This study by Veronica Edwards and co-workers in the United States evaluated the sales and race performance of juvenile Thoroughbreds with surgically corrected large colon displacements.

The medical, sales and racing records of 110 horses <2 years old with a surgical diagnosis of large colon displacement were examined. Surgical cases were compared with their maternal progeny (three maternal siblings close in age for each case; total control group n = 299) whose sales and racing data were evaluated.

There was no significant difference in median sale price between the two groups. Horses undergoing surgery had a reduced number of starts in the 2-year-old year (1 start) when compared with control horses (2.32 starts), but there was no significant difference over the 2- to 4-year-old period. There was no significant association with surgery on earnings within the 2- to 4-year-old period of racing when compared with controls.

These results suggest that if the juvenile Thoroughbred requires surgery for a large colon displacement, there is minimal association with sales price or race performance compared with their siblings.

NSD THRESHOLDS

This study by Erica Macon and co-workers in the United States, the United Kingdom and Australia aimed to determine if insulin dysregulated (ID) horses have thresholds for pure sources of starch and sugar above which there is an augmented insulin response. Identifying intake levels of nonstructural carbohydrates (NSC) that limit the postprandial insulinaemic response in the ID horse may help reduce hyperinsulinaemia-associated laminitis (HAL) risk.

Fourteen adult horses (six ID and eight non-insulin dysregulated, NID; matched for bodyweight) were fed eight dietary treatments in a randomised order. Dietary treatments were formulated using a base of low-nonstructural carbohydrate pellet (LNSC; 0.04 g of water-soluble carbohydrates [WSC]/kg bwt and 0.01 g of starch/kg bwt), to which pure sugar (dextrose) or starch (50:50 mix of waxy-maize and oat starch powder) sources were titrated to create diets with increasing amounts of either WSC (0.06–0.17 g WSC/kg bwt), or starch (0.03–0.1 g starch/kg bwt). Horses were fed each dietary treatment at a rate of 1 g/kg bwt once over 12 weeks. Serial blood samples were collected pre- and up to 240 min postprandially. Serum insulin concentrations were determined using a validated radioimmunoassay, and diet analytes were determined via wet chemistry. Statistical analysis was performed with a mixed-effect model. Positive incremental area under the curve for insulin (IAUCi) was calculated for all horses and dietary treatments.

There was no significant effect of diet on IAUCi within NID horses, but ID horses had an increased IAUCi for diets between 0.07 and 0.13 g of total NSC/kg bwt, depending on the starch and sugar composition, compared with the LNSC. IAUCi in ID horses were also significantly different to all NID for diets with NSC >0.1 g/kg bwt. Based on this study, using supplemental pure starch and sugar sources, ID horses seem to have a threshold for NSC of around 0.1 g/kg bwt/meal, above which significantly increased insulin responses are seen compared with NID horses.

SCHIRMER TEAR TESTS

This study by Eva Martín-Suárez and co-workers in Spain compared Schirmer tear test (STT)-1 results at 30 (STT30) vs. 60 (STT60) seconds in healthy horses.

STT-1 was performed in both eyes of 56 healthy horses, right eye first, and the wetting lengths were measured in STT30 and STT60. The reduction of the initial reflex phase was evaluated by measuring the wetting length velocity during the first 30 s. The effects of eye, age, weight, sex and ambient temperature and humidity on STT values were evaluated. Mean (standard deviation) STT30 and STT60 were 19.06 (3.88) and 24.26 (4.50) mm. There was a linear correlation between STT30 and STT60, expressed according to the following equation: $STT60 = 2.20 + 1.18 \times STT30$ ($P = 0.001$). STT30 or STT60 values did not vary between the sexes or correlate with age, weight, ambient temperature or humidity.

The authors concluded that STT30 allows for an accurate, reliable and applicable diagnosis of tear production compared with the standard STT60 value. The proposed method is shorter and may be a suitable alternative to the 1-min test.

EQUINE WELL-BEING

This study by Rebecca Smith and UK-based co-workers explored UK leisure horse-owners' perceptions of equine well-being.

In order to establish how UK leisure horse-owners use well-being-related terminology, four online focus group discussions (FGD) were held and qualitative data were collected. FGDs involved a semi-structured discussion, followed by a group activity to compare

seven equine well-being-related terms of interest introduced by the facilitator. The collected data were analysed using a constructivist grounded theory approach, and by content analysis, to examine the frequency and subjective meaning of the terms of interest.

The results showed that horse-owners did not clearly delineate between different terms, rather, they used the terms in the context of their own assessments of their horse. The meanings assigned to what horse-owners experienced with their horse were individual and subjective, shaped by past experiences, relationships with their animal, and peers or social groups. This individualised construction of equine well-being impacted on the meaning conveyed when using well-being-related terminology. This study extends the literature on equine well-being terminology usage, and highlights differences between the academic literature and the real-world experiences of horse-owners.

COMMUNITY-ASSOCIATED MRSA

This study by Aliya Abdulkadir and co-workers in Nigeria characterised and reported the prevalence of community-associated methicillin-resistant Staphylococcus aureus *(MRSA) in horses, dogs, cats and their human handlers.*

A cross-sectional study was conducted with 149 handlers and 446 animals (240 horses and 206 companion animals). The isolates were characterised as *S. aureus* and MRSA based on PCR detection of the *nuc*, *mecA* and *mecC* genes and the *pvl* gene for differentiation as community associated/livestock associated or hospital associated. The isolation rate of *S. aureus* from the human handlers' samples was 26 (17.4%) and 170 (38.1%) from the animal samples. The prevalence of MRSA among the isolates was 7 (4.7%) from the human handlers and 19 (4.3%) from the animals. The highest isolation rates were from dogs and dog handlers and were more likely to be colonised by *S. aureus* and MRSA compared with horses, cats and their handlers. The highest prevalence of MRSA was from horses (5.0%) and dog handlers (10.6%).

These findings show a high prevalence of community-associated MRSA in apparently healthy animals and their human handlers. This has important implications for antibiotic selection and use as well as infection control measures.

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Equine Veterinary Education

EDITORIAL

Sustainable development in equine anaesthesia

Abstract

Climate change is 'the biggest global health threat of the 21st century', and healthcare provision itself comes at an environmental cost. Volatile anaesthetic agents are potent greenhouse gases, but emissions arising from these waste anaesthetic gases can be dramatically reduced through a number of simple actions. This editorial lays out the options for more sustainable equine anaesthesia.

CLIMATE CHANGE: A GROWING THREAT

Climate change is 'the biggest global health threat of the 21st century' (Costello et al., 2009), hallmarked by increased frequency of extreme weather events, a rise in mean global temperature of over 1°C since the preindustrial era, and the excessive European summer temperatures seen in 2022. Without immediate and concerted action, warming is projected to lead to catastrophic effects on biodiversity, food security and human and animal health (IPCC, 2022).

VOLATILE ANAESTHETIC AGENTS AS GREENHOUSE GASES

The provision of healthcare, both human and veterinary, comes at an environmental cost. In 2019, the National Health Service (NHS) in England emitted 25 megatonnes of carbon dioxide equivalents, representing 7% of the UK carbon footprint (Tennison et al., 2021). It should be noted that the largest part of healthcare's carbon footprint comes from procurement activities. However, atmospheric release of anaesthetic gases (albeit including nitrous oxide and desflurane) is thought to contribute roughly 2% of the total NHS footprint (NHS England and NHS Improvement, 2020) and is thus an important target of actions to reduce the environmental impact of anaesthesia.

Although volatile anaesthetic agents such as isoflurane, sevoflurane and desflurane are released in relatively low volumes, they are disproportionately potent greenhouse gases due to their ability to absorb infrared radiation within an atmospheric 'cooling window' (approximately 8–14 mm) in which absorption of radiation is normally minimal (Andersen et al., 2012). Fortunately, studies in both humans (Thiel et al., 2018) and small animals (McMillan, 2021) demonstrate that emissions arising from these waste anaesthetic gases can be dramatically reduced through a number of simple actions.

Medical guidance has recently been published to address pollution from inhalational anaesthetics (Devlin-Hegedus et al., 2022). In this editorial, we suggest more sustainable options for equine anaesthesia.[1]

OPTIONS TO REDUCE EMISSIONS FROM VOLATILE AGENTS

- Reduce anaesthetic time: Where appropriate, consider clipping surgical sites prior to anaesthesia; ensure the theatre team are present and coordinated, and minimise avoidable delays during anaesthesia.
- Regular servicing and checks: The gas manifolds, intermediate pressure system, vaporiser, anaesthetic machine, breathing systems and endotracheal tubes are all sources of leaks and wastage.
- Prefilling the breathing system: Prefilling the reservoir bag/ventilator bellows with oxygen/volatile agent enables earlier reduction of fresh gas flow (FGF) and vaporiser settings. A bung can be used to close the patient end of the breathing system between cases.
- Reduce FGF: Where volatile agents are vented to the atmosphere (as with most active scavenging systems) and not captured, reducing FGF results in proportional reductions in greenhouse gas impacts. Reducing FGF to achieve carbon benefits may be described as lower flow anaesthesia; however, in horses, volatile anaesthesia is often delivered using rebreathing circuits using close to the minimum metabolic oxygen requirement (low flow anaesthesia). Lower flow anaesthesia should be performed within the clinician's capabilities and the manufacturer's equipment guidelines. Several potential issues must be considered when reducing flows close to minimum metabolic oxygen requirements (Feldman, 2012; Jones & West, 2019). These concerns, with appropriate mitigation measures, include:
  - Accidental awareness: Prepare injectable intravenous anaesthesia to produce a rapid increase in depth of anaesthesia if required. End-tidal anaesthetic agent monitoring and capnography are also highly recommended. If time allows, the depth of anaesthesia can be adjusted by increasing the vaporiser setting and emptying the rebreathing bag, rather than increasing the FGF.
  - Increased risk of delivery of a hypoxic mixture due to failure to denitrogenate, or accumulation of toxic gases: In horses,

monitoring and maintaining the fraction of inspired oxygen over 30% mitigates the risk of dangerous accumulation of other gases at low flows.
    - Insufficient oxygen FGF: The rebreathing bag or the ventilator bellows should always contain sufficient gas to meet the tidal volume of the horse.

- A recent audit of small animal anaesthetic practices found that FGF could have been reduced in 97% anaesthetics, with a corresponding reduction in carbon footprint of 63% (McMillan, 2021). It is important to note that this degree of reduction may not be achievable in equine practice due to the ratio of maximum deliverable FGF to the minimum oxygen demand of such large patients. An example lower flow protocol is included in Figure 1.
- Recapture and reuse of volatile anaesthetic agents: This promising technology is being trialled in NHS trusts, and life cycle analysis suggests this has potential to significantly reduce carbon emissions from inhalational anaesthesia (Hu et al., 2021).

Replacement of volatile agents

- Consider total intravenous anaesthesia (TIVA): The carbon footprint has been calculated for various active ingredients (Parvatker et al., 2019) and TIVA is likely to have a lower carbon footprint over its life cycle than inhalational agents, although the difference may be small depending on factors such as drug choices, energy sources, drug synthetic pathways and whether recapture is utilised (Hu et al., 2021; Narayanan et al., 2022). It should be noted that TIVA is currently only suitable for short procedures in horses.
- Consider standing sedation and ensure sufficient sedation/analgesia: Where safe and appropriate, some procedures may be carried out in conscious or sedated horses. Use of analgesia, MAC-sparing techniques (such as partial intravenous or regional anaesthesia), or drugs which stabilise the depth of anaesthesia (preventing the need for rapid increases in depth of anaesthesia) will reduce inhalant emissions. Constant rate infusions (CRIs) of ketamine, lidocaine and alpha-2 agonists all have minimum alveolar concentration (MAC)-sparing properties, and studies in horses have demonstrated significantly reduced isoflurane requirements when using CRIs of medetomidine, detomidine or romifidine (Neges et al., 2003; Niimura del Barrio et al., 2017). Robust sedation and analgesia will also reduce the likelihood of accidental awareness in the early phases of anaesthesia, which reduces the need for a rapid change in depth of anaesthesia.

It should be emphasised that the environmental impacts of many drugs and consumables have not been fully assessed. Where volatile agents are to be replaced by, or used alongside, injectable drugs, consideration must be given to additional factors such as single-use plastics, wastage due to poor stock control, waste disposal, energy use and ecotoxicity of excreted drugs/metabolites.

Choice of anaesthetic agent

Different anaesthetic agents have significantly different potencies as greenhouse gases, measured in terms of their global warming potential over 100 years (GWP_{100}). Sevoflurane (with the shortest atmospheric lifetime) has a lower GWP_{100} than isoflurane, and both have a significantly lower GWP_{100} than desflurane (see Table 1; Andersen et al., 2011). Additionally, isoflurane contributes to ozone depletion (Andersen et al., 2012).

First 12-20 minutes:

Deliver 8-12 L/min in order to ensure the minimum recommended inspired oxygen and achieve the desired depth of anaesthesia.

Maintenance phase:

The minimum FGF can equal the horse's oxygen consumption (10 x body weight (kg)$^{0.75}$/min), or approximately 2.2 ml/kg/min.

Example for 500 kg horse:

FGF = 10 L/min for first 12-20 minutes, followed by FGF of approximately 1.1 L/min (2.2 x 500 = 1,100, or 10 x 5003/4 = 1,057). The authors would typically use a FGF of 2 L/min to account for any minor leaks in the circuit.

Recovery phase:

8-12 L/min in order to facilitate recovery from anaesthesia

FIGURE 1 Calculating minimum fresh gas flow (FGF) during anaesthesia (adapted from Muir & Hubbell, 2009). For more information on calculation of FGF during anaesthesia, the reader is directed to Solano et al. (2005) and Wetmore et al. (1987).

TABLE 1 Atmospheric characteristics of commonly used anaesthetic gases.

Atmospheric characteristics	Sevoflurane	Isoflurane	Desflurane	Carbon dioxide
Atmospheric lifetime (years)	1.1	3.2	14	74
GWP_{100}	130	510	2540	1
MAC (Steffey et al., 2015)	2.3%	1.3%	7.0%	
Carbon dioxide equivalent (CO_2e) per MAC-hour for equine anaesthesia at 4 L/min oxygen (direct emissions only) (kg/h)	6.0	12.5	321.4	
Carbon dioxide equivalent (CO_2e) per MAC-hour for equine anaesthesia at 4 L/min oxygen (including emissions from manufacture)[a] (kg/h)	12.7–47.2	14.8–17.3	340.0–356.1	
Equivalent to car driving per MAC-hour of equine anaesthesia at 4 L/min oxygen (including emissions from manufacture)[b] (miles)	49–184	58–68	1328–1391	

Note: Adapted from Jones and West (2019), with values from Andersen et al. (2011) and including extrapolated data from Hu et al. (2021) with permission of the author.

[a]Emissions from manufacture depend on the specific route of manufacture. Figures derived from life cycle analysis by Hu et al. (2021) with permission of the author.

[b]Assuming United Kingdom average car emissions of 160 gCO_2/km (256 gCO_2/mile).

It should be noted that sevoflurane is more expensive and is not currently licenced for use in horses in the United Kingdom. Labelling in the United Kingdom also cautions against the use of long-duration, low flow sevoflurane anaesthesia due to the risk of formation of a nephrotoxic molecule called Compound A. However, current consensus in medical anaesthesia suggests an absence of evidence of harm from the use of lower flow sevoflurane anaesthesia in humans (Kennedy et al., 2019).

Additionally, inclusion of carbon emissions embodied in the manufacturing process reduces the benefit of sevoflurane (Hu et al., 2021; Table 1), although further information regarding the manufacturing process is needed to fully assess the relative greenhouse gas impacts (McGain et al., 2022).

A future for sustainable anaesthesia

The veterinary anaesthetist has a unique opportunity to influence decisions across surgery, anaesthesia and the wider profession. We must look to use the available tools to make equine anaesthesia more sustainable in the short term. Whilst volatile anaesthetics are of significant concern, 62% of NHS England's carbon footprint arises from procurement activities (NHS England and NHS Improvement, 2020), and significant reductions in carbon footprint can be achieved by minimising resource use and maximising reuse (Thiel et al., 2018).

Education and discussion are of vital importance if we are to drive sustainable development within the profession. Further research, including clinical audits and quality improvement initiatives, is needed to demonstrate how the environmental footprint of anaesthesia may be safely and effectively reduced. There is a growing body of literature relating to sustainable development in veterinary (Jones & West, 2019; West, 2021) and medical anaesthesia (Charlesworth & Swinton, 2017; McGain et al., 2020; Skowno & Weatherall, 2021; White et al., 2022). From 2022, the RCVS Practice Standards Scheme includes sustainability at Core, GP and Awards levels; guidance for best practice is readily available through resources such as the clinical checklist produced by the American Society of Anaesthesiologists (Axelrod et al., 2017) and the Greener Veterinary Practice Checklist from Vet Sustain.

Above all, action is needed now to curb the climate emergency. Professional bodies, veterinary hospitals and individual veterinary anaesthetists all have a role to play in mitigating and adapting to the current ecological crises.

AUTHOR CONTRIBUTIONS

All authors were involved in the preparation of the manuscript and all authors have approved the final version of the manuscript.

KEYWORDS

horse, anaesthesia, climate change, greenhouse gas, sustainability

CONFLICT OF INTEREST

No conflicts of interest have been declared.

ETHICS STATEMENT

No live animals were included in this study.

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ENDNOTE

[1] Specialist anaesthesia advice should be sought before all changes in protocol with which the user is not familiar.

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Equine Veterinary Education BEVA AAEP

CASE REPORT

Vaccination with human tyrosinase DNA as a therapy for equine intraocular melanoma—4 cases: 2016-2021

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SUMMARY

Four horses with intra-ocular melanoma presented between 2016 and 2021. Case 1 presented with bilateral intra-ocular melanomas. The left eye demonstrated a large pigmented mass, in contact with the posterior cornea, and the right eye demonstrated a smaller mass. On ultrasonography, both had homogenous soft tissue echogenicity. The horse received the initial course of four doses of Oncept (Merial Ltd) vaccine, 14 days apart. Five months later, there was a deterioration in comfort and appearance of the left eye, which was subsequently enucleated. The mass in the right eye was noted to have increased in heterogeneity on ultrasound, and the horse continued with booster vaccinations at six monthly intervals. At the last follow-up two years after initial presentation, the mass had changed in shape and dimensions, and the adjacent cornea appeared thickened, suggesting subtle deterioration. Case 2 presented with an intra-ocular melanoma in the left eye and subsequently received the initial course of Oncept (Merial Ltd) vaccine. At the fourth appointment (6 weeks after presentation), there was diffuse corneal oedema and buphthalmos. Glaucoma was suspected, and the horse was discharged on dorzolamide and timolol. Four months after presentation, the corneal oedema had improved, but there was no change in the mass. Case 3 presented with recurrent corneal ulceration and uveitis in the left eye associated with an intra-ocular melanoma, which was in contact with the posterior cornea. The horse received the initial Oncept (Merial Ltd) vaccine course and topical treatment for ulceration. The horse re-presented every 6 months for booster vaccinations over the next four years. Ultrasonographically, the mass had reduced in size 1 year following initial presentation, and then further at 18 months. The owner reported a marked reduction in the frequency of corneal ulceration over this period. The mass showed no further change over the following 2½ years. Case 4 presented with an intra-ocular melanoma in the right eye and associated corneal oedema. The horse received the initial Oncept (Merial Ltd) vaccine course, and at the fourth appointment, the corneal oedema had worsened. Six months later, the eye had deteriorated significantly and was enucleated. The small number of cases in this report precludes firm conclusions. However, the outcomes suggest limited justification for the routine recommendation of the Oncept[1] vaccine in cases of intra-ocular melanomas. No adverse effects were reported, but prognosis for retaining the eye is guarded.

KEYWORDS
horse, melanoma, Oncept, vaccine, ocular

Key points

- The Oncept vaccine can be considered as an alternative, or adjunctive to surgical management of intra-ocular melanoma in the horse.
- Ultrasonography is helpful for monitoring response to treatment.
- Prognosis for retaining the eye is guarded. Three out of four cases in this report demonstrated clinical progression after commencement of the vaccine course.

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Equine Veterinary Education BEVA AAEP

CASE REPORT

Acute tracheal trauma and chronic tracheal collapse in an asthmatic mature pony

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SUMMARY

A 17-year-old cross-bred pony gelding was presented for acute onset of respiratory distress and an inspiratory honking noise. He had been previously diagnosed with moderate mastocytic equine asthma and chronic tracheal collapse on endoscopy (grade 3/4). At admission, predominantly inspiratory dyspnoea with open mouth breathing, hypoxaemia and hypercapnia were present. Cervical and thoracic radiographs showed severe tracheal collapse from the mid-cervical region to the thoracic inlet and a moderate generalised bronchial pattern. Due to the poor prognosis, euthanasia was elected. On post-mortem examination, some granulomatous nodules compatible with tracheobronchopathia osteochondroplastica were present in the tracheal lumen, and the entire trachea was laterally flattened (Figure 1). This uncommon laterolateral collapse was more severe at the thoracic inlet and in the mid-cervical area, where a peri-tracheal haematoma was detected. A blunt trauma that exacerbated the tracheal collapse was suspected. Tracheal histology revealed degenerative changes. Degeneration of tracheal cartilage and connective tissue can be a predisposing factor to chronic collapse and recurrent episodes of inspiratory and expiratory distress. It is unknown whether chronic tracheal collapse is an independent condition, or a consequence of an underlying lung disease.

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KEYWORDS
horse, chondromalacia, collapse, equine asthma, respiratory distress, trachea

FIGURE 1 Post-mortem macroscopic view of the cervical part of the trachea which is flattened in a laterolateral plane and deviated (yellow arrowheads). A 5-cm haematoma is visible (white arrows).

Key points

- Tracheal collapse may occur in different breeds of ponies secondary to progressive degenerative changes and breakdown in the cartilaginous tracheal rings (chondromalacia) and may be associated with concomitant lung disease.
- Suggestive clinical signs are honking inspiratory noise and respiratory distress, that may be exacerbated by exercise, stressful events and a dusty, windy or hot environment.
- The combination of tracheal endoscopy, cervical and thoracic radiographs and fluoroscopy allows the identification, location and assessment of the severity of the collapse.

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Equine Veterinary Education BEVA

CASE REPORT

Tracheal obstructive mastocytoma in a pony

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SUMMARY

A 15-year-old pony was presented for tracheal mass removal with a one-week history of dyspnoea with inspiratory and expiratory noise. The clinical examination was within normal limits except for a loud inspiratory noise associated with inspiratory dyspnoea at rest and short episodes of respiratory distress during feeding or when stressed. Blood analysis revealed a total nucleated cell count within normal ranges except a mild eosinophilia (1.3×10^9/L [reference ranges $0–1\times10^9$/L]), a mild anaemia (red blood cells 5.3×10^{12}/L [reference ranges: 6.2–10.2 10^{12}/L]) and a packed cell volume of 24% (reference ranges: 31%–43%) and a hyperfibrinogenaemia (3.4 g/L [reference ranges 0–2.5 g/L]), consistent with chronic inflammation. Tracheoscopy revealed two intraluminal masses. The first mass was located approximately 60 cm from the nostrils, obstructing the tracheal lumen by approximately 20%. The second mass was located at the thoracic inlet, obstructing approximately 90% of the tracheal lumen. Because of a severe respiratory distress episode, a standing emergency tracheotomy was performed, and an endotracheal tube was inserted into the tracheotomy site and advanced to place the tip caudal to the second mass. Standing surgical removal was planned for the following day in two steps. First, surgical removal of these masses was performed in a minimally invasive approach under endoscopic guidance with an instrument portal in the tracheotomy incision (Figure 1). Long-handle scissors and Babcock forceps were inserted and used to sever and remove the masses. Second, a diode laser was used to cauterise the resection sites. A diagnosis of tracheal mastocytoma was made based on gross findings and histopathologic examination. The tracheotomy site healed by second intention. Eleven months post-operatively, a recheck examination was performed. The pony had not had any subsequent clinical signs of airways obstruction, and endoscopic examination of the upper airways did not show any signs of reoccurrence of the tracheal masses.

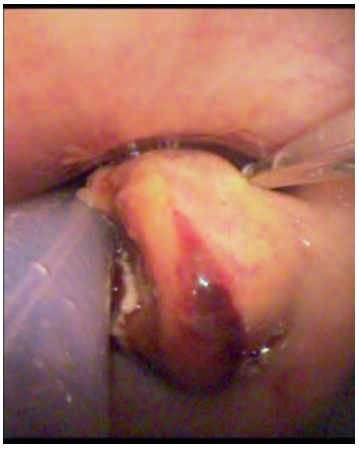

FIGURE 1 Tracheoscopic image showing the attachment of the mass by passing a Babcock forceps between this mass and the tracheal wall

KEYWORDS
horse, mast cell tumour, mastocytoma, mastocytosis, trachea

Key points

- Upper respiratory mastocytoma is rare in horses but should be considered in the differential diagnosis of respiratory obstruction.
- Surgical access to intraluminal lesions located at the distal cervical and thoracic parts of the trachea can be difficult.
- Temporary tracheotomy can be used as an instrument portal to gain access to distal intraluminal lesions in a minimally invasive way.

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Equine Veterinary Education BEVA AAEP

CASE REPORT

Traumatic synovial herniation of the medial femorotibial joint as an aetiology of hindlimb lameness in a horse

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SUMMARY

A 3-year-old Quarter Horse filly presented with an acute right hindlimb lameness of unknown aetiology. Examination of the right hind found moderate effusion in the medial femorotibial joint and a consistent Grade 4/5 right hindlimb lameness that improved with intra-articular anaesthesia of the medial femorotibial joint. Radiographs of the stifle found no significant abnormalities. Ultrasonographic evaluation of the medial femorotibial joint revealed a disruption of the synovial membrane in the medial recess of the joint capsule and a possible lesion of the medial meniscus. Arthroscopy of the medial femorotibial joint found normal meniscal and cartilaginous anatomy. A rent in the joint capsule was identified at the attachment of the capsule to the medial collateral ligament. The horse was diagnosed with an idiopathic traumatic tear of the medial femorotibial joint capsule. Repair of the tear in the capsule was not attempted due to the tension on the tissues. Medical treatment was initiated with autologous conditioned serum (IRAP-II™, Arthrex Vet Systems), rest and firocoxib. Follow-up examination at 14 weeks revealed an improved lameness score on baseline and in circles both directions at the trot. Repeat ultrasonographic evaluation of the right stifle revealed mild medial femorotibial joint effusion in the medial recess of the joint. The extracapsular hypoechoic area that had previously been identified and confirmed as a synovial tear on arthroscopic evaluation was located. This area had formed an outpouching of the synovial membrane and was filled with fluid, consistent with a synovial hernia (Figure 1). A second 10 mL intra-articular injection of autologous conditioned serum and 9 mg of triamcinolone acetate was aseptically administered into the medial femorotibial joint. The patient was discharged and continued hand walking with turn out and groundwork. The daily firocoxib dose was continued to aid in managing inflammation that arises during rehabilitation.

KEYWORDS
horse, orthopaedics, joint, hernia, synovial, stifle

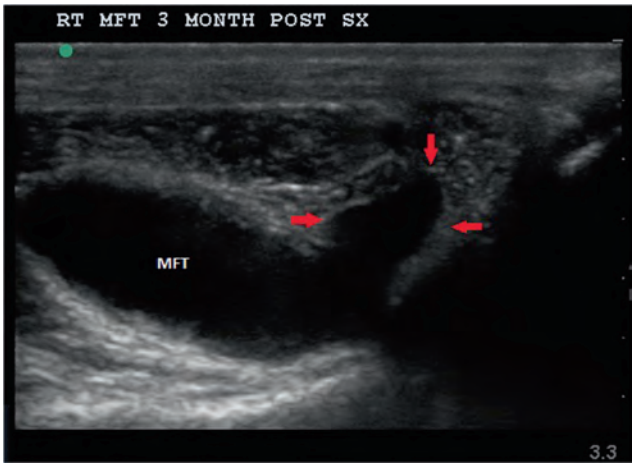

FIGURE 1 Ultrasound at follow-up showing a defined synovial hernia (red arrows) where the previous surgically identified tear had been shown.

Key points

- Traumatic tearing of the joint capsule with subsequent synovial herniation whilst rare presents a possible differential diagnosis for cases of idiopathic stifle lameness.
- Ultrasound of the stifle joint can be a useful diagnostic tool in both identifying causes of equine hindlimb lameness that are not identifiable on radiographs, as well as follow-up evaluations of the healing process.
- Conservative medical therapy with a combination of regenerative and steroidal therapies can aid in the healing and repair of the joint capsule with improvement of lameness scores on tears that may not be able to be repaired surgically.

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Equine Veterinary Education

CASE REPORT

Aortic valve stenosis associated with valvular heterotopic ossification with extramedullary haematopoiesis in a horse

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SUMMARY

A 20-year-old Thoroughbred mare was evaluated for suspected colic, tachycardia (92 beats/min), an irregular heart rhythm and lethargy. The owners were concerned at her weight loss, despite a good appetite in the preceding 2 months. The horse was depressed and in poor body condition, weighing 430 kg. The mare was pyrexic (rectal temperature 38.9°C), tachypnoeic (32 breaths/min) and tachycardic (60–80 beats/min) with a chaotic rhythm and pulse deficits, jugular pulsation that reached the angle of the jaw and a grade II/VI holosystolic murmur audible over both the left and right heart base. A resting modified base-apex ECG confirmed the suspected diagnosis of atrial fibrillation. Echocardiography revealed a subjectively enlarged heart. The aortic valve leaflets were thickened and distorted with the left and right coronary cusps fused in a 'V' shape at their junction. Moderate aortic regurgitation was evident. Multifocal, hyperechoic areas throughout the myocardium were present. The mitral valve leaflets were subjectively thickened, and a large jet of mitral regurgitation was identified. In addition, marked enlargement of the pulmonary artery, along with systolic flattening of the interventricular septum suggested pulmonary hypertension. Medical management was discussed, but considering the poor prognosis, the owners elected for euthanasia of the horse. At necropsy, the left and right coronary cusps of the aortic valve were irregularly distorted and thickened over an area of approximately 3.5 cm by 1.5 cm extending from the intercoronary commissure and fused in a Y-shaped configuration (Figure 1). Microscopic examination of the aortic left and right coronary cusps revealed chronic degenerative changes with foci of cartilaginous metaplasia. The expanded right cusp contained metaplastic bone with elements consistent with heterotopic ossification with extramedullary haematopoiesis. To the authors knowledge, this is the first reported case of valvular aortic stenosis in a horse.

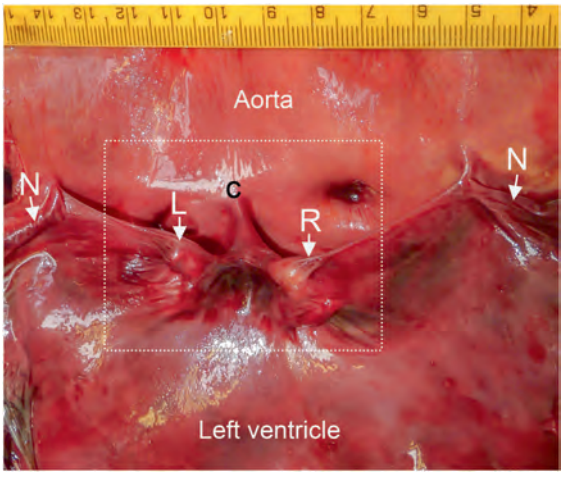

FIGURE 1 Aortic valve. Within the rectangular area indicated, the left (L) and right (R) coronary cusps are distorted. N, noncoronary cusp; C, intercoronary commissure.

KEYWORDS
horse, aortic valve stenosis, heterotopic ossification

Key points

- This is the first reported case of aortic valvular stenosis in a horse.
- The aortic valve was calcified and contained bone marrow elements.
- Aortic valve stenosis may not be clinically recognised until the disease process is advanced.
- The process of calcification could be due to chronic damage or could have resulted from a congenital abnormality.

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Equine Veterinary Education BEVA AAEP

CASE REPORT

Surgical treatment of exuberant granulation tissue of the glans penis and paraphimosis in a stallion

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SUMMARY

A 15-year-old male Quarter Horse stallion was referred for the evaluation of a lesion involving the glans penis following a wire laceration while attempting to breed a mare through a fence. During anamnesis, it was found that the lesion occurred about four months previously, and the animal had not received emergency medical care from a veterinarian. Examination of the external genitalia revealed a large granulating wound involving the craniodorsal aspect of the glans penis. Exuberant granulation tissue (EGT) was readily identified and was painful on palpation. The voluminous tissue was pink, irregular and had a cauliflower shape, preventing the retraction of the penis into the prepuce indicative of paraphimosis (Fig. 1). A sample of the proliferative tissue was collected by biopsy and submitted for histopathological examination. Histopathology confirmed EGT with no evidence of neoplasia. The mass was characterised by fibroblasts arranged in bundles, largely parallel to the surface, with blood vessels parallel to each other and practically perpendicular to the surface.

After confirming the diagnosis, surgical removal of EGT, followed by penile reconstruction, was indicated. Following aseptic preparation and anaesthaesia of the penis and prepuce, a tourniquet was placed encircling the penis as far proximal as possible, the penile urethra was catheterised to avoid iatrogenic damage and after complete removal of the EGT, and the surgical wound was closed using Ford interlocking pattern. Three months after surgery and postsurgical drug treatment, the stallion had returned to natural breeding.

KEYWORDS
horse, exuberant granulation tissue, paraphimosis, penis injury, surgical excision

FIGURE 1 Exuberant granulation tissue in the glans penile

Key points

- Severe equine penile lesions should be treated with emergency medical care by a veterinarian.
- Histopathology is the complementary test of choice for the diagnosis of EGT.
- Surgical treatment is indicated in cases of EGT with voluminous tissue, irregular and with a cauliflower shape with paraphimosis.

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Equine Veterinary Education BEVA AAEP

CASE REPORT

Treatment of a stallion for penile deviation caused by haemorrhage into the corpus cavernosum penis

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SUMMARY

A 19-year-old Thoroughbred stallion developed severe curvature of its penis, preventing intromission, after breeding a mare. The cause of the curvature was found, during the ultrasonographic examination of the penis, to be a rupture of the trabeculae on the left side of the corpus cavernosum penis (CCP) between the *cul de sac* of the external preputial cavity and the scrotum, resulting in the formation of a seroma. Aspiration of the seroma performed 13 days after injury, with the horse anaesthetised, largely resolved the deviation. A complication of treatment was excessive belling of the glans penis during erection, which may have been the result of a temporary shunt created between the CCP and the corpora spongiosum penis (CSP) by inadvertently passing the spinal needle used to aspirate the seroma through the CSP. Excessive belling resolved after 3 weeks.

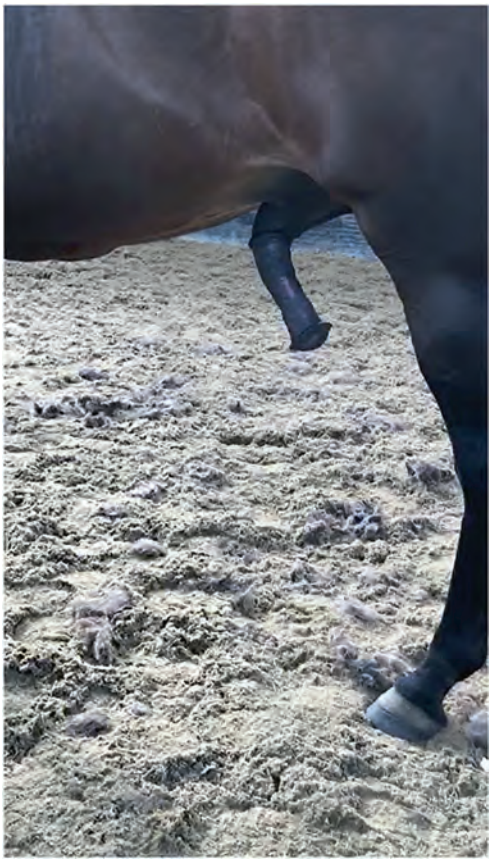

FIGURE 1 Stallion's erect penis, presumably injured during breeding 12 days earlier, curved to the left and ventrally, making intromission impossible.

KEYWORDS

horse, aspiration, deviation, penis, seroma

Key points

- Penile deviation of a stallion may be caused by haemorrhage into the corpus cavernosum penis (CCP).
- Haemorrhage into CCP can be detected by examining the penis ultrasonographically.
- Penile deviation of a stallion caused by haemorrhage into the CCP can be corrected by aspirating the seroma resulting from the haemorrhage.

Work was performed at Fethard Equine Hospital, Fethard, Co. Tipperary, Ireland.

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Equine Veterinary Education

CASE REPORT

Caecocolic ligament hypoplasia in a Thoroughbred filly with volvulus of the large colon

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SUMMARY

An 11-month-old Thoroughbred filly was referred to the hospital with signs of severe abdominal pain unresponsive to medical treatment. Upon arrival, the filly had a heart rate of 60 beats/min, pink mucous membranes and continued to show severe colic signs despite sedation. Due to the severity of colic signs, general anaesthesia was induced immediately. Exploratory celiotomy revealed a large colon volvulus of more than 360 degrees and hypoplasia of the caecocolic ligament between the caecum and right dorsal colon. The large colon volvulus was corrected, and the filly recovered uneventfully from general anaesthesia. Post-operative treatment included intravenous fluids and intravenous lidocaine continuous rate infusion, parenteral nutrition, systemic antibiotics, nonsteroidal anti-inflammatories, gastroprotectants and restricted feeding. The filly experienced several post-operative complications associated with severe enterocolitis such as pyrexia, leukopenia and diarrhoea but made a full recovery and was discharged to the farm after 16 days of hospitalisation. Long-term follow-up was available for 22 months after surgery without further reports of colic, and the filly had entered race training. Hypoplasia of the caecocolic ligament in association with colic in horses has rarely been reported in the equine literature. To date, only two further case reports of young horses with large colon volvulus and hypoplasia of the caecocolic ligament have been published. In contrast to the presenting case, several other mesenteric abnormalities were present in these two cases in addition to the hypoplastic caecocolic ligament, and both horses were eventually euthanised. The pathogenesis of hypoplasia of the caecocolic ligament remains unclear but may include the disruption of early stages of embryogenesis. An abnormal caecocolic ligament has been suggested to potentially predispose horses to large colon volvulus. The presenting case indicates that as an isolated abnormality, a hypoplastic caecocolic ligament may not result in reduced long-term survival in horses with large colon volvulus.

KEYWORDS
horse, caecocolic, colic, mesenteric, surgery

Key points

- Hypoplasia of the caecocolic ligament is a rarely reported mesenteric abnormality in horses with colic and large colon volvulus.
- As an isolated finding, a hypoplastic caecocolic ligament may not result in decreased post-operative long-term survival or an increased risk of recurrent colic.
- The aetiology of this mesenteric abnormality is currently unknown but may include defects during early stages of embryogenesis.

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Equine Veterinary Education BEVA AAEP

CASE REPORT

Cryoglobulinaemia in a Thoroughbred gelding with multicentric lymphoma

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SUMMARY

A 17-year-old Thoroughbred gelding was presented to the hospital with epistaxis, multiple alopecia skin lesions, firm swelling in the masseter region and acute cellulitis of the right hind distal limb. At the time of admission, he was quiet but comfortable with a heart rate of 52 beats/min and otherwise normal vital parameters. Initial blood work abnormalities included severe leucocytosis (white cell count 30 x 10^9/l) with a left shift and marked hyperproteinaemia. Coagulation times and platelet count were adequate. Upper airway endoscopy confirmed that epistaxis originated from ulcerative lesions within the nasal passages and nasopharynx. A skin biopsy was performed near the left elbow, and histopathology examination revealed necrotising dermatitis. During routine storage of the initial blood samples in the refrigerator, marked precipitation was noted. Since triglyceride levels were normal, the precipitate was deemed to be the result of cryoprecipitation rather than lipaemia. When a blood sample was submitted for serum protein gel electrophoresis to investigate the hyperproteinaemia, the test yielded inconsistent results depending on sample preparation at the referral laboratory. While high-speed centrifugation resulted in normal serum protein levels and normally distributed protein fractions, an uncentrifuged sample showed hyperproteinaemia and a gammopathy with a peak in the beta 2 globulin fraction. This globulin fraction was apparently removed during sample preparation and likely represented the cryoprecipitate noted earlier, suggestive of cryoglobulinaemia. Due to financial constraints of the owner, further investigation was not performed at the time. Treatment was symptomatic and empirical and primarily focused on the clinical diagnosis of vasculitis, dermatitis and a suspected underlying neoplastic condition. Medications included a tapering course of intravenous corticosteroids (dexamethasone), topical anti-inflammatory skin preparations, gastroprotectants (omeprazole) and supportive care. Due to little clinical improvement and a guarded prognosis, the gelding was euthanised. Necropsy revealed multicentric lymphoma with a trophism for perivascular spaces and skeletal muscles.

KEYWORDS
horse, cryoglobulin, cryoglobulinaemia, lymphoma, epistaxis

Key points

- *Cryoprecipitation* in equine serum blood samples in the absence of lipaemia can significantly influence the results of serum gel-electrophoresis and serum biochemistry evaluation and can indicate the presence of cryoglobulins.
- Unspecific clinical signs including *vasculitis, dermatitis and epistaxis* with concurrent hyperproteinaemia and hyperglobulinaemia that are refractory to treatment can be related to cryoglobulinaemia in horses.
- Lymphoproliferative diseases such as *lymphoma* have been associated with cryoglobulinaemia in humans and may be associated with cryoglobulinaemia in horses.

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Equine Veterinary Education BEVA AAEP

CASE REPORT

Diagnosis and arthroscopic removal of an intra-articular epidermoid cyst in the distal interphalangeal joint of a 15-year-old horse

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SUMMARY

A 15-year-old Warmblood gelding was presented for evaluation of left forelimb lameness first observed 9 months previously. Examination in the referral hospital revealed a firm swelling on the dorsal aspect of the pastern, in the region of the dorsal pouch of the distal interphalangeal (DIP) joint. Moderate lameness was observed at the walk, and marked lameness with a significantly shortened cranial phase of the stride was observed at the trot. Diagnostic analgesia localised the source of lameness to the foot.

Radiographic examination of the left front foot revealed mild osteoarthritis of the distal interphalangeal (DIP) joint and a non-mineralised soft tissue swelling centred on the dorsum of the second phalanx. An oval-shaped mass consisting of heterogenic linear and branching structures localised within the dorsal recess of the joint was identified on ultrasonographic examination of the DIP joint. Standing, low-field MRI revealed marked distension and capsular thickening of the DIP joint. There was a soft tissue oval mass filling the dorsal recess of the DIP joint, that appeared hyperintense in T1-weighted sequences, and hypointense in T2-weighted and STIR sequences (Figure 1). The mass contained a small volume of intermixed fluid signal and serpentine structures.

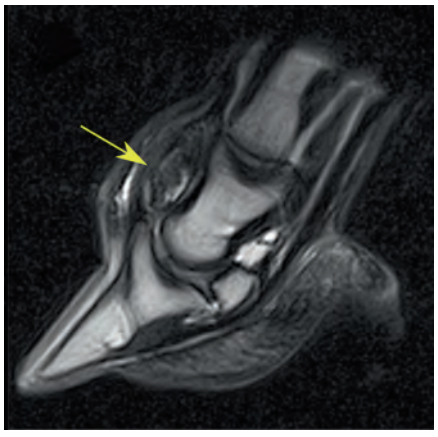

FIGURE 1 Sagittal T2-weighted FSE MRI of the LF foot, note a hypointense mass filling the dorsal recess of the DIPJ (arrow)

Arthroscopy of the left front DIP joint was performed to remove the mass and to further evaluate the joint. Thick, semitranslucent white-grey, lamellar material was found within the dorsal recess of the joint and was removed in piecemeal fashion. An area of thickened, hyperaemic synovial capsule containing abnormal granular grey material was identified and was also removed. Mild hyperaemia and proliferation of the synovial membrane were identified throughout the joint.

Histological features of the resected tissues were consistent with a ruptured epidermoid cyst and secondary granulomatous and lymphoplasmacytic synovitis.

Following the surgery, ridden exercise was gradually re-introduced. Four months postoperatively, the horse was in full ridden work, including jumping, and remained sound for further 6 months. When the horse became lame again, 12 months postoperatively, it was retired. This case report, in line with previous reports, provides some evidence that surgical removal of intrasynovial epidermoid cysts is indicated.

KEYWORDS
horse, arthroscopy, cyst, epidermoid

Key points

- Epidermoid cysts are benign, encapsulated, pseudoneoplastic lesions, usually localised in the skin and subcutaneous tissue. However, they also have been found in other locations. They can be congenital or traumatic in origin. Intraosseous or intrasynovial epidermoid cysts localised within the digit can cause severe lameness in horses.
- Standing MRI can be helpful in the diagnosis of intra-articular masses. Ruptured epidermoid cysts should be considered as differential diagnosis when masses consisting of mixed intensity material are found on MRI.
- Rupture of an intrasynovial epidermoid cyst can cause marked inflammatory response and exacerbation of clinical signs. Surgical removal of intrasynovial epidermoid cysts is indicated.

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Equine Veterinary Education BEVA AAEP

CLINICAL COMMENTARY

Solving equine distal limb lumps and bumps

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The case report of a ruptured epidermoid cyst in the distal interphalangeal joint (DIP) is the first intra-articular epidermoid cyst described in the equine veterinary literature (Hibner-Szaltys et al., 2023). Articular synovial cysts, also called hernias, have been identified previously in many joints but none to date either in the DIP (Lacourt et al., 2013). The description of the clinical history, presentation, diagnosis, treatment and histopathology of this novel case is valuable as it will inform equine veterinarians if they encounter a similar challenge in the future.

Epidermoid cysts develop progressively from errant epidermal cells, either congenital or traumatic, and arise most commonly in superficial subcutaneous tissues but also in other tissues such as bone. Osseous epidermoid cysts are most frequently diagnosed in the phalanges in people, but also arise in the skull (Wang et al., 2003). It is interesting that epidermoid cysts have also previously been identified in the equine distal limb including the distal part of digital flexor tendon sheaths (Joostens et al., 2019; Sanz et al., 2006), distal phalanx (Fraser et al., 2006; Headley et al., 2009) and the foot (Mageed & Elfadl, 2020). The current report (Hibner-Szaltys et al., 2023) of the DIP epidermoid cyst expands the repertoire of equine distal limb sites where cysts may arise.

In the report by Hibner-Szaltys et al. (2023), the affected horse was treated for lameness 9 months prior to the final presentation and responded to intra-articular medication at that time. It is speculated that the epidermoid cyst was present for some time in this 15-year-old horse as they are benign and slow growing. A local trauma at the site may have led to its rupture on the initial occasion when the horse presented with a soft tissue swelling over the dorsal DIP. However, the exact cause of the cyst remains unknown.

Radiography and ultrasonography were employed to image the cyst when the lameness reappeared. Although the ultrasound images were not included, it was mentioned that heterogenic linear and branching structures were localised in the circumscribed mass within the dorsal recess of the joint. The ultrasonographic appearance of an equine epidermoid cyst in the foot has been recently illustrated by others (Mageed & Elfadl, 2020). It was also well defined and, similar to the Hibner-Szaltys et al. case (2023), contained heterogenous echogenic material. In contrast, articular synovial cysts are characterised by an outpouching of anechoic fluid from the joint cavity on ultrasonographic examination, with a canal through the joint capsule and communication with the joint space can be detected (Lacourt et al., 2013). The ultrasonographic description could help veterinarians in the field orient their diagnosis in similar cases.

The MRI characterisation of this ruptured intra-articular epidermoid cyst is also similar to two cases reported in tendon synovial sheaths (Joostens et al., 2019; Sanz et al., 2006). In the latter study, the epidermoid cyst was not visible on ultrasound suggesting that MRI imaging is superior. Taken together, these three cases of epidermoid cysts in equine distal limb synovial structures suggest that a distinguishing feature is they develop in the synovial membrane and expand within the synovial cavity, whereas articular synovial cysts or hernias protrude beyond the joint cavity (Lacourt et al., 2013).

It is not surprising that the authors state that the arthroscopic removal of the cyst was difficult. The size of the epidermoid cyst in the current report was substantial, expanding and filling the dorsal synovial outpouching of the DIP. The dorsal recess of the DIP attaches to the extensor process of the third phalanx and is located palmar to the extensor tendon that restrains distension. The large size of the mass, the presence of chronic synovitis, fibrosis and thickening of the joint capsule all precluded satisfactory visualisation. The primary surgeon must have had a sinking feeling when they encountered the unusual 'thick, semitranslucent, white-grey lamellar material' on entering the joint. This was the remaining hydrophobic keratin protein in the centre of the cyst and is normally on the outer layer of the skin, protecting epithelial cells. This protein was the cause of the presence of a granulomatous synovitis in the DIP.

The authors did not mention whether they employed a motorised synovial resector during the arthroscopic procedure. Synovectomy with a synovial resector greatly enhances visualisation in the dorsal DIP in the presence of chronic synovitis. Arthrex's tapered torpedo shaver is particularly useful in this joint as its narrow nose facilitates entry and movement around the joint providing access to hard-to-reach areas for soft tissue debridement.

The histological diagnosis was clear-cut because of the very characteristic appearance of the epidermoid cyst tissue. The epidermoid cyst was lined with a stratified squamous epithelium with a

granular layer, but had no other skin appendages, characteristic of dermoid cysts. When intact, it is keratin filled.

This histologic appearance clearly differentiates it from the more common articular synovial cysts that are lined by synoviocytes (Lacourt et al., 2013) and usually filled with synovial fluid in vivo. It is also worth mentioning here that synovial cysts associated with tendon sheaths are not lined by synoviocytes (Crawford et al., 2011). The histological findings help interpretation of the characteristic ultrasound and MRI findings observed with cysts in equine in synovial structures.

Finally, there was a happy ending: the case returned to competition, although was retired a year later. This is a more satisfying outcome than that reported for epidermoid cysts in the third phalanx and foot: All were subjected to euthanasia because of the guarded prognosis (Headley et al., 2009; Mageed & Elfadl, 2020).

A take-home message, based on a very limited number of cases (Hibner-Szaltys et al., 2023; Joostens et al., 2019; Sanz et al., 2006), is that equine distal limb epidermoid cysts located in joints or tendon sheaths have more favourable outcome than their counterparts within the third phalanx or foot.

CONFLICT OF INTEREST

No conflicts of interest have been declared.

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Equine Veterinary Education BEVA AAEP

CASE REPORT

Successful treatment of an aneurysmal bone cyst in the third metacarpal bone of a foal

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SUMMARY

A 4-week-old Warmblood foal presented with a rapidly growing firm mass on the dorsomedial aspect of the distal left third metacarpal bone. Initial examination revealed no pain on palpation or associated lameness. Radiography demonstrated a solitary expansile large osseous cyst-like lesion with a coarse intraluminal trabecular pattern associated with the cortical bone (Fig 1). Computed tomographic imaging was performed prior to surgery for planning and revealed similar findings. A transcutaneous surgical approach was used to excise the mass followed by curettage of the parent bone. Removal of the mass was achieved by successive small excisions to ensure complete preservation of the underlying dorsal cortex. The mass consisted of a large cavity mainly filled with gelatinous haemorrhagic content surrounded by a thin layer of bone. Histopathology of the mass revealed a reactive process consisting of trabecular bone, haemosiderophages, granulation tissue and mesenchymal tissue proliferation consistent with an aneurysmal bone cyst. Aneurysmal bone cysts in horses are not common, they have been described in the axial and the appendicular skeleton with the mandible most commonly affected. The prognosis for this condition depends on anatomical location, involvement and distortion of surrounding structures and degree of lysis and cortical thinning. The severity of the latter finding may increase the risk of a pathological fracture. Aneurysmal bone cysts should be included in the differential diagnosis of rapidly growing masses on long bones in foals and are amenable to surgical resection. In the current case, CT provided excellent detail with respect to the location, expansion and morphology of the bone mass. This allowed accurate pre-surgical planning. Despite the inability to confidently identify the cause of the aneurysmal bone cyst, surgical removal was successful and the foal did not develop any secondary lesions at the 12-month follow-up, justifying a surgical approach to similar cases.

KEYWORDS
horse, diaphysis, juvenile, unicameral bone cyst, distal extremity

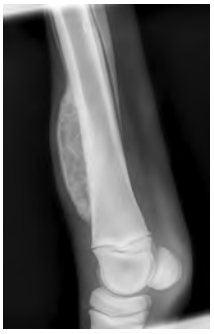

FIGURE 1 Radiograph of the left third metacarpal bone showing the large expansile bony cavitary lesion at the dorsal cortex

Key points

- Aneurysmal bone cysts show radiographically as large oval-shaped smoothly marginated expansile cavities with a thick wall of mineralised bone protruding from the affected cortex of the bone.
- Three-dimensional imaging such as CT offers the additional diagnostic potential to determine the nature of the mass. It is a quick presurgical procedure reducing the risk of the anaesthetic procedure compared to MRI.
- Previously reported cases of aneurysmal bone cysts in horses show high mortality rates with most of the reports describing post-mortem findings only. Early surgical intervention, in this case, resulted in a good outcome.

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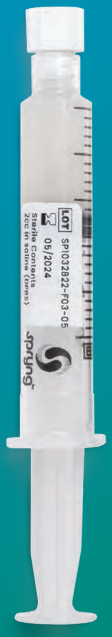

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Equine Veterinary Education

CLINICAL COMMENTARY

Aneurysmal bone cysts as a diagnostic consideration in juvenile patients: Considerations from humans and animals

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Summary

Aneurysmal bone cysts are benign, locally aggressive bone lesions observed in both human and veterinary species, most frequently in a juvenile population. These lesions are now accepted to have a neoplastic aetiology, cause local pain and may result in secondary pathologic fractures. Advanced imaging modalities, such as computed tomography and magnetic resonance imaging, are helpful in characterisation and diagnosis; however, biopsy and histopathology are required to definitively diagnose these lesions. Surgical excision of aneurysmal bone cysts is the most frequently reported treatment method; however, numerous other surgical and medical management strategies are reported across the human and veterinary literature. The limited number of published equine studies on aneurysmal bone cysts suggests that horses may have unique considerations in terms of location and associated morbidity that warrant further discussion.

KEYWORDS
horse, bone, CT, cyst, juvenile, MRI

The term aneurysmal bone cyst (ABC) first appeared in the human literature in 1942, to describe what appeared to be a blood-filled lesion within long bones which, when ruptured, had a radiographic appearance analogous to a saccular aneurysm (Jaffe & Lichtenstein, 1942). Ironically, this lesion is neither an aneurysm, nor a cyst, as it lacks an endothelial wall, which makes it difficult to appropriately describe using these terms. Currently, ABCs are accepted to be a benign bone lesion, which can aggressively expand and destroy the affected bone. Although ABCs are observed in both human and veterinary species, review of the human literature yields a more comprehensive description of the clinical aspects of these lesions. ABCs primarily affect children and young adults, with a median age of 13 years. Sixty per cent of these lesions are found in individuals less than 20 years of age, and 90% of lesions are found prior to 30 years. Females appear to be slightly more affected than males (Hay et al., 1978; Leithner et al., 1999). ABCs are predominantly reported in the metaphysis of long bones, including the femur, tibia, fibula, and upper extremity, but have also been reported in the spine (70% thoracolumbar, <25% cervical spine; 60% of the time in the pedicles, laminae, spinous processes (Weinstein & McLain, 1987), pelvis, sacrum, clavicle, foot, and fingers). The aetiology of ABCs remains poorly elucidated at the current time. Historically, it was believed that ABCs were reactive lesions due to increased venous pressure resulting in expanding bone voids that filled with blood (Park et al., 2016). Understanding the aetiologic basis for these lesions has been a contemporary area of debate, and while an initial report described ABCs as a non-neoplastic condition (Saccomanni, 2008), just 2 years later a description of the oncogenic basis of ABCs was published, which informs the current thought on the pathophysiology of these lesions (Ye et al., 2010). A specific oncogene has now been identified in ABC cases, USP6, which causes extracellular matrix (ECM) degradation via induction of NF-κB and matrix metalloproteinases. This ECM destruction facilitates the characteristic rapid growth and expansion of ABCs. Despite this, ABCs are believed to not have any malignant potential,

but may occur in association with other tumours or malignant processes (30% of cases [Martinez & Sissons, 1988]), as well as solitary lesions (Brinker et al., 1991).

Regardless of the location, ABCs clinically present with pain and swelling of the affected bone. The aggressive erosion of bone may cause a secondary pathologic fracture and if within the spine may also result in neurological deficits secondary to a mass effect on the spinal cord or nerve root impingement (Novais et al., 2011). Importantly, as these lesions commonly affect juveniles, if the ABC is located in the region of the physis, limb deformity or discrepancy is also possible. Diagnosis typically occurs after onset of symptoms, at least initially using radiography. Radiographically, ABCs appear as a radiolucent, eccentrically located cystic lesion within the affected bone, circumscribed by a thin layer of cortical bone (Park et al., 2016). Volumetric imaging modalities, such as computed tomography (CT) and magnetic resonance imaging (MRI), have been recognised as the standard of care for comprehensive characterisation of these lesions. CT is helpful to define the osseous borders of these lesions with cortical erosion and expansion and reveal the characteristic "soap bubble" appearance of ABCs, consistent with a multi-locular lytic lesion (Ameli et al., 1985; De Kleuver et al., 1998; Saccomanni, 2008). Pathologic fractures and in the case of vertebral lesions, vertebral body collapse, are best assessed using CT (Chan et al., 2002). MRI can delineate "fluid–fluid levels," which represent the layering of blood of differing densities (Park et al., 2016). Additionally, MRI can identify septations within ABCs, as well as peri-lesional oedema (Mahnken et al., 2003; Revel et al., 1992). Observation of multilocular cysts with fluid-filled interfaces on T2-weighted images is highly suggestive of an ABC. Use of both radiography and MRI improves the sensitivity and specificity of the diagnosis, compared to the use of each modality individually (Mahnken et al., 2003). However, the commonly reported "soap bubble" and "fluid–fluid levels" are not pathognomonic for ABCs, as other types of neoplasia and bone cysts demonstrate these features. Nuclear scintigraphy (De Kleuver et al., 1998) and angiography (Ameli et al., 1985) have both also been reported in the diagnosis of ABCs, observed as increased radiopharmaceutical uptake or blood supply—including arteriovenous shunts—associated with these lesions.

Lesion biopsy is the current standard for definitive diagnosis of ABCs, typically via incisional biopsy. Fine needle aspirate biopsies lack specificity for definitive diagnosis of these lesions (Creager et al., 2007; Layfield et al., 1993). An accurate and efficient diagnosis is critical for the management of ABCs, as differential diagnoses include both benign and malignant lesions. Most notably, it is critical to differentiate an ABC from telangiectatic osteosarcoma, which exhibits similar features across imaging modalities, but has unique histological features (Murphey et al., 2003). On gross evaluation, ABCs are spongy, haemorrhagic masses covered by a thin shell of reactive bone. Microscopically, ABCs have a dense cellular composition, with red blood cells and pale haemosiderin filling the cystic spaces. They typically have septal proliferations of fibroblasts, mitotically active spindle cells, osteoid, calcifications, scattered multi-nucleated giant cells, and fibrous tissue associated with enlarging vascular spaces with thin-walled blood vessels (Buraczewski & Dabska, 1971; Hay et al., 1978; Kumar et al., 2015).

Despite their benign nature, ABCs rarely resolve spontaneously (Malcom et al., 1978). In the original report of ABCs, curettage and reconstruction of the defect with bone graft were used, and this remains the mainstay of treatment today (Jaffe & Lichtenstein, 1942). Complete surgical resection is associated with the lowest rate of the recurrences, but the highest morbidity to the patient. Studies report a highly variable level of recurrence, ranging from 0%–59% with en bloc resection, but post-operative pain, limb length discrepancies, muscle weakness, decreased range of motion, or post-laminectomy kyphosis (in cases related to the cervical spine) are all common post-operative morbidities (Bell et al., 1994; Biesecker et al., 1970; Campanacci et al., 1986; Hay et al., 1978; Lonstein, 1977; Mankin et al., 2005; Mostafa, 2015; Schaffer et al., 1985; Vergel De Dios et al., 1992; Yasuoka et al., 1981). Use of high-speed burring of the lesion following curettage, argon beam coagulation, phenol "washing" of lesions, cryosurgery, augmentation of defects with polymethylmethacrylate have all been reported in the current management of ABCs (Park et al., 2016). The benefit of these additional management strategies has yet to be consistently and reproducibly demonstrated. The use of radiation therapy for management of ABCs is also somewhat controversial. Excellent local control with radiotherapy—ranging from 83% to 100%—has been reported; however, the complications and chronic effects have prevented the adoption of this therapy into practice (Marcove et al., 1995; Papagelopoulos et al., 1998). As most cases of ABCs occur in young individuals, complications of radiation therapy, especially within the spine, may include myelopathy or radiation-induced spinal injury (Mayfield et al., 1981; Palmer, 1972). Selective arterial embolisation is also an adjunctive therapy that has been espoused by some reports, especially for lesions in the spine, in areas difficult to access (i.e. pelvis, sacrum), or that are at risk for haemorrhage. Importantly, multiple embolisation procedures may be required to fully resolve these lesions, and complications including skin necrosis or paresis have been reported, although high overall rates of local control (94%) have been reported with this technique (Rossi et al., 2010).

Despite the relatively low overall incidence of these lesions, numerous techniques and therapies for ABCs continue to emerge in the literature. Development and refinement of these additional techniques are seemingly motivated by a desire to minimise aggressive resection and reconstruction of the local bone. Sclerotherapy, or induction of damage to the endothelium of the vessels of the ABC, is commonly performed to facilitate local control of the lesion. Ethibloc, a radiopaque alcoholic solution with fibrogenic and thrombogenic effects, and polidocanol (hydroxypolyaethoxydodecan), a sclerosant used for the treatment of varicose veins, have both been used with some success in the treatment of ABCs. Multiple injections of Ethibloc have been reported with healing rates up to 92% (Falappa et al., 2002), while multiple injections with polidocanol have resulted in resolution of up to 93% healing of lesions (Varshney et al., 2010). Notably, sclerotherapy with Ethibloc has been associated with a high level (94%) of complications, resulting in decreased

clinical use. Similar to sclerotherapy, the emerging techniques for treatment of ABCs are focused on investigation of less aggressive surgical techniques and medical managements, in an effort to reduce complications. "Curopsy," a term referring to curative biopsy, stems from the observation that some ABCs are resolved following biopsy alone (Reddy et al., 2014). A percutaneous biopsy is performed to remove material from the ABC, including a portion of the lining membrane. It has been hypothesised that the biopsy process destroys a sufficient amount of the internal architecture of the ABC, allowing the lesion to heal. Recurrence rates with "curopsy" have been reported at 19% in a single study; however, the morbidity is much less compared to conventional techniques. Most interesting is the use of denosumab, a monoclonal antibody that inhibits the receptor-activator of nuclear kappa B ligand (RANKL) signalling pathway. RANKL is an important mediator of bone homeostasis, and ABCs have been reported to have higher than normal levels of expression of this molecule (Yamagishi et al., 2016). To date, a limited number of reports have been published citing the use of denosumab as a neoadjuvant therapy for osteolytic bone lesions, such as ABCs (Dubory et al., 2016; Lange et al., 2013). Encouragingly, Lange et al. (2013) reported tumour regression, pain reduction, and resolution of neurological symptoms in a case series of two spinal ABCs, demonstrating this therapy may become a mainstay of treatment moving forward. Intralesional doxycycline and systemic bisphosphonates are two additional therapies that have been reported in the human literature (Cornelis et al., 2014; Fife et al., 1997). Doxycycline has reported anti-neoplastic properties, including inhibition of MMPs and angiogenesis, while bisphosphates work through inhibition of osteoclast-mediated bone resorption. Ultimately, the safety and efficacy of new medical management and surgical techniques for ABCs require further investigation through prospective clinical trials.

Aneurysmal bone cysts have also been reported in a limited number of cases in the veterinary literature. ABCs have been reported in both dogs and cats, primarily in the long bones of the forelimb (Anson et al., 2020; Barnhart, 2002; Dowdle et al., 2003; Olimpo et al., 2022; Pernell et al., 1992; Sarierler et al., 2004; Vignoli et al., 2015,), but also in the tibia (Duval et al., 1995), rib (Biller et al., 1987), and pelvis (Nomura & Sato, 1997). Similar to humans, the majority of cases were observed in juvenile (<12 months) animals. The majority of cases were treated via surgical excision and/or curettage, followed by augmentation with bone graft or bone cement. More recently, Anson et al. (2020) reported the use of sclerotherapy with 95% ethanol, suggesting some treatments used in humans may also be extrapolated for the treatment of veterinary species. The use of sclerotherapy has also recently been reported for use in a mandibular ABC in the horse (Perez et al., 2022). Interestingly, three case reports in the equine literature present ABCs that affect the mandible (David et al., 2015; Perez et al., 2022; Steiner & Rendano, 1982,), which has not been reported in other species. The two more recent reports in the mandible demonstrate the value of CT in both the diagnosis, characterisation of the lesion, and in the surgical planning and treatment. The seemingly higher prevalence of mandibular ABCs, in comparison to ABCs of the limbs (Bryant et al., 2012; Thomas et al., 1997), demonstrates the horse may have unique morbidity challenges in the treatment of mandibular ABCs compared to other species where surgical excision in other locations is easier to facilitate. In addition to the aforementioned sclerotherapy, implantation of a collagen-hydroxyapatite scaffold has been successful in the treatment of mandibular ABCs. For ABCs affecting the appendicular skeleton, such as the case discussed in this issue of Equine Veterinary Education (Looijen et al., 2023), surgical treatment and curettage—especially in juvenile animals with rapidly modelling bone—may be sufficient for treatment and lesion resolution. Regardless of the location or species, the same principles of early and accurate diagnosis and treatment remain.

Aneurysmal bone cysts, although benign, are locally aggressive lesions if left untreated. Volumetric imaging, including CT and MRI, is highly valuable for comprehensive characterisation of these lesions and should be considered as the standard of care for all species. Lesion biopsy is likely still required for definitive diagnosis, and ABCs should remain as a differential diagnosis for these rapidly expanding bone-associated lesions that are primarily observed in young animals. The standard of treatment of curettage and bone grafting stems from the success with this therapy in humans, and although investigation of adjuvant or alternative treatment methods is numerous, prospective, comparative clinical trials are lacking at this time. Limited reports suggest there may be species-specific differences that require consideration for the equine patient. Although infrequently observed, the high level of morbidity (or even mortality) associated with ABCs warrants clinical awareness of these lesions across all species.

CONFLICT OF INTEREST

No conflicts of interest have been declared.

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Equine Veterinary Education

CASE REPORT

Trigeminal neuropathy in two horses following trauma to the infraorbital and mental nerves

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SUMMARY

Case history: Case 1: An 8-year-old Warmblood gelding presented for investigation of acute onset, intermittent headshaking following trauma to the right rostral maxillary region 10 days earlier. Case 2: A 6-year-old Welsh section B mare presented for investigation of facial pain, rubbing of the muzzle, difficulty eating and swelling of the right rostral hemi-mandible of 12-h duration.

Clinical findings: Case 1 had a small chronic skin wound just rostral to the right infraorbital foramen, with pain on palpation of the region and spontaneously headshaking. Case 2 had a focal painful enlargement of the right rostral hemi-mandible at the level of the interdental space, with tactile allodynia of the rostral right head.

Imaging findings: Computed tomography and ultrasonography of Case 1 showed a bony fragment adjacent to the infraorbital nerve associated with the wound. Computed tomography of Case 2 revealed a nondisplaced, comminuted fracture of the right hemi-mandible (Figure 1). Ultrasound showed an irregular outline to the mental foramen and multiple small bone fragments.

Diagnosis: Both horses were diagnosed with traumatic trigeminal neuropathy of the infraorbital nerve (Case 1) and the mental nerve (Case 2), associated with a small bony fragment adjacent to the infraorbital foramen in Case 1 and a nondisplaced, comminuted fracture of the right hemi-mandible at the level of the mental foramen in Case 2.

Treatment: Treatment consisted of surgical removal of the fragment in Case 1 and taking the incisors and rostral premolars out of occlusion to reduce shear forces on the fracture in Case 2. Both horses were treated with anti-inflammatories and multimodal analgesia.

Outcome: The fracture in Case 2 healed without requiring stabilisation. Clinical signs fluctuated in both cases before resolving with no reported signs of dysaesthesia or headshaking at 6 months (Case 1) and 4 months (Case 2) following diagnosis.

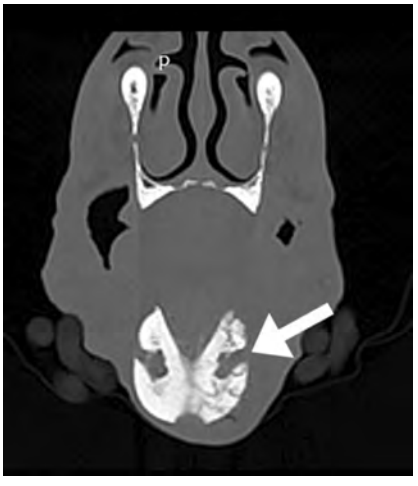

FIGURE 1 CT image from Case 2 showing the fracture of the right hemi-mandible involving the mental foramen (white arrow)

KEYWORDS

horse, headshaker, infraorbital, mental, trauma

Key points

- Traumatic neuropathy should be considered in horses presented for headshaking behaviour and/or facial dysaesthesia, particularly if there is evidence of trauma to the head.
- Severity of headshaking in horses with traumatic trigeminal neuropathy may fluctuate, and it may take several months to resolve.
- Computed tomography and ultrasonography can be useful in reaching a diagnosis in horses with suspected traumatic neuropathy. Differentiating horses with traumatic neuropathy from horses with idiopathic trigeminal mediated headshaking is important as treatment and prognosis may differ.

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Equine Veterinary Education

CLINICAL COMMENTARY

Trigeminal-mediated headshaking: A diagnostic challenge

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The term 'equine trigeminal-mediated headshaking' (TMHS) has replaced the previously used diagnosis of 'idiopathic headshaking' to reflect the clinical signs, which are considered to characterise trigeminal neuropathic pain (Pickles et al., 2014). This diagnosis is reserved for horses with a presumed functional, rather than structural, disorder of the trigeminal nerve, as distinct from horses such as those described in the case series by Ogden et al. (2023) where gross pathology, in this case trauma of trigeminal nerve branches, induces clinical signs of headshaking.

Other pathological or behavioural causes of headshaking behaviour, as listed by Ogden et al. (2023), have also been described. It is only by exclusion of these other causes, or via invasive neurophysiological studies which are outwith the realm of clinical practice, that a diagnosis of TMHS can be confidently assigned. A gross pathological cause of headshaking behaviour is only rarely diagnosed (Pickles et al., 2014), and therefore, it can be tempting to try and take shortcuts in the diagnostic procedure; however, if we do not look for these other causes, we will surely not find them. Indeed, Ogden et al. (2023) report that the traumatic skin lesion in one of their cases was disregarded as self-inflicted from headshaking-induced muzzle rubbing by the initial treating veterinary surgeon. Careful anamnesis, physical examination and often diagnostic imaging are therefore required for correct diagnosis (Pickles et al., 2014). Previously, 'idiopathic headshaking' was diagnosed in 97% of headshakers presenting to equine hospitals; however, with the advent of advanced imaging modalities such as computed tomography (CT), causative pathology is identified in up to 10% (Fairburn et al., 2023). This emphasises the merits of these ancillary diagnostic techniques and suggests that, perhaps, we may even still be overdiagnosing true, functional, trigeminal-mediated headshaking.

Conclusive involvement of the trigeminal nerve in headshaking has only been confirmed within the last 10 years (Aleman et al., 2013, 2014), although it was first proposed well over a century previously (Williams, 1897, 1899). Nerve conduction studies by Aleman et al. (2013, 2014) established a 10-fold reduction in the activation threshold of the maxillary branch of the trigeminal nerve in 6 headshaking horses compared with 6 control horses. Other neurophysiological parameters, including conduction velocity, were normal. This is in contrast to the cases of human trigeminal neuralgia where reductions in conduction velocity and ephaptic conduction occur due to demyelination of the trigeminal nerve caused by focal compression at the root entry zone (Devor et al., 2002). It is likely, given the traumatic aetiology, that neurophysiologic alterations would have been present in the cases reported by Ogden et al. (2023).

The aberrant trigeminal nerve function documented in TMHS, the seasonality of clinical signs in many headshaking horses and the absence of gross or histopathological lesions in the trigeminal nerves and ganglia from headshaking horses (Aleman et al., 2013; Newton, 2001; Roberts et al., 2017) distinguish it as a physiological or functional, rather than a structural pathological, nerve disorder. In further support of this hypothesis is the finding that the activation threshold of the trigeminal nerve in one horse with seasonal TMHS tested during a time of remission showed a threshold for activation similar to control horses (Aleman et al., 2014). Unfortunately, this horse was not tested during seasonal exacerbation of headshaking signs, which would appear to be the next logical step in elucidating any seasonal malleability of the activation threshold of the trigeminal nerve.

Quantitative sensory testing (QST) of the trigeminal nerve is a sensitive, clinical diagnostic aid used in human trigeminal neuralgia that allows accurate mapping of the areas of altered sensory perception (Maier et al., 2010). A standardised test is performed to determine thresholds for thermal and mechanical stimuli, pain thresholds for several stimulus modalities, suprathreshold pinprick tests and wind-up, plus a specific assessment for dynamic mechanical allodynia and paradoxical heat sensation. Calibrated stimuli are applied to capture perception and pain thresholds providing information on patterns of sensory loss (for the functioning of the thick and thin nerve fibres), as well as a gain of function (hyperalgesia, allodynia and hyperpathia) with simultaneous detection of cutaneous and

deep tissue sensibility, which can be compared against a reference population (Mücke et al., 2021). Such testing would be ideal to aid the diagnosis of TMHS; however, as it entails the cooperation of the subject being examined to report findings, it is challenging in the horse. The author has had difficulty achieving consistent results (unpublished data); however, Veres-Nyéki et al. (2021) report reliable measurements for QST of the equine face of healthy horses, which was well-tolerated. The face did not require shaving or clipping, but age had a significant effect on tactile sensory and mechanical and thermal nociceptive threshold ($p = 0.001$) with threshold values increasing with age. The most consistent values were reported over the nostril (tactile sensory threshold), temporomandibular joint (mechanical nociceptive threshold) and supraorbital foramen (thermal nociceptive threshold). Such testing of horses requires a subjective assessment of the horse's response to stimulation by an observer, rather than reporting by the individual themselves, and thus has limitations compared with humans. However, based on the results reported by Veres-Nyéki et al. (2021), the assessment of trigeminal sensory function, by von Frey filament stimulation of the nostril area, in control horses and horses with TMHS is warranted.

The hunt is now on for the cause of the aberrant trigeminal nerve activity in equine TMHS, which, thus far, remains frustratingly elusive. The role of gonadotrophin fluctuations (Sheldon et al., 2019a), dietary cation-anion balance (Sheldon et al., 2018, 2019b, 2019c) and caecal microbiota (Aleman et al., 2022) has recently been investigated for involvement in the aetiology of TMHS. Finally, after decades of inactivity, there has been a resurgence of interest in demystifying this syndrome.

CONFLICTS OF INTEREST

No conflicts of interest have been declared.

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Equine Veterinary Education BEVA AAEP

ORIGINAL ARTICLE

Successful management of an outbreak of Tyzzer's disease on a Thoroughbred breeding farm in central Kentucky; use of sorbitol dehydrogenase to identify sub-clinical cases

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Summary

Background: Tyzzer's disease is rare and usually fatal in the foal. Outbreaks are especially rare and have high educational value. Long-term recovery of multiple survivors and the use of sorbitol dehydrogenase (SDH) as a time-sensitive screening test during an outbreak have not been reported. The epidemiology of *Clostridium piliforme* acute necrotising hepatitis is enigmatic because the original source is unknown.

Objectives: The aims of this study were to present highly variable clinicopathologic findings, clinical presentation, progression and challenges, diagnostic and treatment protocols of two confirmed and two presumptive Tyzzer's cases, to determine the utility of plasma SDH measurement in the identification of subclinical or early Tyzzer's disease in the on-farm population and to identify carrier animals.

Study design: Case series. Prospective cohort study. Case–control study.

Methods: A cut-off value for SDH was determined based on values observed in the critically ill foals and applied to 39 on-farm foals (114 samplings). SDH values >20 IU/L prompted prophylactic treatment. Faecal PCR testing was performed on the surviving foals, the dams of all affected foals and the dams of foals with SDH values >20 IU/L. A control group of dams with matched foaling dates was sampled.

Results: The screening test identified one subclinical hepatitis case, in the youngest foal on the farm. No further clinical cases occurred. SDH lacked sensitivity in one confirmed case and specificity in foals >40 days of age. All faecal PCR samples were negative.

Main limitations: Sorbitol dehydrogenase sampling of on-farm foals may have been biased by economic value or athletic prospect.

Conclusions: Two foals were successfully treated; one is a successful athlete. Refractory hypoglycaemia presented the greatest clinical challenge and required aggressive treatment. The source of infection and carrier status remains unknown. SDH lacks specificity but might be the most sensitive test available during a Tyzzer's disease outbreak.

KEYWORDS
horse, acute necrotising hepatitis, foal, outbreak, sorbitol dehydrogenase, Tyzzer's disease

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Clinical relevance

- Tyzzer's disease is commonly fatal within 2–48 h and may occur alongside sepsis or gastrointestinal dysmotility, making presentation highly variable. Survival is possible with early, aggressive treatment. Early anticipation of refractory hypoglycaemia in affected foals, and disease outbreak on the farm, is essential.
- Two presumptive Tyzzer's cases were successfully treated in between two confirmed cases. Sorbitol dehydrogenase might be a useful time-sensitive screening tool for Tyzzer's disease in an outbreak scenario, although can lack specificity.
- Faecal PCR testing of survivors and the dams of all affected foals did not identify carrier animals; therefore, the source of *C. piliforme* remains unknown.

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ORIGINAL ARTICLE

Unintentional intracarotid injections in the horse—15 cases (2010–2020)

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SUMMARY

Background: Intracarotid injections are a well-recognised complication of jugular injections in the horse; however, little information is available about outcome and complications.

Objectives: To describe venepuncture techniques, short- and longer-term complications related to intracarotid injections.

Study design: Case series.

Methods: The survey was distributed through the American College of Veterinary Internal Medicine (ACVIM) mailing list and a veterinary social media site. Data collected in the questionnaire included injection technique, drug administered, personnel performing the injection, clinical signs and short and longer-term complications.

Results: Accidental intracarotid injections were most commonly performed by veterinarians (n = 9/13). The majority of reported accidental intracarotid injections occurred during routine sedation in healthy horses (n = 8/13) and occurred when the jugular vein was accessed at the middle jugular groove (n = 6/13). Most events occurred while using longer needles (1.5 inches; n = 10/13) of 20-G calibre (n = 9/13), attached to the syringe (n = 11/13) and against jugular blood flow (n = 11/13). Short-term injuries included superficial abrasions (n = 13/13) and cardiac ailments (3/13). One of the 15 horses described in this report died. Longer-term complications were not reported for the remaining 14 horses.

Main limitations: Potential selection, recall and response bias, putative risk factors identified cannot be correlated with an increased risk of carotid injection without a control group.

Conclusion: Accidental intracarotid injections led to seizures but rarely resulted in death of the patient. Premonitory clinical signs occurred while or soon after injection and can be used for early identification of this inadvertent injection and to establish safety measures for the horse and handlers.

KEYWORDS
horse, accidental injection, inadvertent injection, injection complication, seizures

Clinical relevance

- Accidental intracarotid injections led to seizures but did not result in death of the patient when a sedative was used.
- Premonitory clinical signs such as flared nostrils, sweating and muscle tremors occurred in most cases soon after injection and can be used for early identification of this inadvertent injection.
- All cases had seizures after the injection, but subsequent complications were minor.

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ORIGINAL ARTICLE

Conventional imaging is useful for assessment of equine pharyngeal squamous cell carcinoma but underestimates bone involvement

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SUMMARY

Background: Squamous cell carcinoma is the most common pharyngeal neoplasm but is poorly documented with diagnostic imaging in the veterinary literature.

Objectives: To describe the diagnostic imaging findings in horses with confirmed pharyngeal squamous cell carcinoma.

Study design: Retrospective case series.

Methods: Horses with a definitive diagnosis of pharyngeal squamous cell carcinoma based on in-situ biopsy and/or cytology were identified in two different centres. Multimodality imaging findings are reported.

Results: Six equids were included. On radiographic examination, increased pharyngeal opacity and reduction of pharyngeal lumen were observed in all horses, centred on the oropharynx (three cases), the laryngopharynx (two cases) or the nasopharynx (one case). A clearly delineated mass was visualised in four cases. Additional radiographic findings were border effacement of the epiglottis (5/6) or soft palate (3/6), thickening of the pharyngeal walls (4/6) or soft palate (3/6) and suspected retropharyngeal lymphadenomegaly (3/6). Ultrasonography was useful to highlight retropharyngeal (4/6) and mandibular (4/6) lymphadenopathy suggestive of metastatic dissemination. Computed tomographic images were available for two horses and detected bone lysis not visible on radiographs.

Main limitations: The number of cases was low and computed tomography was not realised in all cases.

Conclusions: Findings support the usefulness of radiography and ultrasonography in horses with suspected pharyngeal neoplasia for a first-line imaging diagnosis, in particular when endoscopy is impaired by a mass effect or dyspnoea. Computed tomography gives a more accurate assessment of bone involvement.

KEYWORD
horse, computed tomography, pharynx, radiography, squamous cell carcinoma, ultrasonography

Clinical relevance

- Radiography and ultrasonography of the pharynx are useful to characterise location, extent and invasiveness in horses with suspected pharyngeal squamous cell carcinoma, particularly when endoscopy is impaired by mass effect or respiratory distress.
- Ultrasonography is complementary to radiography to confirm lymphadenopathy and guide fine needle aspirations.
- Conventional imaging underestimates bone lysis and osseous metastasis

Previous presentations: The preliminary findings described in this article have been used to produce a poster at the EVDI congress (2014) and the Voorjaarsdagen congress (2018).

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Equine Veterinary Education

ORIGINAL ARTICLE

Computed tomographic findings in 101 horses presented for the investigation of headshaking

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SUMMARY

Background: The majority of horses presenting to a hospital with signs of headshaking receive a diagnosis of trigeminal-mediated headshaking. This diagnosis is one of exclusion, so ruling out other possible causes of headshaking is essential.

Objectives: The aims of this study were to describe the computed tomographic (CT) findings in horses that were presented for investigation of headshaking and underwent CT examination of the head under standing sedation. A secondary aim was to establish whether the proximity of the apices of the caudal maxillary cheek teeth to the infra-orbital canal varied between age-matched groups of horses presented for CT examination for investigation of headshaking and those presented for investigation of other conditions.

Study design: Retrospective case series of horses undergoing CT examination of the head as part of the investigation of headshaking at the Equine Centre, University of Bristol.

Methods: Case records of horses that had standing CT images of the head obtained, reviewed and reported at the Equine Centre, University of Bristol, over a five-year period (February 2012–March 2017) were reviewed. Cases that had presented for investigation of headshaking were included. The proximity of the apices of the maxillary cheek teeth to the infra-orbital canal was assessed in age-matched horses presenting for CT for investigation of headshaking and other reasons.

Results: A total of 101 horses presented for investigation of headshaking were included. There were four horses in which CT detected likely causative pathology for the headshaking, which had not been identified by other diagnostic tests; however, radiographs had not been obtained in two of these horses. Three horses had periapical infection of the maxillary cheek teeth, and one horse had a fracture of the paracondylar process of the occipital bone. Clinical signs resolved following treatment in three horses; two with periapical infection and one with a paracondylar process fracture. One horse with periapical infection was lost to follow-up. There was no significant difference in the proximity of the apices of the maxillary cheek teeth to the infra-orbital canal between the headshaking and non-headshaking groups.

Main limitations: Retrospective study; pre-selection of cases through discussion of the case and, in some instances assessment of videos of the behaviour, by the last author, prior to referral; lack of radiographs.

Conclusions: Whilst trigeminal-mediated headshaking is the most common cause of headshaking in horses, it is a diagnosis of exclusion so thorough investigation to rule out other possible causes is essential. We consider CT examination of the head to be a valuable tool in this investigation although the diagnostic yield is likely to be low.

KEYWORDS
horse, computed tomography, headshaking, paracondylar process, periapical infection

Clinical relevance

- Computed tomography (CT) is a valid technique for the investigation of horses presenting with headshaking.
- Computed tomography identified likely causative pathology for the headshaking in 4/101 horses. Periapical disease of the maxillary cheek teeth was identified in three horses, and a fracture of the paracondylar process of the occipital bone was found in one horse.
- Headshaking resolved following treatment in two horses with maxillary periapical disease and the horse with a fracture of the paracondylar process of the occipital bone. The third horse with a periapical disease of a maxillary cheek tooth was lost to follow-up.

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Equine Veterinary Education BEVA AAEP

REVIEW ARTICLE

Post-anaesthetic spinal cord myelomalacia in horses: A review and a presumptive clinical case presentation

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Summary

This article reviews the previous reports of post-anaesthetic spinal cord myelomalacia (PSCM) in horses and summarises the speculated pathogenesis, potential risk factors, clinical signs, differential diagnosis, histopathology findings, treatment and prognosis of this post-anaesthetic complication. Furthermore, a clinical case of suspected PSCM is presented.

KEYWORDS
horse, anaesthesia, complication, myelomalacia

INTRODUCTION

Post-anaesthetic spinal cord myelomalacia (PSCM) is a rare but life-threatening complication in horses (Wan et al., 1994). The perianaesthetic equine mortality rate after healthy elective surgeries ranges between 0.6% and 0.9% (Gozalo-Marcilla et al., 2021; Johnston et al., 2002): Only 0.03%–0.19% was associated with PSCM (Dugdale et al., 2016; Gozalo-Marcilla et al., 2021; Jago et al., 2015).

A literature review using the keywords of "equine," "horse," "myelomalacia" and "myelopathy" on PubMed and Google Scholar database was performed: 33 suspected PSCM cases in horses were found. The first case was described in 1979 (Schatzmann et al., 1979); since then, another 32 cases were reported, and only five of these cases occurred in the United Kingdom (Blakemore et al., 1984; Brearley et al., 1986; Hughes et al., 2019; Joubert et al., 2005; Küls & Rocchi, 2017; Lam et al., 1995; Lerche et al., 1993; Patschova et al., 2014; Ragle et al., 2011; Raidal et al., 1997; Van Loon et al., 2010; Wan et al., 1994; Yovich et al., 1986; Zink, 1985).

The aim of this review is to summarise the previously described cases of PSCM in horses and to provide current knowledge regarding the suspected pathogenesis, potential risk factors, clinical signs, differential diagnosis, histopathology findings, treatment and prognosis. Furthermore, a case of suspected PSCM in a horse is presented.

PATHOGENESIS

The pathogenesis of PSCM is not fully understood, and the aetiology is most likely multifactorial (Wan et al., 1994). The nature and location of PSCM are consistent with a poliomyelopathy and congestion of vessels following a perianaesthetic hypoperfusion and ischaemia of the lower spinal cord, resulting in necrosis of the spinal cord neuroectodermal cells (Blakemore et al., 1984; Brearley et al., 1986; Lerche et al., 1993; Ragle et al., 2011; Raidal et al., 1997; Schatzmann et al., 1979; Wan et al., 1994; Zink, 1985). The ischaemic damage might also produce oedema and haemorrhage with consequential degeneration of the spinal neurons (Schatzmann et al., 1979).

The exact mechanism leading to the spinal cord hypoperfusion is still unknown (Ragle et al., 2011). A reduction in cardiac output and arterial blood pressure and congestion of the venous system caused by the weight of the abdominal viscera were suggested as possible causes (Brearley et al., 1986; Schatzmann et al., 1979; Wan et al., 1994). However, cardiac output was not measured in any of the reported 33 PSCM cases (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011) while arterial blood pressure was well maintained in most of the cases (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011). Furthermore, even if the congestion of the venous system commonly occurs in dorsal recumbent anaesthetised horses, the incidence of PSCM development is low (Trim, 1997). Therefore, a direct mechanistic relation between low cardiac output and arterial blood pressure, venous congestion and PSCM development cannot be made.

Arterial vasoconstriction of the intramedullary vessels of the spinal cord could be another potential reason for developing PSCM. It could be caused by a sudden increase in catecholamine levels and concomitant persistent sudden increase in arterial blood pressure (Lerche et al., 1993; Patschova et al., 2014; Zink, 1985). However, it might be possible that both catecholamine release and arterial

vasoconstriction are a consequence of primary spinal cord ischaemic damage (Patschova et al., 2014) rather than a triggering factor. Therefore, it is unclear if the spinal cord ischaemia is the cause or a consequence of the increased adrenergic state (Patschova et al., 2014). A sudden but persistent increase in arterial blood pressure (mean arterial pressure [MAP] > 115 mmHg or systolic arterial pressure > 121 mmHg), reported in four cases, and tachycardia (heart rate [HR] > 60 beats/min), reported in two cases, both not associated with surgical manipulation or evident signs of light plane of anaesthesia, were considered suspicious of PSCM (Brearley et al., 1986; Küls & Rocchi, 2017; Lerche et al., 1993; Patschova et al., 2014;). Profuse sweating was also observed in three cases during general anaesthesia in horses that developed PSCM and may be indicative of a prolonged period of catecholamine release from the adrenal medulla. Therefore, profuse sweating was presumed to be a metabolic response to the cardiovascular events potentially leading to PSCM in horses anaesthetised in dorsal recumbency (Patschova et al., 2014; Schatzmann et al., 1979; Zink, 1985). Spinal cord infarction secondary to a fibrocartilaginous embolus causing a local perturbation to the blood flow was also suggested as a possible underlying mechanism (Trim, 1997).

Vitamin E and selenium were measured in two horses with suspected PSCM: While vitamin E was deficient in both of them, low levels of selenium were found in one horse (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011). Vitamin E and selenium are essential components of the oxidative metabolic pathway (Lerche et al., 1993; Ragle et al., 2011; Stolk et al., 1991). Hypovitaminosis E, in particular, might destabilise the spinal cord membranes due to a lipoperoxidative process, increasing the susceptibility of the spinal cord to the hypoxic damage (Stolk et al., 1991). Further investigations are required to evaluate the presence of a cause-effect relationship between PSCM and low vitamin E and selenium levels in horses.

RISK FACTORS

Prevention of PSCM is impossible due to the likely multifactorial and unknown pathogenesis. This, in combination with difficulties in management of the condition, is responsible for a poor prognosis for survival (Patschova et al., 2014; Ragle et al., 2011). Knowledge of the risk factors could be important to identify the animals at risk and to inform the owner exhaustively.

Age, gender and horse conformation can be considered important risk factors, although variability exists (Table 1): PSCM was mainly described in healthy young (24 out of 33 were ≤2 years old), heavily muscled or large-framed male horses. According to Schatzmann et al. (1979), the immature microcirculation of the spinal cord of young horses might not be able to compensate for the pressure changes associated with dorsal recumbency and general anaesthesia. However, PSCM was also reported in adult horses (Küls & Rocchi, 2017; Ragle et al., 2011). No breed disposition was found, although Friesians represented 18% of the cases (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011).

The type of surgery (10 out of 33 PSCM cases underwent orthopaedic surgery, 22 out of 33 PSCM cases soft-tissue surgery, 1 case went through both of them) was not considered a remarkable risk factor, and all of them, except one, were elective surgeries (Hughes et al., 2019; Küls & Rocchi, 2017; Lam et al., 1995; Patschova et al., 2014; Ragle et al., 2011). Regardless of the type of surgery, PSCM developed predominantly in horses placed in dorsal recumbency (32 out of 33 cases). For this reason, dorsal recumbency has been considered an important risk factor for the development of PSCM. Similarly, PSCM was also described in a young calf anaesthetised for evacuation of abomasum in dorsal recumbency (de Lahunta & Glass, 2009). However, while the pressure of the abdominal viscera compressing the caudal vena cava severely reduces venous return, cardiac output and causes venous congestion (Blakemore

TABLE 1 Previously reported cases of myelomalacia in horses

Information	*n* = 33	
Age (months)	18 (6–120)	
Sex (*n*)	24 Male, 7 Female, 2 Gelding	
Weight (kg)	400 (214–650)	
Most representative breeds (*n*)	6 Friesian, 4 Quarter horse, 3 Shire, 3 Clydesdale	
Most common surgical procedures (*n*)	10 arthroscopy, 9 cryptorchidectomy, 4 umbilical herniorrhaphy	
Most common anaesthetic drugs used (*n*)	Premedication	18 xylazine, 13 acepromazine, 7 butorphanol
	Induction	25 ketamine, 14 guaifenesin, 11 diazepam
	Maintenance	17 halothane, 11 isoflurane
Recumbency during anaesthesia (*n*)	30 dorsal, 2 dorsal with 10–20° angle to the right, 1 right lateral	
Anaesthesia time (min)	90 (25–205)	
End of anaesthesia to euthanasia/death (h)	17.5 (2–192)	

Note: Data are reported as median (range) where appropriate.
Abbreviation: *n*, number.

et al., 1984; Wan et al., 1994; Yovich et al., 1986), it is not clear why PSCM does not occur more frequently in horses undergoing colic surgery. It is possible that while the gross gas distension of the viscera in a colicking horse increases the intra-abdominal pressure and shifts the diaphragm cranially (Boesch, 2013), the overall weight of the viscera and its direct compressive effect on the caudal vena cava are not much greater compared with a noncolicking horse placed in dorsal recumbency. To minimise the compressive effect, it was suggested to position horses slightly oblique but, despite this precaution being used in two cases, PSCM occurred regardless (Blakemore et al., 1984; Ragle et al., 2011; Wan et al., 1994). One case of PSCM was also reported in a horse placed in lateral recumbency (Raidal et al., 1997). To decrease the overall weight of the intestine and the compression on the caudal vena cava, it was suggested to starve the horse from solid food for a period of at least 12 h (Lerche et al., 1993).

Anaesthesia is usually uneventful, and no association between PSCM and a specific anaesthetic technique was found (Hughes et al., 2019; Joubert et al., 2005; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011). Alpha-2 agonists (i.e. xylazine) were the most common pre-anaesthetic medication used (Table 1). They could have potentially participated in the development of PSCM by causing vasoconstriction and reducing cardiac output (Ringer et al., 2013). In addition, volatile anaesthetic agents and the body position during the surgery could have adversely affected the cardiovascular system, possibly contributing to the development of PSCM. Cardiac output and arterial blood pressure decreased significantly over time in dorsally recumbent isoflurane anaesthetised horses (Blissitt et al., 2008), but arterial blood pressure was well maintained in most of the described cases (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011).

In the 33 horses that developed PSCM, the median (range) time of general anaesthesia was 90 (25–205) min. Because of this wide range, time of general anaesthesia was not considered an important risk factor (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011).

CLINICAL SIGNS

Horses affected by PSCM showed progressive worsening flaccid pelvic limb paresis/paralysis with absence of patellar reflexes. These clinical signs are representative of a lumbosacral spinal cord damage. However, a thoracic spinal cord involvement was also described in few cases. Generally, while mentally alert, horses with suspected PSCM tended to assume a dog sitting-like position during recovery from general anaesthesia but were unable to stand (Blakemore et al., 1984; Joubert et al., 2005; Lam et al., 1995; Lerche et al., 1993; Patschova et al., 2014; Ragle et al., 2011; Raidal et al., 1997; Van Loon et al., 2010; Yovich et al., 1986; Zink, 1985). Lack of voluntary movement of the pelvic limbs was the first recognised sign in 26 of 33 horses (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011). The neurological function of the neck and of the thoracic limbs was usually normal, but with time, the thoracic limbs may become paretic (Joubert et al., 2005). In a few instances, horses were able to stand at first, but collapsed later (Raidal et al., 1997; Zink, 1985). Two reports of initial improvement were also reported (Hughes et al., 2019; Zink, 1985), possibly due to initial treatment with corticosteroids reducing spinal cord inflammation and oedema (Hughes et al., 2019). Moreover, in four occasions, the same horse underwent two surgeries: While it was able to stand after the first one, it did not after the second one (Brearley et al., 1986; Lam et al., 1995; Ragle et al., 2011; Van Loon et al., 2010). In three of them, surgeries were performed within 24 h; in one case, the time between surgeries was 8 days (Brearley et al., 1986; Lam et al., 1995; Ragle et al., 2011; Van Loon et al., 2010).

Only one severe PSCM presentation was reported: The horse was unable to move from lateral position to sternal (Patschova et al., 2014). Usually, horses were able to move into sternal and rise with the thoracic limbs, without being able to push with the pelvic limbs (Joubert et al., 2005). Loss of strength to stay in sternal was reported in a horse the day after the surgery (Joubert et al., 2005).

Other less common clinical signs are paralysis of intercostal muscles, reported in one horse, and sweating in recovery, reported in two cases (Hughes et al., 2019; Ragle et al., 2011; Yovich et al., 1986). Sweating, starting at the mid-thorax and extending caudally, was probably caused by sympathetic paralysis following focal loss of function of the preganglionic sympathetic neurons in the spinal cord (de Lahunta & Glass, 2009). In humans, changes in sweat production (hyper-, hypo- or anhidrosis) often occur after spinal cord injuries and sweating occurring exclusively below the level of the injury is usually a symptom of a massive autonomic response (Hagen, 2015). Therefore, presence of well delineated area of sweat might help to diagnose and localise a spinal cord lesion (de Lahunta & Glass, 2009).

DIFFERENTIAL DIAGNOSIS

Post-anaesthetic spinal cord myelomalacia needs to be differentiated from post-anaesthetic myopathies and/or neuropathies, and fractures (Joubert et al., 2005; Küls & Rocchi, 2017; Ragle et al., 2011). Post-anaesthetic myopathies are associated with decreased muscle perfusion related to hypotension, prolonged anaesthetic duration, lateral recumbency and larger body mass (Deutsch & Taylor, 2021). Raised muscle enzyme levels and the presence of myoglobinuria might develop and could be used to support the diagnosis of post-anaesthetic myopathies (Grandy et al., 1987; Trim, 1997). Furthermore, in the presence of a post-anaesthetic myopathy, the affected muscle(s) might be painful, firm and swollen on palpation (Deutsch & Taylor, 2021; Young, 2005). A horse with a bilateral femoral nerve paralysis may show pelvic limb motor deficits similar to a horse with PSCM (Küls & Rocchi, 2017). However, in horses with PSCM, the withdrawal reflex, anal and tail tone, and pelvic limb deep pain sensation might be lost (Dyson et al., 1988; Ragle et al., 2011). Fractures are usually extremely painful with swelling of the affected

area and tend to be associated with a fall during the recovery phase (Joubert et al., 2005).

HISTOPATHOLOGY

Histological examination was performed in 30 cases out of 33 PSCM cases. The most consistent histopathological finding was poliomyelomalacia, varying in severity from degeneration to severe necrosis of neurons (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011). Oedema and haemorrhage of the grey matter and vessel congestion were the most variable findings (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011). The difference in severity of the neuronal degeneration found on histological examination in horses with PSCM was likely to be associated with the duration of the condition prior to euthanasia (Hughes et al., 2019). Changes occurred at different levels of the spinal cord, from cervical to sacral spinal cord (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011). Histopathological findings of the horses placed in right lateral or right oblique recumbency were confined to the right (dependent) side of the cord, consistent with compression or occlusion of venous outflow (Raidal et al., 1997; Wan et al., 1994). Therefore, supporting the theory that haemodynamic disturbances play a primary role in the development of the disease (Raidal et al., 1997).

The grey matter of the spinal cord might be more affected than the white matter as it has a greater vascularity and metabolic rate; therefore, it is more sensitive to haemorrhage and hypoxia (Yovich et al., 1986; Zink, 1985). Oedema and haemorrhage of the grey and white matter were found in 14 and 8 out of 30 horses, respectively (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011). This would suggest an interference with delivery of oxygenated blood to the spinal cord, further adding to the theory of ischaemia (Yovich et al., 1986; Zink, 1985). In an experimental study in mice in which the spinal cord blood supply was cut, the ischaemic damage occurred more extensively in the grey matter (Lang-Lazdunski et al., 1999).

TREATMENT

Fluid therapy, dantrolene, corticosteroids, nonsteroidal anti-inflammatory drugs, dimethyl sulfoxide, calcium, vitamin E and sedatives were previously used in different combinations, also depending on their availability (Hughes et al., 2019; Joubert et al., 2005; Küls & Rocchi, 2017; Lam et al., 1995; Lerche et al., 1993; Patschova et al., 2014; Ragle et al., 2011; Raidal et al., 1997; Zink, 1985). All the treatments instituted were palliative in nature and not evidence based, mainly directed towards relieving the spinal cord inflammation (Muir & Hubbell, 2009). In some cases, no medications were administered and only supportive care (i.e. padding, rolling) was given due to prolonged recumbency (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011).

Horses with PSCM might try to stand repeatedly, increasing the risk of self-trauma (Nout & Reed, 2005; Ragle et al., 2011). Moreover, they might become easily stressed as they do not tolerate the loss of function of their pelvic limbs. For this reason, the administration of sedative or tranquilisers might be indicated, and in some cases, romifidine (Hughes et al., 2019; Küls & Rocchi, 2017), xylazine (Hughes et al., 2019; Lerche et al., 1993; Patschova et al., 2014) and acepromazine (Hughes et al., 2019; Lerche et al., 1993) were administrated.

PROGNOSIS

Overall, recumbent horses independent of the cause have a poor prognosis with a mortality rate of 74%, with 76% of those horses being subjected to euthanasia or death within the first 3 days (Winfield et al., 2014). All reported horses with suspected PSCM died or were subjected to euthanasia from a few hours to 8 days postoperatively (Hughes et al., 2019; Küls & Rocchi, 2017; Patschova et al., 2014; Ragle et al., 2011).

CASE PRESENTATION

A 4-year-old, gelding, 638 kg, Irish Cob—Irish Sports Horse cross, was referred for surgical removal of fragmentations from both tarsocrural joints. Clinical examination was unremarkable. Preoperative blood analysis was deemed unnecessary. The horse's physical status was classified as one according to the American Society of Anesthesiologists. According to the passport, the horse was excluded from the human food chain consumption. The horse was fasted for 12 h.

On the day of the surgery, a 14-gauge intravenous (IV) cannula (MilaCath; Mila International, Inc.) was placed in the left external jugular vein under subcutaneous local anaesthetic infiltration (1 ml of 2% mepivacaine; Intra-Epicaine; Dechra Pharmaceuticals PLC). Enrofloxacin (5 mg/kg bwt; Baytril; Bayer UK Ltd.), phenylbutazone (4 mg/kg bwt; Equipalazone; Dechra Pharmaceuticals PLC), acepromazine (15 μg/kg bwt; Tranquinervin; Dechra Pharmaceuticals PLC) were administrated IV. Approximately 30 min later, the horse was walked into a rubber padded induction/recovery box and sedated IV with romifidine (66 μg/kg bwt; Sedivet; Boehringer Ingelhaim UK Ltd.). Five minutes later, general anaesthesia was induced IV with ketamine (2.5 mg/kg bwt; Ketavet; Zoetis UK Ltd.) and diazepam (0.05 mg/kg bwt; Diazepam; Hameln Pharma Ltd.), mixed in the same syringe. No complications occurred during induction of anaesthesia. After orotracheal intubation with a 30 mm internal diameter cuffed rubber tube, the horse was hoisted to an adequately padded equine operating theatre table (Haico Telgte II; Haico OY) and placed in dorsal recumbency. Care was taken to fit the horse well on the table, not to put any part of the body under strain, and pelvic limbs were kept flexed for the entire anaesthetic time. General anaesthesia was maintained with isoflurane (Iso-Vet; Chanelle Pharma) vapourised in 100% oxygen and delivered using

a large animal circle breathing system. Immediately after induction of anaesthesia, dobutamine (0.5 μg/kg/min; Dobutamine; Hameln Pharma Ltd.) continuous infusion was started, and the rate adjusted at to maintain the MAP >70 mmHg (Young, 2005). Romifidine constant rate infusion (CRI) was also started at 40 μg/kg/h. The right facial artery was catheterised with a 20-gauge cannula (Beckton Dickinson UK Ltd.), and the arterial blood pressure transducer was positioned at the level of the shoulder and zeroed to atmospheric pressure. Presence of arterial waveform damping was assessed performing a flush test (Kleinman, 1989). Capnography, end-expiratory gas analysis, pulse-oximetry (SpO_2), invasive arterial blood pressure and electrocardiography were monitored and recorded every 5 min (Datex-Ohmeda S/5; Datex-Ohmeda Ltd.). The anaesthetic depth was assessed by evaluating the cervical muscle tension, the position of the eyes and the presence/absence of palpebral reflex and nystagmus. The horse's lungs were mechanically ventilated to maintain an end-expiratory carbon dioxide partial pressure ($PE'CO_2$) of 35–45 mmHg using a pressure-controlled modality (LAVC-2000-D; JD Medical Dist. Co., Inc.): Peak inspiratory pressure was set at 24 cmH_2O resulting in a tidal volume of 6 L, respiratory rate (*f*R) was 10–11 breaths/min, and positive end-expiratory pressure of 7.5 cmH_2O was applied. Hartmann's solution (Vetivex 11; Dechra Pharmaceuticals PLC) was infused as fast as gravity allowed (approximately 5.5 ml/kg/h). Ten minutes after induction of anaesthesia, ketamine (0.5 mg/kg bwt) and romifidine (8 μg/kg bwt) were administrated IV as nystagmus was present. At 20 min from induction of anaesthesia, the infusion of dobutamine was discontinued as the MAP was 97 mmHg. No more dobutamine was needed as MAP remained >70 mmHg throughout the anaesthesia. An arterial blood sample was collected and analysed (i-STAT; Abaxis UK Ltd.) 35 min after induction of anaesthesia, and the results were unremarkable (Table 2). Romifidine CRI was stopped at the end of anaesthesia. The median (range) end-expiratory isoflurane concentration during anaesthesia was 1.3% (1.1–1.4), $PE'CO_2$ was 34 mmHg (32–40), MAP was 85 mmHg (82–97), HR was 32 beats/min (32–35), and SpO_2 was 97% (97–99). General anaesthesia and surgical time were 55 and 30 min, respectively.

The horse was hoisted to the induction/recovery box where it was positioned in right lateral recumbency with the right thoracic limb extended forward. It regained spontaneous ventilation immediately and the trachea was extubated 7 min later. Acepromazine (7.5 μg/kg bwt) and romifidine (12 μg/kg bwt) were administrated IV to smooth the recovery. The horse was left alone 45 min after the end of general anaesthesia when the first attempt to move to sternal position was made, unsuccessfully. The horse was able to raise only the head and neck, and it was paddling with the thoracic limbs intermittently. No movement of the pelvic limbs was noted. On palpation of the hindquarter muscles, no evidence of swelling, stiffness or pain was noted. Neurological examination revealed lack of anal and tail tone and loss of deep pain sensation of both pelvic limbs. Excessive sweating caudally from caudal thoracic region, sharply demarcating at the level of the presumed spinal cord injury (negative *cutaneous trunci* reflex), was also evident (Figure 1). Blood creatinine phosphokinase and aspartate aminotransferase were measured, and values were within reference intervals (Table 2). The clinical symptoms were consistent with ascending PSCM. Dexamethasone (0.15 mg/kg bwt; Dexa-Ject; Dopharma) and hypertonic saline 7.2% (4 ml/kg; Hypertonic; Dechra Pharmaceuticals PLC) were administrated IV. Oxygen (10 L/min) was supplemented inserting a cannula into the left nostril. As the horse was becoming exhausted and stressed by trying to move to sternal recumbency (HR raised from 36 to 56 beats/min and *f*R from 12 to 16 breaths/min), acepromazine (15 μg/kg bwt) was repeated IV 2 h after the end of general anaesthesia. A 24 Fr indwelling urinary catheter (Portex Horse Catheter; Portex Ltd.) was placed, and 4 litres of yellow-coloured urine were drained. Over the course of the day, no clinical improvement was observed and in view of the grave prognosis, and the horse was subjected to euthanasia 5 h after the end of general anaesthesia. Postmortem examination was declined by the horse's owner.

TABLE 2 Blood parameters measured in a horse with myelomalacia

Parameter	Value	Reference interval
Fraction of inspired oxygen (%)	88	
Oxygen saturation (%)	97	
End-expiratory carbon dioxide pressure (mmHg)	34	
pH	7.45	7.39–7.52
Partial pressure of carbon dioxide (mmHg)	49	35.5–51.7
Partial pressure of oxygen (mmHg)	335	90–108
Bicarbonate (mmol/L)	34	27.4–33.8
Base excess (mmol/L)	10	(−5)–(+5)
Arterial oxygen saturation (%)	100	>90
Sodium (mmol/L)	135	132–140
Potassium (mmol/L)	3.6	3.1–4.4
Ionised calcium (mmol/L)	1.47	1.35–1.68
Glucose (mmol/L)	13.6	3.9–6.5
Creatinine phosphokinase (IU/L)	288	100–300
Aspartate aminotransferase (IU/L)	355	150–600

Note: Reference values for parameters except creatinine phosphokinase and aspartate aminotransferase: Zoetis (2020) 'i-STAT 1 cartridge test reference ranges' https://www.zoetisus.com/products/diagnostics/vetscan/pdf/i-stat-1-cartridge-test-reference-ranges-sellsheet-vts-00029.pdf (Accessed 24 January 2022). Reference values for creatinine phosphokinase and aspartate aminotransferase: instrument specific reference values.

CONCLUSION

Post-anaesthetic spinal cord myelomalacia is a rare, but fatal complication, with an unknown aetiology. Some predisposing factors have been identified: young age, male gender, heavier conformation and dorsal recumbency. Neither hypertension nor hypotension were

FIGURE 1 Horse in the recovery box in right lateral recumbency. Excessive sweating caudally from the caudal thoracic region

associated with PSCM. It is most likely that a localised reduction in the spinal cord blood flow triggers PSCM. This event can occur at any point of the anaesthesia, and it is not associated with the duration of anaesthetic. The lack of a full understanding of the pathogenesis makes prevention of PSCM not always possible and recommendations difficult to make.

AUTHOR CONTRIBUTIONS

I. Viilmann was responsible for drafting the manuscript. M.R.W. Smith was responsible for reviewing and editing. E. Vettorato was responsible for reviewing and editing. All authors approved the final revision.

CONFLICT OF INTEREST

No conflict of interest has been declared.

ETHICS STATEMENT

No ethics approval was required as this manuscript is a review article.

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Equine Veterinary Education

REVIEW ARTICLE

Poor performance in the horse: Diagnosing the non-orthopaedic causes

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Summary

Poor performance and gait deficits in the equine athlete are most commonly caused by lameness of orthopaedic origin; however, there are numerous other potential sources that should be taken into consideration. This review describes the clinical signs and diagnostic tests for non-orthopaedic causes of poor performance and gait deficits in the equine athlete.

KEYWORDS
horse, gait deficits, non-orthopaedic, poor performance

INTRODUCTION

Poor performance can encompass various clinical signs including reluctance to work, exercise intolerance, a decline in the ability to perform certain athletic tasks, and sometimes the horse is not meeting expectations. Top performance requires excellent function and coordination between all body systems. Lower-level performance is often possible despite suboptimal functioning of one or more systems; however, for peak performance there is no room for error. Because the complaint of 'poor performance' is vague, diagnosis of the specific cause or causes can be challenging (Martin et al., 2000). Often a thorough evaluation of multiple body systems is needed. Lameness of orthopaedic origin is the most common cause of poor performance in the horse (Fraipont et al., 2011; Knight & Evans, 2000; Martin et al., 2000); however, there are numerous other potential sources that should be taken into consideration. This review will describe clinical signs and diagnosis of common respiratory, cardiovascular, muscular, neurological and gastrointestinal causes of poor performance. Figure 1 shows a summary of conditions and diagnostic testing methods for the various body systems that, when affected, can lead to poor performance.

RESPIRATORY CAUSES OF POOR PERFORMANCE

Equine asthma syndrome

Presentation

Equine asthma syndrome is a chronic respiratory disease syndrome characterised by excess tracheobronchial mucus and cough. Equine asthma is a more recent term that encompasses both recurrent airway obstruction (RAO) and inflammatory airway disease (IAD). Previously thought to be unrelated diseases, it is now recognised that RAO and IAD present a spectrum of the same disease.

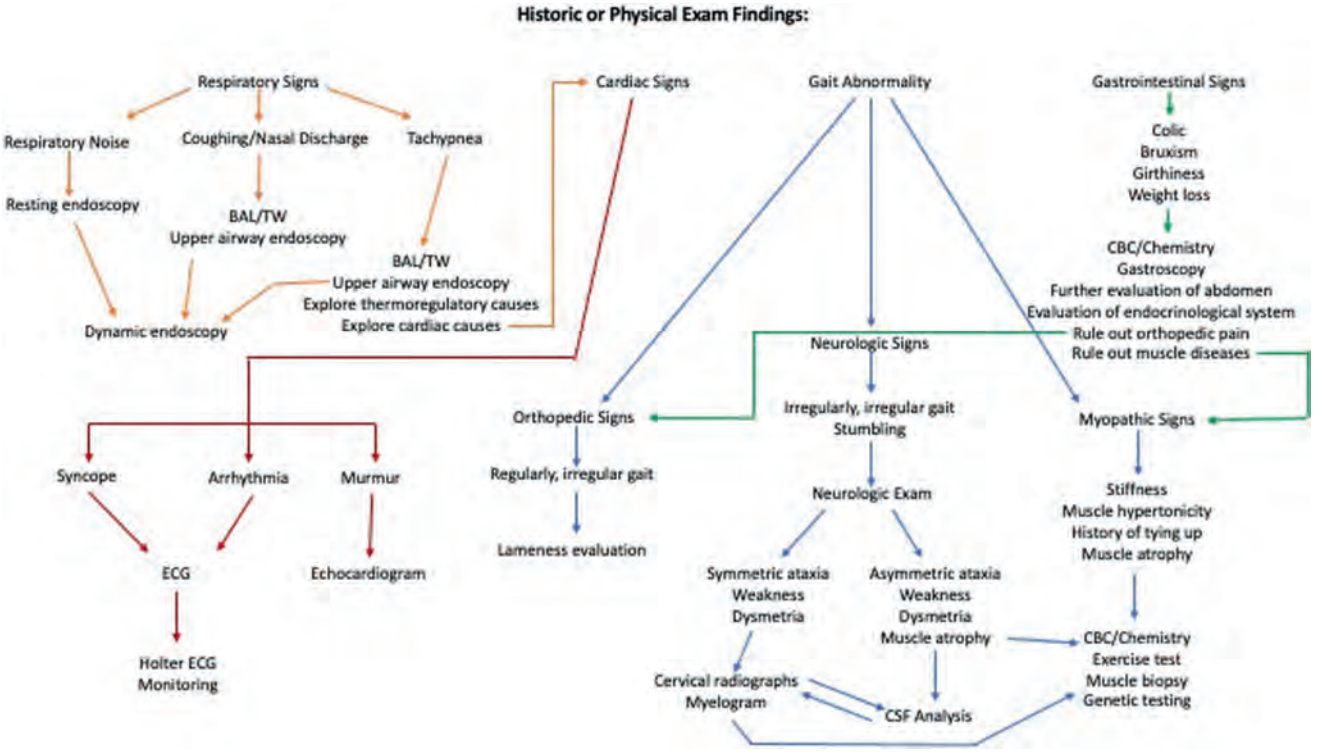

FIGURE 1 Flow chart summary of conditions and diagnostic testing methods for poor performance. BAL, Bronchoalveolar lavage; CBC, complete blood count; CSF, cerebrospinal fluid; ECG, electrocardiogram; TW, transtracheal wash.

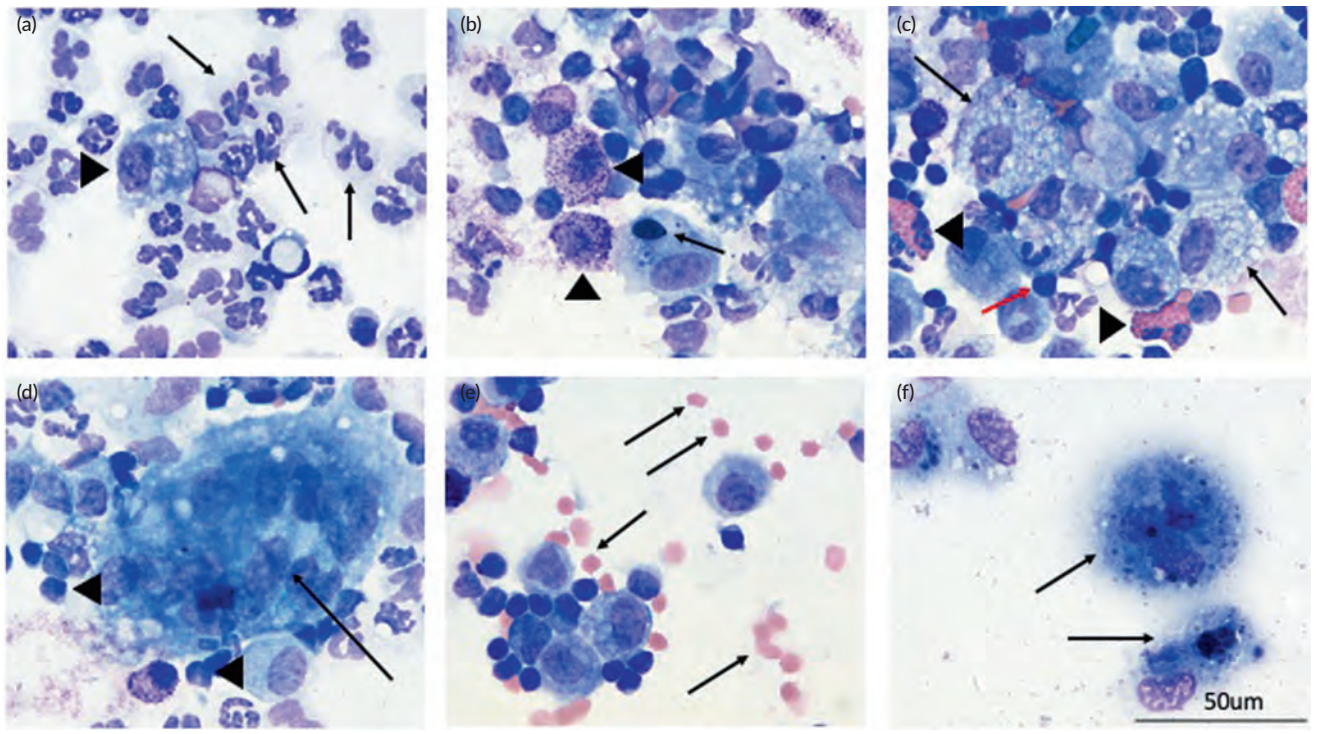

FIGURE 2 Examples of cytology from bronchoalveolar lavage fluid. All images are stained using haematoxylin and eosin at 100x magnification. (a) Neutrophils (black arrows) and macrophages (black arrowhead). (b) Mast cells (black arrowheads) and pollen (black arrow). (c) Macrophages (black arrow), eosinophils (black arrowhead) and lymphocytes (red arrow). (d) Multinucleated giant cell (black arrow). (e) Erythrocytes (black arrows) with macrophages and lymphocytes. (f) Hemosiderin-laden macrophages (black arrow). Images courtesy of Dr Andrea Bohn, Department of Clinical Pathology, Colorado State University.

TABLE 1 Bronchoalveolar lavage and tracheal wash cytologic evaluation in equine asthma

		Neutrophils	Macrophages	Lymphocytes	Eosinophils	Mast cells
Healthy control	BAL[a]	2% (1%–4%)	63% (49%–90%)	32% (6%–47%)	0% (0%–1%)	0% (0%–1%)
	TW[c]	<20%	37.4% (median: 49.3%)	2.0% (median: 3.3%)	0% (median: 0.0%)	
Mild to moderate Equine Asthma	BAL[b]	(5%–20%)	Decreased	Decreased	(>1%)	(>2%)
	TW[c]	(>20%)	-	-	(>1%)	-
Moderate to marked Equine Asthma	BAL[b]	(>25%)	Decreased	Decreased	-	-
	TW[c]	(>70%)	-	-	-	-

[a]Nolen-Walston et al. (2013).
[b]Couëtil et al. (2016).
[c]Rossi et al. (2018).

Recurrent airway obstruction represents moderate to severe equine asthma while IAD represents mild to moderate disease. Horses with equine asthma can progress in disease severity if the condition is not recognised and controlled or treated when mild (Couetil et al., 2020; Couëtil et al., 2016).

Horses with equine asthma syndrome can present with overt signs of respiratory disease including tachypnoea, nasal discharge and cough, but they can also present with subtle signs, including poor performance, chronic cough and prolonged recovery after exercise (Couëtil, 2014; Couetil et al., 2020; Couëtil et al., 2016). Horses with moderate to severe asthma will commonly have an elevated resting respiratory rate, whereas horses with mild asthma have a normal resting respiratory rate. In Thoroughbred racehorses, cough is only present in 38% of horses with mild asthma; however, 85% of coughing horses have mild asthma (Burrell et al., 1996; Christley et al., 2010). Increased mucopurulent exudate in the tracheobronchial tree is detected in the majority of horses with mild asthma; however, this finding is not specific to asthma (Couëtil, 2014).

Diagnosis

Cytology of bronchoalveolar lavage fluid (BALF) or tracheal wash (TW) is necessary to definitively diagnose equine asthma (Figure 2a–d). Horses with mild disease may show one of three (or a combination of the three) profiles on BALF assessment: (1) mild neutrophilia, lymphocytosis and monocytosis; (2) increased mast cells and (3) increased eosinophils. Horses with moderate to severe disease may have a significant increase in neutrophils, combined with a relative decrease in lymphocytes and macrophages (Couëtil, 2014; Couëtil et al., 2016). A recent study has shown that TW samples are also very useful for the identification of airway inflammation and have the advantage of the ability to be performed in unsedated horses with fewer complications compared with BALF samples (Rossi et al., 2018). Reference values for TW and BALF are available in Table 1.

Endoscopic examination of the respiratory tract may be a helpful adjunct diagnostic tool. A mucus scoring system (0–5 scale) has been developed and validated for use in the horse (Table 2) (Gerber et al., 2010). While the presence of tracheal mucus does not provide a definitive diagnosis of equine asthma syndrome, it can be useful to grade severity of asthma, since a positive association has been found between tracheal mucus score, BALF neutrophilia and poor performance (Holcombe, Robinson, et al., 2006; Koblinger et al., 2011).

Plethysmography, a technique that measures changes in lung volume, can also be used for detecting airway reactivity in response to inhaled irritants, such as histamine. Exaggerated airway narrowing in response to an irritant is called airway hyperresponsiveness and is a feature of IAD characterised by an increase in eosinophils and mast cells (Hare & Viel, 1998; Hoffman et al., 1998).

TABLE 2 Endoscopic mucus grading scale for horses with equine asthma[a]

Grade 0: No mucus, clean or a single small blob of mucus
Grade 1: Little, multiple small blobs of mucus
Grade 2: Moderate, larger blobs of mucus
Grade 3: Marked, confluent or stream forming mucus accumulation
Grade 4: Large, pool-forming mucus
Grade 5: Extreme, profuse amounts of mucus

[a]Couëtil et al. (2016).

Exercise-induced pulmonary haemorrhage

Presentation

Exercise-induced pulmonary haemorrhage (EIPH) is a common cause of poor performance, particularly in Thoroughbred racehorses. However, it can affect other breeds/disciplines that work at speed including Quarter Horse barrel and flat racing horses, as well as endurance horses (Gold et al., 2018; Hinchcliff, Jackson, Brown, et al., 2005; Hinchcliff, Jackson, Morley, et al., 2005; Léguillette et al., 2016; Sundman et al., 2020; Tarancón et al., 2019). Racehorses with EIPH of a grade 1 or less (on a scale of 0–4, see Table 3) were 4 times more likely to win, 1.8 times more likely to finish in the top three positions and 3 times more likely to earn above the 90th percentile of earnings than horses with EIPH of grade 2 or greater (Hinchcliff, Jackson, Brown, et al., 2005; Hinchcliff, Jackson, Morley, et al., 2005). While the exact pathogenesis of EIPH is unknown, the likely cause is a rupture of alveolar capillary membranes secondary to an increase in transmural pressure (West & Mathieu-Costello, 1994). Lesions are most commonly seen in the dorsocaudal lung fields (Hinchcliff, 2014).

The most common presenting complaints for horses with EIPH are poor athletic performance and epistaxis. When epistaxis occurs due to EIPH, it is seen during or shortly after strenuous exercise, is usually bilateral, and resolves within hours after exercise is ceased. The occurrence of epistaxis in horses with EIPH is low—one study found epistaxis occurred in only 0.15% of racing starts, while endoscopy has identified EIPH in 14%–75% of racehorses (Takahaski et al., 2001). Other signs of EIPH are non-specific and include coughing and frequent swallowing (Hinchcliff, 2014). The recurrence rate after one episode of epistaxis in Thoroughbred horses in Hong Kong is approximately 13.5% despite affected horses not being permitted to race for one month after the initial episode (Takahaski et al., 2001). This indicates that pulmonary lesions can be slow to heal.

TABLE 3 Endoscopic grading scale for horses with exercise-induced pulmonary haemorrhage[a]

Grade 0: No blood detected in the pharynx, larynx, trachea or mainstem bronchi
Grade 1: Presence of 1 or more flecks of blood or 2 or fewer short narrow streams of blood in the trachea or mainstem bronchi visible from the tracheal bifurcation
Grade 2: 1 long stream of blood or more than 2 short streams of blood occupying less than a third of the tracheal circumference
Grade 3: Multiple, distinct streams of blood covering more than a third of the tracheal circumference, with no blood pooling at the thoracic inlet
Grade 4: Multiple, coalescing streams of blood covering more than 90% of the tracheal surface with blood pooling at the thoracic inlet

[a]Hinchcliff, Jackson, Brown, et al. (2005).

Diagnosis

When epistaxis associated with exercise is not present, the preferred diagnostic test is dependent on timing. Tracheobronchoscopic examination is most appropriate within 1–2 h of exercise, while BALF cytology can be performed days to weeks after strenuous exercise. Observation of blood in the trachea or large bronchi via tracheobronchoscopy after exercise provides a definitive diagnosis of EIPH (Hinchcliff, 2014). A validated grading system has been developed in the horse (Table 3) (Hinchcliff, Jackson, Brown, et al., 2005; Hinchcliff, Jackson, Morley, et al., 2005). The presence of erythrocytes or hemosiderophages in TW or BALF samples is reported to be both sensitive and specific in the diagnosis of EIPH (Figure 2e–f) (Fogarty & Buckley, 1991; McKane et al., 1993). Evidence of pulmonary haemorrhage can last for weeks after a single bleeding episode. Several studies have shown that hemosiderophages are present for at least 21 days after an episode of pulmonary haemorrhage (Fogarty & Buckley, 1991; McKane et al., 1993).

It is not uncommon to find evidence of mild airway inflammation in conjunction with EIPH on BALF cytology; however, it is difficult to determine whether the airway inflammation is a consequence of the EIPH episode, or whether equine asthma might be involved in the pathogenesis of EIPH in these horses. The interaction between these two conditions remains poorly understood.

Upper airway obstructive diseases

Presentation

Upper airway obstructive diseases impede airflow and limit athletic performance by decreasing minute ventilation, exacerbating exercise-induced hypoxaemia and decreasing maximal oxygen consumption (Lane et al., 2006; Strand et al., 2012). Many of these obstructive diseases occur dynamically and can coexist—two or more forms of upper airway obstruction have been reported in 30%–70% of horses (Lane et al., 2006; Strand et al., 2012). Upper airway obstruction typically manifests in one of two clinical presentations—abnormal upper respiratory noise and/or exercise intolerance. Coughing during exercise is also reported with many of the diseases listed above (Ducharme & Cheetham, 2014), as well as frequent swallowing, breath holding and abnormal breathing patterns (Fitzharris et al., 2015; Pigott et al., 2010).

Abnormal upper respiratory noise

Abnormal respiratory noise can be expiratory or inspiratory. Expiratory noise is caused by alar fold collapse, dorsal displacement of the soft palate (DDSP) and palatal instability (Allen, 2015; Allen & Franklin, 2013a, 2013b; Franklin, 2008). Horses with alar fold collapse have an expiratory flutter; a small percentage will have exercise intolerance. This condition is most commonly seen in Standardbreds and Saddlebreds (Franklin, 2008). Intermittent DDSP is common in racehorses and is often described as 'choking down' or 'hitting a wall' by trainers, as the expiratory obstruction limits minute ventilation (Ducharme & Cheetham, 2014). Sport horses with intermittent DDSP often have an expiratory noise during exercise; however, 20%–30% of horses have no noise present (Allen, 2015; Derksen et al., 2001). While poor performance can be seen in sport horses with intermittent DDSP, these horses are more likely to present for making abnormal noise and coughing (Davidson et al., 2011). Horses with upper airway obstruction that perform with the head and neck flexed, such as dressage horses and Saddlebreds, are more likely to suffer poor performance (Barakzai & Hawkes, 2010; Petsche et al., 1995). Palatal instability can precede and progress to DDSP (Allen & Franklin, 2013a, 2013b); however, palatal instability alone can result in exercise intolerance as minute ventilation, tidal volume and oxygen consumption decrease with worsening palatal instability (Allen & Franklin, 2013a, 2013b).

Inspiratory noise can occur with arytenoid cartilage collapse, vocal cord collapse, rostral displacement of the palatopharyngeal arch and nasopharyngeal collapse (Franklin, 2008; Holcombe, Rodriguez, et al., 2006; Taylor et al., 2006). Arytenoid cartilage collapse causes inspiratory noise in addition to poor performance in racehorses and sport horses. Many sport horses are unaffected until 5–6 years of age and have progressively noisier breathing. Exercise intolerance of horses with arytenoid cartilage collapse is associated with decreased ventilation associated with a reduction in airway diameter (Ducharme & Cheetham, 2014). Vocal cord collapse can occur with or without arytenoid collapse (Franklin, 2008; Holcombe, Rodriguez, et al., 2006; Taylor et al., 2006). Vocal cord collapse on its own may not cause exercise intolerance, particularly in horses exercising at submaximal levels (Franklin, 2008). Rostral displacement of the palatopharyngeal arch is associated with congenital malformation of the fourth/sixth branchial arch. The severity of the inspiratory noise and exercise intolerance is related to the severity of the congenital malformation (Ducharme & Cheetham, 2014; Franklin, 2008).

Nasopharyngeal collapse can cause variable degrees of exercise intolerance, dependent on severity, as well as inspiratory noise. Nasopharyngeal collapse can affect the dorsal and/or lateral pharyngeal walls or can be circumferential (Franklin, 2008). Horses with HYPP are at an increased risk for developing nasopharyngeal collapse (Carr et al., 1996). Nasopharyngeal collapse can be induced by sedation administration or application of topical local anaesthetics. In dressage horses, dorsal nasopharyngeal collapse can be induced during rollkur (excessive thoraco-cervical and atlanto-occipital hyperflexion) (Cehak et al., 2010).

Exercise intolerance

Exercise intolerance associated with upper airway obstruction can occur with the conditions listed above as well as epiglottic entrapment, epiglottic retroversion, subepiglottic cysts, medial deviation of the aryepiglottic folds, epiglottitis, arytenoid chondritis, palatal instability or nasopharyngeal collapse (Ducharme & Cheetham, 2014). Clinical signs of arytenoid chondritis during exercise can mimic arytenoid collapse. The degree of exercise intolerance is dependent on the presence and size of granulomas or presence of cartilage deformity (Ducharme & Cheetham, 2014).

Diagnosis

Endoscopy is required to diagnose upper airway obstructive diseases, apart from alar fold collapse, which can be diagnosed by placing a temporary suture to hold the nares open. Several abnormalities can be diagnosed at rest (epiglottic entrapment, subepiglottic cysts, epiglottitis, arytenoid chondritis, rostral displacement of the palatopharyngeal arch and complete arytenoid cartilage collapse); however, exercising (dynamic) endoscopic examination is required for diagnosis of dynamic disorders including nasopharyngeal collapse, intermittent DDSP, epiglottic retroversion, medial deviation of the aryepiglottic folds and arytenoid collapse. Dynamic endoscopy is also recommended if rostral displacement of the palatopharyngeal arch is found during resting endoscopy to assess which other airway abnormalities are associated with the malformation. It should also be kept in mind that horses can have nasopharyngeal collapse during resting endoscopy that is not present during dynamic examination (Ducharme & Cheetham, 2014). Examples of causes of upper airway obstruction on endoscopy are present in Figure 3.

Ultrasound evaluation can be performed for horses with arytenoid collapse, arytenoid chondritis and rostral displacement of the palatopharyngeal arch (Ducharme & Cheetham, 2014). An increase in echogenicity in the cricoarytenoid lateralis muscle in horses with recurrent laryngeal neuropathy has been shown to correlate with arytenoid collapse at exercise with a sensitivity and specificity of 90% and 98%, respectively (Garrett et al., 2011). Ultrasound provides more information about the laryngeal cartilage and surrounding musculature than endoscopy, which may influence treatment recommendations (Garrett et al., 2009, 2011).

CARDIOVASCULAR CAUSES OF POOR PERFORMANCE

Arrhythmias

Atrial fibrillation

Presentation

Atrial fibrillation is the most common cardiovascular cause of poor performance in horses engaging in aerobically challenging

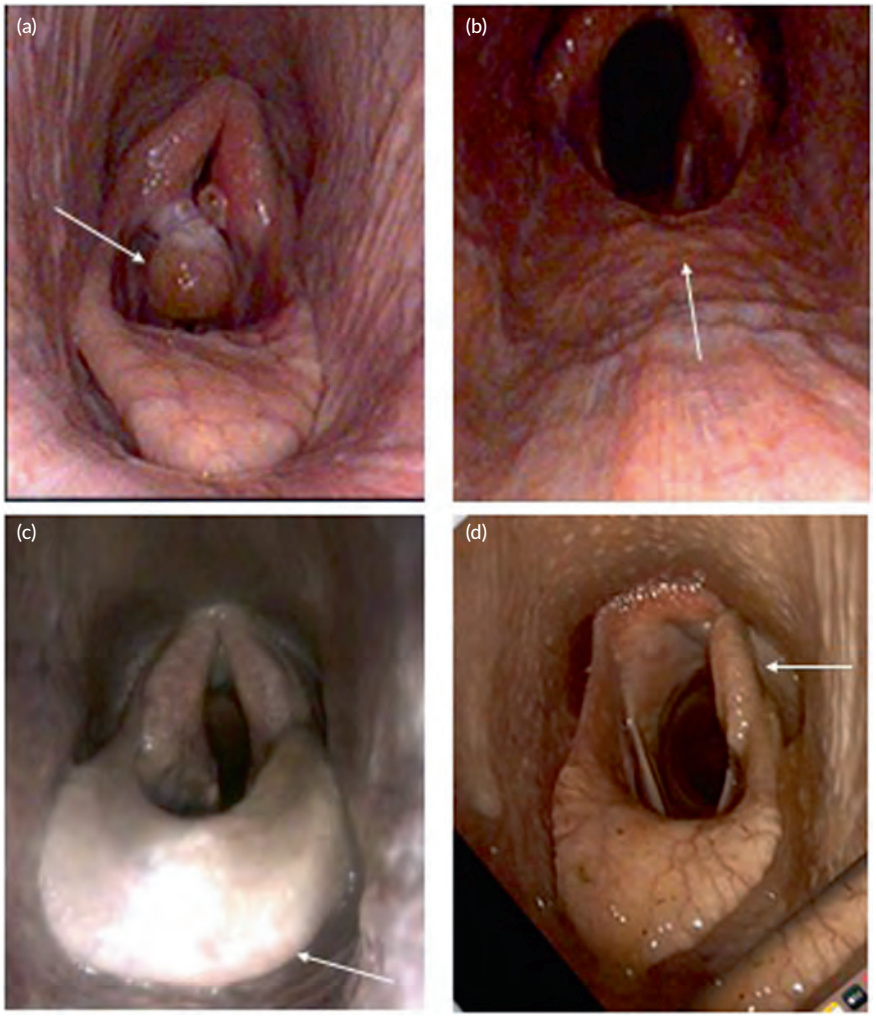

FIGURE 3 Examples of abnormalities detected on upper airway endoscopy. All images are from resting endoscopic examination. (a) Arytenoid granuloma (arrow): A round mass is present at the base of the right arytenoid cartilage that has caused inflammation and a small ulcerative lesion at the base of the left arytenoid cartilage. (b) Dorsal displacement of the soft palate (arrow): The epiglottis is not visible since it is covered by the dorsally displaced soft palate. (c) Epiglottic entrapment (arrow): The tip of the epiglottis is not visible since it is covered by the aryepiglottic membrane. (d) Left arytenoid cartilage collapse (arrow): The right arytenoid cartilage is in full abduction, whereas the left arytenoid cartilage is positioned in the centre of the airway. Note that the image is obtained with the endoscope in the left nostril, which may affect the ability to grade collapse accurately.

sports (racing, eventing and hunting) (Durando, 2019; Young & van Loon, 2014). The irregular heart rhythm precludes maximal atrial filling which, with increasing heart rate during exercise, can result in decreased cardiac output to the body, which in turn can lead to poor performance. Atrial fibrillation can occur in isolation in some horses with no other signs of cardiac disease. This occurs most in large breed horses (Young & van Loon, 2014). Atrial fibrillation can also occur secondary to atrial dilation caused by underlying heart disease—most commonly subsequent to long-standing mitral regurgitation (Reef et al., 1988). Atrial fibrillation can be sustained once initiated, but can also be short-lived, termed paroxysmal atrial fibrillation. Paroxysmal atrial fibrillation can resolve spontaneously following exercise, making it difficult to detect. Atrial fibrillation can also occur secondary to electrolyte and fluid derangements in endurance horses (Young & van Loon, 2014). Horses with atrial fibrillation can present with exercise intolerance or an abrupt decrease in performance. If occurring during peak exercise, the horse may suddenly slow or pull up. Horses often appear distressed and may vocalise. Epistaxis and haemoptysis can also occur (Durando, 2019).

Diagnosis

Diagnosis of atrial fibrillation is made by auscultation of an irregularly irregular rhythm. If atrial fibrillation is secondary to atrial dilation from cardiac disease, there may also be elevated heart rate and cardiac murmur; however, heart rate is often normal at rest if no underlying cardiac disease is present (Young & van Loon, 2014). Electrocardiography will reveal an irregularly irregular R-R interval with a complete absence of P waves, normal QRS morphology and the presence of fibrillation waves (Figure 4a). Electrocardiography

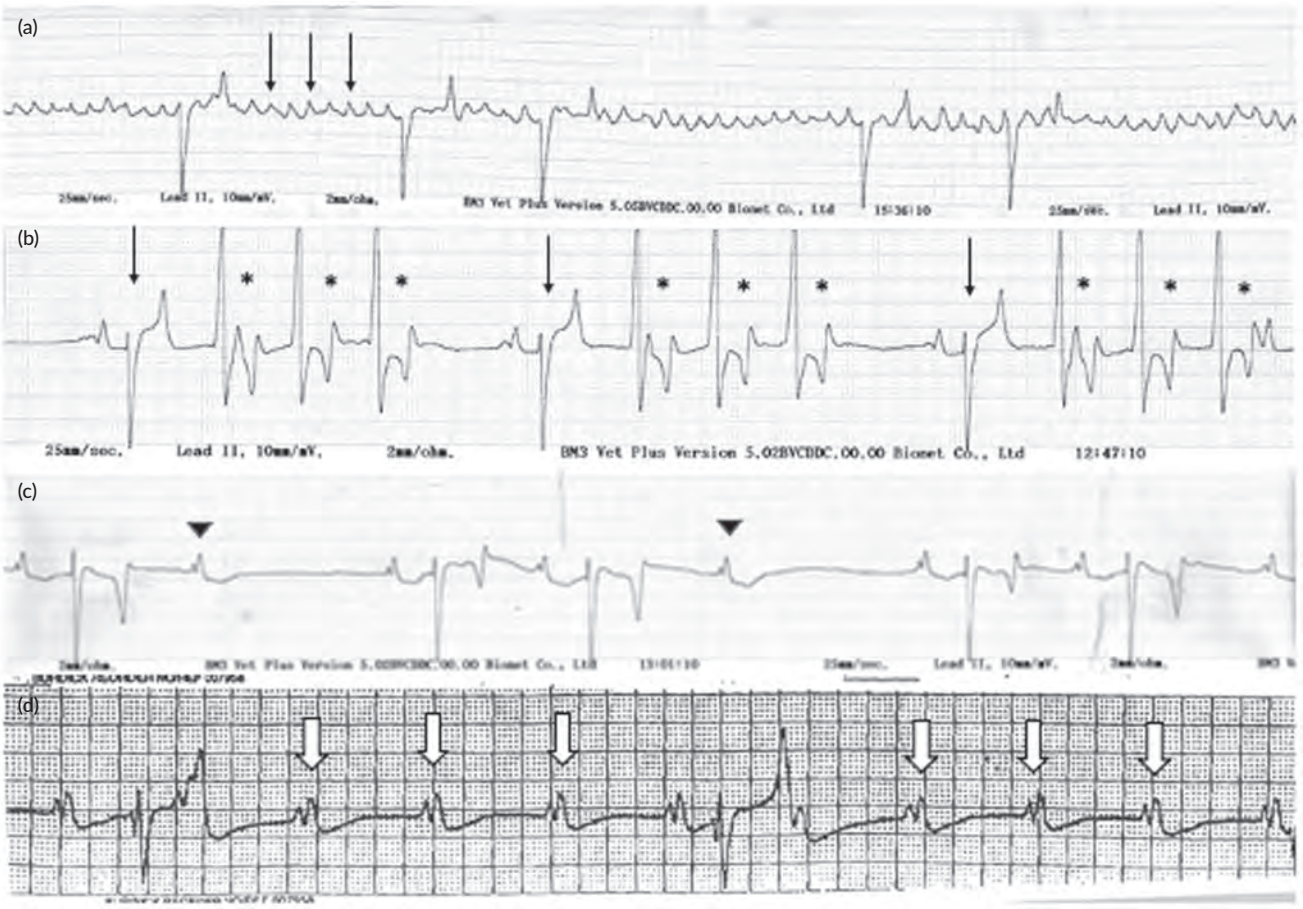

FIGURE 4 Examples of ECG tracings of equine arrhythmias. (a) Atrial fibrillation: Note the lack of P waves and presence of F waves (black arrows). (b) Premature ventricular complexes: Note the presence of normal P-QRS complexes (arrows) followed by runs of three premature ventricular complexes (triplets) (asterisk). (c) Second-degree AV block: Note the presence of P waves (black arrowhead) without an associated QRS complex. (d) Third-degree AV block: Note the presence of multiple P waves in a row (open arrows) with QRS complexes unrelated to a P wave (AV dissociation). Images courtesy of Dr Brian Scansen, Department of Clinical Sciences, Colorado State University.

during exercise is a valuable tool and, in induced atrial fibrillation, has been shown to show abnormal QRS complexes which are a risk factor for collapse or sudden cardiac death (Buhl et al., 2018). Cardiac troponin I levels are rarely elevated in cases of atrial fibrillation if there is no underlying cardiac disease (Young & van Loon, 2014).

Ventricular premature complexes

Presentation

Premature complexes are often detected during auscultation at rest and require electrocardiography to determine whether they are ventricular or supraventricular in origin. Ventricular premature complexes (VPCs) that can be abolished during exercise, or are only detected during the post-exercise period, are unlikely to affect performance (Durando, 2019; Marr et al., 2021). However, if VPCs persist or increase during maximal exercise, they may cause a sudden drop in exercise tolerance. This is a safety concern for the horse and rider, as a more malignant arrhythmia can then occur, such as ventricular tachycardia or ventricular fibrillation, which can result in collapse or sudden death (Durando, 2019; Reef et al., 2014).

Diagnosis

An exercising ECG must be performed to determine whether VPCs detected at rest resolve or persist with exercise (Figure 4b) (Durando, 2019; Reef et al., 2014). In addition, thorough upper airway examination should be performed, as exercise-associated upper airway obstructions may increase the risk of rhythm disturbances developing (Marr et al., 2021).

Third-degree AV block

Presentation

Unlike second-degree AV block (Figure 4c), third-degree AV block (Figure 4d) is rare in horses, but when present, will affect performance. Second-degree AV block disappears during exercise and thus is not a cause of exercise intolerance (Young

& van Loon, 2014). Third-degree AV block is the most common form of atrioventricular dissociation, in which the impulse generated in the sinoatrial node is not propagated to the ventricles (Ertelt et al., 2018). Typical presenting signs of third-degree AV block include a history of syncope, ataxia, exercise intolerance and collapse.

Diagnosis

In cases of third-degree AV block, an inappropriately slow heart rate will be auscultated. Resting heart rate can be less than 20 beats per minute. In third-degree AV block, S4 can be audible in a regular fast rhythm underlying the predominant slow ventricular rhythm. Pronounced waves will also be present that travel all the way up the jugular vein (Young & van Loon, 2014). Electrocardiography will provide a definitive diagnosis (Figure 4d). P waves generally occur at an increased rate but have no relation to the slower ventricular rate (Young & van Loon, 2014).

Valve disease

Presentation

A number of cardiac murmurs unassociated with structural cardiac disease occur in horses—functional, physiological, innocent, flow murmurs—and are common in athletic horses (Young & van Loon, 2014). It is important to differentiate between murmurs that are of no clinical significance and those that are or could become of clinical significance using echocardiography.

If compensated, aortic, mitral and tricuspid valve regurgitation have been shown to have no negative associations with performance in flat or jump racehorses in the UK (Kriz et al., 2000; Young et al., 2008). However, aortic insufficiency, which occurs commonly in older horses, can occasionally be associated with ataxia during exercise (Young & van Loon, 2014). Horses with aortic insufficiency will have a diastolic murmur with point of maximal intensity over the left heart base that is usually decrescendo (Jago & Keen, 2017; Young & van Loon, 2014). Because of the musical quality of this murmur, prediction of severity of cardiac disease based on murmur intensity is difficult and further evaluation is warranted in the majority of cases due to the possibility of sudden cardiac death (Jago & Keen, 2017).

Poor performance can also occur with severe mitral valve regurgitation, a systolic murmur heard on the left side with the point of maximal intensity over the apex of the heart (Jago & Keen, 2017); however, if severe enough to affect performance, other signs of heart failure are usually present (Young & van Loon, 2014). In general, mitral regurgitation heard on auscultation should be investigated further if a high-grade murmur is present (grade 3/6 or greater) (Jago & Keen, 2017).

Tricuspid regurgitation is the most common murmur identified in performance horses but is rarely of clinical significance (Jago & Keen, 2017). This murmur is a systolic murmur heard on the right side of the chest with point of maximal intensity over the apex of the heart. Further evaluation is recommended in horses with a murmur grade 4/6 or higher (Jago & Keen, 2017).

Diagnosis

The cause of the murmur may be determined using echocardiography. For horses with aortic insufficiency, echocardiography will also allow evaluation of the presence of secondary mitral regurgitation. When both conditions are present, prognosis is much more guarded. Electrocardiogram is also warranted to ensure that secondary atrial fibrillation is not present (Young & van Loon, 2014). Horses should have repeat echocardiograms performed every 6–12 months, depending on the severity of the disease, to determine whether/how quickly structural changes are progressing (Reef et al., 2014).

Aortoiliacofemoral thrombosis

Presentation

Horses with aortoiliacofemoral thrombosis can initially present with increasingly poor performance before the onset of more dramatic signs (Young & van Loon, 2014). Clinical signs vary from mild hindlimb stiffness that can reduce performance to more severe unilateral or bilateral hindlimb lameness induced by strenuous exercise. The affected limb can feel cooler after exercise (Young & van Loon, 2014).

Diagnosis

Aortoiliacofemoral thrombosis can be diagnosed by careful rectal palpation (Young & van Loon, 2014). However, transrectal ultrasonography is the most practical method to make a definitive diagnosis (Reef et al., 1987).

Thermoregulatory cause of poor performance: Anhidrosis

Presentation

Anhidrosis is characterised by decreased or absence of sweating. The disease most commonly occurs in hot, humid environments, particularly in the summer months (Hubert & Beadle, 1998). Anhidrosis is thought to occur due to prolonged stimulation of the sweat glands by epinephrine that is secreted in response to environmental conditions. The prolonged stimulation results in desensitisation of the sweat glands to the epinephrine. The water channel aquaporin-5 may also play a role with strong activity present in the glandular secretory cells and little activity found in horses with anhidrosis (Johnson, Burton, & Sweeney, 2010; Johnson, Mackay, & Hernandez, 2010). An epidemiologic study of 4620 horses in Florida showed a prevalence of 2%. Thoroughbreds and Warmbloods were predisposed, as well as horses that were foaled in the Western or Midwestern United States. There also appears to be a familial relationship with the odds of a horse having anhidrosis being 6.87 times

higher if there was a familial history of the disease (Johnson, Burton, & Sweeney, 2010; Johnson, Mackay, & Hernandez, 2010).

Decreased or lack of sweating may be missed by the rider or trainer. Horses often present with a complaint of poor performance, exercise intolerance or laboured breathing after exercise. Marked tachypnoea may be present (60–120 breaths per minutes) and is the most common clinical sign (Hubert & Beadle, 1998; Johnson, Burton, & Sweeney, 2010; Johnson, Mackay, & Hernandez, 2010). Tachypnoea occurs as the horse tries to dissipate heat. Elevated rectal temperature will also be present after exercise. Horses with long-standing anhidrosis may also present with dermatologic changes, including a dry, sparse hair coat, scaling and alopecia, particularly of the face, neck and shoulders (Hubert & Beadle, 1998). Horses with partial anhidrosis may continue to sweat under the mane, saddle, halter, axillary, inguinal and perineal regions, as these areas have the highest density of sweat glands (McCutcheon & Geor, 2014).

Diagnosis

A definitive diagnosis is obtained by intradermal skin testing using a beta-2-agonist, such as terbutaline or salbutamol. Serial dilutions of the beta-2-agonist, as well as a saline control, are injected intradermally in the neck region. A normal horse will sweat after injection of all dilutions. Horses with partial anhidrosis will only sweat with the highest concentration, while horses with severe anhidrosis may not sweat at all (McCutcheon & Geor, 2014).

Muscular causes of poor performance

Muscle disease can result in pain, usually associated with exercise, or weakness, sometimes associated with muscle atrophy. In horses with muscle atrophy, it is necessary to determine whether there is an orthopaedic, neurogenic or myogenic cause of the atrophy. Conditions that can lead to muscle atrophy include diseases such as equine protozoal myelitis, equine motor neuron disease, vitamin E-dependent myopathy, immune-mediated myositis, polysaccharide storage myopathy type 1 (PSSM-1), trauma, nerve impingement and polyneuropathy. Furthermore, secondary muscle atrophy can occur through insufficient nutrient intake, chronic disease and endocrinological disease. Some of these conditions are further discussed below.

Muscle pain is usually associated with exercise and diseases that lead to muscle pain can either result in muscle breakdown (rhabdomyolysis) or not. Sporadic exertional rhabdomyolysis (ER) is a condition in which a horse develops ER on a single occasion due to extrinsic causes, whereas chronic ER and chronic exertional myopathies are thought to result from intrinsic abnormalities in muscle structure and function (Valberg, 2018). As the name suggests, sporadic ER typically is only seen intermittently in a given horse and occurs secondary to extrinsic factors. These factors can include dietary imbalances, intense exercise or an increase in training to which the horse has not been conditioned, exercise in excessive environmental conditions and exercise with concurrent viral disease. Chronic ER and chronic myopathies, on the other hand, result in repeated episodes of ER, muscle pain or poor performance in a given horse. For many disorders that are characterised by abnormalities in muscle structure and function, underlying genetic causes have been identified. Inherited muscle disorders can also occur sporadically and may not be related to changes in exercise routine (Piercy & Rivero, 2014).

Clinical signs of muscle pain can range from presence of reluctance to move, mild stiffness, a stilted gait to severe stiffness, sweating or recumbency (Valberg, 2018). Pain can also manifest in other ways. For example, horses may posture to urinate frequently, while others may display signs of colic. In severe cases of muscle damage, pigmenturia can occur secondary to myoglobin breakdown. Some horses with mild signs will appear clinically normal on examination but will have a history of poor performance or gait changes (Piercy & Rivero, 2014).

With rhabdomyolysis, elevations in creatine kinase (CK) and aspartate aminotransferase (AST) are present on routine blood profile. Plasma CK is the most specific marker of acute muscle damage, peaking 4–6 h following muscle damage. It then declines, with a half-life of approximately 12 h (Toutain et al., 1995). Peaks of AST occur approximately 24 h after an episode and can remain elevated for several days to weeks (Piercy & Rivero, 2014). Therefore, the timing of the most recent episode should be taken into consideration when interpreting bloodwork.

An exercise test, in which blood CK concentrations are evaluated before and 4–6 h after the horse undergoes 15 min of light or moderate exercise, can be helpful in confirming an underlying muscle disorder. A positive test, defined as a 2–3 fold increase in CK activity over the baseline value, can indicate subclinical ER (Valberg, 2018).

Genetic testing can be performed from blood or hair root samples to detect inherited forms of exertional rhabdomyolysis (PSSM, Malignant Hyperthermia) (Piercy & Rivero, 2014). A definitive diagnosis for inherited forms of exertional rhabdomyolysis is performed by muscle biopsy (Piercy & Rivero, 2014). More details of individual muscle diseases are listed below.

Evaluation of routine blood work, serum vitamin E concentrations, electrodiagnostics (electromyography, nerve conduction velocities), genetic testing, and muscle biopsies can aid in determining a cause for muscle atrophy.

Inherited myopathies

Recurrent exertional rhabdomyolysis (RER)

Recurrent exertional rhabdomyolysis is an inherited disorder of Thoroughbreds that are affected intermittently with episodes of exertional rhabdomyolysis, often without a clear inciting cause (Piercy & Rivero, 2014; Valberg, 2018). Between 4.9% and 6.7% of racing Thoroughbreds can be affected by RER during a single race season with fillies being more frequently affected (MacLeay et al., 1999; McGowan et al., 2002).

Malignant hyperthermia (MH)

Malignant hyperthermia has been found in Quarter Horses and Paint horses with a mutation in the RYR1 gene. This autosomal dominant mutation has been reported in halter Quarter Horses with histories of sudden death, rhabdomyolysis, and persistent increases in muscle enzyme activity. Genetic testing can be performed from whole blood or hair root plucks (Piercy & Rivero, 2014).

Immune-mediated myositis

Immune-mediated myositis is a disorder affecting Quarter Horse-related breeds, caused by a genetic mutation in MYH1 (Valberg, 2018). Incidence is highest in working cow and reining horses (Gianino et al., 2019) and can frequently be seen 3–4 weeks following Streptococcal infection, vaccination for Strangles or Flu/Rhino, or following administration of immune stimulants (Finno et al., 2018). Horses with immune-mediated myositis will have symmetric, rapid muscle atrophy, which initially affects the topline muscles. Serum CK and AST levels can be markedly elevated early in the disease process. Biopsy of the gluteal or epaxial musculature is also diagnostic in the early stages of atrophy (Valberg, 2018). Genetic testing is available using hair samples.

Polysaccharide storage myopathy type 1 (PSSM 1)

Polysaccharide storage myopathy type 1 is most prevalent in Quarter Horses, particularly halter Quarter Horses, and draught breeds (Firshman et al., 2008; Valberg et al., 1996). A single missense mutation in the skeletal muscle glycogen synthase gene (GYS1) has been identified in PSSM-affected Quarter Horses (McCue et al., 2008; Valberg, 2018). Muscle of affected horses has either excessive amylase-sensitive glycogen or the presence of amylase-resistant polysaccharide (Firshman et al., 2006). This material accumulates as the animal ages (Valentine & Cooper, 2006). Because of the excessive glycogen in muscle, horses have a deficit in energy when exercised and persistent elevations in serum CK activity. Homozygote PSSM 1 horses are more severely affected than heterozygotes. Muscle biopsy of the semimembranosus can be performed for a definitive diagnosis if genetic testing (blood, hair) is not conclusive (Valberg, 2018).

Other myopathies

Polysaccharide storage myopathy type 2 (PSSM 2)

Polysaccharide storage myopathy type 2 occurs more commonly in Warmbloods and in Quarter Horses, particularly those used for barrel racing, reining, and cutting (Valberg, 2018). These horses do not carry the GYS1 mutation associated with PSSM 1 but have similar glycogen accumulation within the muscle (McCue et al., 2008). Gait abnormalities are more commonly seen in Warmbloods with PSSM2 whereas rhabdomyolysis is much more common in Warmbloods with PSSM1 (Lewis et al., 2017). In Warmbloods, clinical signs may include vague lameness, reluctance to collect or go forward under saddle, or a slow onset of topline atrophy (Valberg, 2018). Unlike in Quarter Horses, elevated CK and AST are rarely present in Warmbloods with PSSM2. A scientifically validated genetic test for PSSM 2 does not exist, therefore, muscle biopsy of the semimembranosus must be performed for diagnosis (Valberg, 2018).

Myofibrillar myopathy (MFM)

Myofibrillar myopathy has been identified relatively recently and has been identified predominantly in Arabians and Warmbloods (McKenzie et al., 2016; Valberg et al., 2017). This disorder involves disruption of the orderly alignment of the myofibrils (contractile proteins). There is currently no scientifically validated genetic test for MFM and therefore, muscle biopsy of the semimembranosus is needed for a definitive diagnosis. False negative tests can occur in young horses with MFM—desmin staining is used for identification of the disease which may be less obvious in horses <8 years of age (Valberg, 2018). Clinical signs in Arabians are similar to other causes of exertional rhabdomyolysis (Valberg et al., 2016). However, in Warmbloods, a decline in performance due to MFM may not occur until the animal is 8–10 years of age. Specific signs in Warmbloods include a change in energy level, reluctance to collect, unwillingness to go forward, abnormal canter transitions, or poor quality gaits (Valberg, 2018). Sore muscles and a vague, poorly localised hindlimb lameness can also be seen with MFM (Valberg, 2018; Valberg et al., 2017).

Vitamin E deficiency myopathy

Vitamin E deficiency can result in equine motor neuron disease (EMND) (see more details below); however, some horses may develop a myopathy. Horses will have generalised muscle atrophy and weakness that may be similar to, or more subtle than, horses with EMND (Valberg, 2018). Diagnosis is made by muscle biopsy of the sacrocaudalis dorsalis muscle, which can differentiate the two diseases. Serum vitamin E levels may be normal in affected horses. Horses with vitamin E deficiency myopathy tend to be more responsive to vitamin E supplementation than horses with EMND (Valberg, 2018).

Neurological causes of poor performance

Although severe neurological disease will result in more significant impairment of gait, if disease is mild, subtle gait deficits might go unnoticed and animals may present for poor performance. Examples of specific clinical signs that should lead to further evaluation of the

neurological system include stumbling, falling, intermittent thoracic limb lameness, gait asymmetry or irregularities, difficulties with transitions, and difficulty initiating or maintaining gaits (e.g. backing up or canter on a small circle). Neurological disorders that have been shown to result in these gait deficits include cervical vertebral compressive myopathy (CVCM), equine degenerative myeloencephalopathy (EDM), equine protozoal myeloencephalitis (EPM), EMND, and other neuropathies.

Cervical vertebral compressive myelopathy

Presentation

Cervical vertebral compressive myelopathy is a disorder that is caused by narrowing of the cervical vertebral canal with subsequent compression of the spinal cord and resulting symmetrical ataxia, dysmetria, and weakness affecting all four limbs (Carr & Maher, 2014; Nout & Reed, 2003). Although CVCM is most commonly recognised in young horses associated with developmental orthopaedic disease (CVCM Type I), it is also frequently diagnosed in older horses. In this older group of horses, diagnosis is often delayed because of clinical signs being subtle, or onset of clinical signs occurs later in life associated with osteoarthritis (CVCM Type II). The underlying aetiology of CVCM is considered multifactorial with factors such as genetics, nutrition, biomechanics of the axial spine, and trauma playing important roles. Young, large, rapidly growing, male Thoroughbred and Warmbloods are overrepresented in the prevalence of CVCM (Carr & Maher, 2014; Levine et al., 2010; Nout & Reed, 2003). With CVCM Type II, enlargement of the articular process joints associated with osteoarthritis in the caudal cervical vertebrae result in compression of the spinal cord (Nout & Reed, 2003).

Clinical signs of CVCM include symmetric ataxia, dysmetria, and weakness. Pelvic limbs are often more severely affected than the thoracic limbs which is likely related to the more lateral location of the ascending proprioceptive tracts of the pelvic limbs within the spinal cord and the length of the descending motor tracts to the pelvic limbs. Specific abnormalities seen during a neurological examination include an irregularly irregular uncoordinated gait, hyper- or hypo-metric foot flight, circumduction of pelvic limbs on circles, knuckling, limb interference, dragging of the toes with excessive hoof wear, a strong standing tail pull, and weakness on a dynamic tail pull. Mentation and cranial nerves are generally normal unless secondary trauma has occurred due to a fall (Carr & Maher, 2014; Nout & Reed, 2003).

Diagnosis

Screening lateral radiographs of the cervical spine are recommended and can show malformation of vertebrae, malalignment of adjacent vertebrae, flaring of the caudal vertebral epiphysis of the vertebral body, extension of the caudal edge of the dorsal arch of the vertebral body, and enlargement of the articular processes (Carr & Maher, 2014). From lateral radiographs, indications of narrowing of the vertebral canal can be obtained by several methods. For the intravertebral minimal sagittal diameter ratio (MSDR) the width of the spinal canal at the cranial aspect of the vertebral body is compared to the maximum width of the cranial vertebral body (Figure 5a), while for intervertebral MSDR the width of the spinal canal is determined between two adjacent vertebral bodies compared to the maximum width of the cranial vertebral body. Multiple studies have evaluated the accuracy of these methods in predicting the presence of CVCM. Rush-Moore et al. (1994) evaluated intravertebral MSDRs for predicting CVCM and found that a MSDR cut-off of 0.52 for C4, C5 and C6, and 0.56 for C7 had the highest sensitivity and specificity, and by using these CVCM was in fact diagnosed by myelography in 89% of cases. A later study showed that by using a cut-off of 0.485 for intra- or intervertebral MSDRs, CVCM was correctly predicted in all cases (Hahn et al., 2008). Importantly, these methods do not allow for prediction of what sites of the spinal cord are compressed (Hahn et al., 2008; Janes et al., 2014; Moore et al., 1994). Although lateral standing cervical radiographs can be used to accurately diagnose

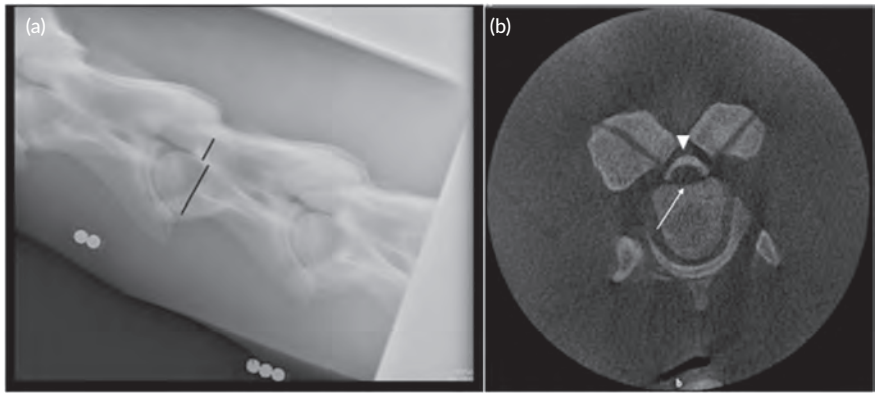

FIGURE 5 Examples of diagnostic imaging of two cases of cervical vertebral compressive myelopathy. (a) Lateral radiograph of the cervical spine showing the caudal aspect of C2, C3, C4 and cranial aspect of C5. Narrowing of the vertebral canal is evident at the cranial aspect of C4 based on an intravertebral sagittal ratio measurement <50%. Black lines at C4 show the location for the measurement of the intravertebral sagittal ratio. (b) Transverse CT myelogram image of the cervical spine at C5-C6. Note the dural space present dorsally, but not ventrally (arrowhead) and compression and flattening of the spinal cord (arrow).

CVCM in horses, relying on measurements can result in false positive and false negative diagnoses. Additionally, significant inter- and intraobserver variability in measurement of MSDRs has been reported (Scrivani et al., 2011), further highlighting the fact that making a definitive diagnosis of CVCM based on radiographs should be done with caution.

Myelography remains the gold standard for diagnosis of CVCM and is necessary to accurately identify the site(s) of compression. The most common criteria to diagnose CVCM on myelogram is a >50% height reduction of the minimal dorsal myelographic column (DMC) at the intervertebral site compared to the maximal diameter of the DMC at the level of the cranial vertebral body (Carr & Maher, 2014). One study suggested that the compression criteria should vary with the vertebral site and neck position—a 50% reduction in the DMC is adequate when evaluating the neutral views at C3-C6, but a more conservative approach should be used in the flexed views at these sites. In addition, a cut-off of 50% can be used for a flexed view at C6-C7, but 70% should be used for the neutral view at this location (Biervliet et al., 2010). Alternatively, reduction of the dural diameter can be quantified and used to determine site of compression. More recently, CT myelography is used with increasing frequency to provide more detail of the cervical spinal column in three dimensions (Figure 5b), however, acquiring dynamic views using this technique is challenging.

Equine protozoal myeloencephalitis

Presentation

Equine protozoal myeloencephalitis is caused by *Sarcocystis neurona* or *Neospora hughesi* infection and occurs when horses ingest contaminated feed/pasture. The opossum is the definitive host of *S. neurona*; intermediate hosts include cats, skunks, armadillos, and raccoons. All horses can be infected with EPM however, Thoroughbreds, Quarter Horses, Standardbreds, and young horses are overrepresented in epidemiological surveys (Carr & Maher, 2014; Reed et al., 2016). Equine protozoal myeloencephalitis is a North American disease, however, is occasionally seen in other parts of the world when horses with EPM are imported.

Clinical signs of EPM can be highly variable, depending on the location of organism in the central nervous system. However, asymmetric gait deficits with focal muscle atrophy and/or cranial nerve deficits are the most common clinical signs (Carr & Maher, 2014).

Diagnosis

Definitive diagnoses of EPM requires postmortem confirmation of protozoal infection of the central nervous system. For highest accuracy in antemortem diagnosis, the diagnosis of EPM should be based on (1) presence of clinical signs consistent with EPM; (2) exclusion of differential diagnoses; (3) immunodiagnostic testing of serum and cerebrospinal fluid (CSF) to confirm intrathecal antibody production against the causative organisms *S. neurona* or *N. hughesi* (Reed et al., 2016). Seroprevalence of EPM has been reported to range from 50% to 60% in the USA, indicating that most horses are exposed to *S. neurona* at some stage. Therefore, CSF and serum titers should be evaluated as a ratio and should not be evaluated in isolation (Carr & Maher, 2014; Reed et al., 2016).

Multiple tests have been developed to identify active EPM infection. Early tests included a Western Blot and a modified Western Blot to detect *S. neurona* of which the sensitivity and specificity was reported as 89%–100% and 69%–98%, respectively (Duarte et al., 2003; Rossano et al., 2000). An enzyme linked immunosorbent assay (ELISA) was developed against the SAG-1 protein of *S. neurona* however, the sensitivity and specificity were shown to be poor (Johnson, Burton, & Sweeney, 2010; Johnson, Mackay, & Hernandez, 2010). Subsequently, an ELISA against the *S. neurona* SAG-2 protein was developed and was shown to have a high sensitivity (93.2%) and specificity (95.9%) (Reed et al., 2013). The currently recommended tests for the diagnosis of EPM are the SnSAG2, 4/3 ELISA serum:CSF titre ratio and the NhSAG1 ELISA serum:CSF titre ratio, which provide information regarding intrathecal antibody production based on serum and CSF titers (Reed et al., 2013; Reed et al., 2016 – consensus statement JVIM).

Equine degenerative myeloencephalopathy

Presentation

Equine degenerative myeloencephalopathy is a severe, diffuse form of neuroaxonal dystrophy. It has been recognised in most breeds however, there are reports of familiar disease in Appaloosas, Morgans, Standardbreds, and Quarter Horses. This condition most commonly becomes apparent in the first year of life and is associated with vitamin E deficiency and rapid decline (Carr & Maher, 2014; Dill et al., 1990). However, more recently the condition has been reported in horses in work, typically between ages 5 and 15 years, with Warmbloods overrepresented in this group (Johnson, 2021).

Clinical signs are typically present within the first year of life and include symmetric ataxia, proprioceptive deficits, and weakness. Forelimbs can be equally or less severely affected compared to the hindlimbs. Profound hypometria is often present, as well as hyporeflexia of the cutaneous trunci (Burns & Finno, 2018; Carr & Maher, 2014; Finno & Valberg, 2012). Horses that survive to 2–3 years of age can exhibit lifelong, stable neurological deficits (Finno & Valberg, 2012).

For the older subset of horses, behavioural changes under saddle, including unpredictable and severe spooking, bucking, bolting, rearing, or spinning are reported (Johnson, 2021). A change in personality of the horse is common, including increasingly dull and lethargic demeanour that may be interspersed with anxious and aggressive behaviour. Ataxia may or may not initially be apparent; topline muscle atrophy and a dull haircoat may also be seen (Johnson, 2021).

Diagnosis

While vitamin E deficiency plays a role in the development of EDM in young horses, dietary and serum levels of vitamin E are not always

abnormal in affected horses. Serum vitamin E levels are most useful as a screening test for inadequate dietary intake and are not definitive evidence of EDM (Carr & Maher, 2014). No definitive antemortem test exists for EDM. For both younger and older age groups, a complete neurological examination and diagnostics including cervical radiographs and CSF tap should be performed to rule out other causes of the clinical signs. Onset of signs at a young age and low serum vitamin E levels are suggestive of EDM (Carr & Maher, 2014; Finno & Valberg, 2012). More recently biomarkers such as phosphorylated neurofilament heavy (pNF-H) are being investigated as antemortem tests for EDM, however, to date sensitivity remains low (Intan-Shameha et al., 2017; Johnson, 2021).

Equine motor neuron disease

Presentation

Equine motor neuron disease is a diffuse neurodegenerative disorder affecting the somatic, lower motor neurons. Quarter Horses are overrepresented with an increased risk with increasing age. The risk of EMND peaks at 16 years of age and then declines (Carr & Maher, 2014). The exact aetiology of EMND is unknown, though a deficiency in dietary vitamin E is thought to play a role—risk factors include housing in dirt paddocks and lack of access to green pasture (de la Rúa-Domènech et al., 1997; Divers et al., 1994).

Clinical signs include muscle fasciculations, weight shifting and a base narrow stance. Severe muscle atrophy occurs, particularly the antigravity muscles including the triceps, gluteals and quadriceps (Carr & Maher, 2014). Clinical signs are due to loss of somatic motor neurons; type 1 fibres are more severely affected. Secondary lesions include peripheral nerve degeneration and severe neurogenic muscle atrophy (Carr & Maher, 2014; Finno & Valberg, 2012).

Diagnosis

A presumptive diagnosis can be made on clinical signs. Mild to moderate elevations in CK and AST may be present in acute cases. For antemortem diagnosis, two diagnostics can be performed: (1) biopsy of the sacrocaudalis dorsalis muscle or (2) biopsy of the spinal accessory nerve (Carr & Maher, 2014; Finno & Valberg, 2012).

Trigeminal-mediated headshaking

Presentation

Trigeminal-mediated headshaking (formerly known as idiopathic headshaking) is characterised by abrupt motion of the head and neck, typically in the vertical direction, with sharp ventral flicks (Pickles et al., 2014; Roberts, 2019). Signs of nasal irritation are also frequently present, including snorting, anxious facial expression and muzzle rubbing (Pickles et al., 2014; Roberts, 2019). Clinical signs can be mild to severe and can result in self-trauma (Pickles et al., 2014). The condition is typically seen during exercise but can occur at rest in more severely affected horses. A seasonal component is present in 60% of cases with increased clinical signs during the Spring and Summer (Pickles et al., 2014; Roberts, 2019). The condition is typically seen in mature, adult horses (median age of onset 6–10 years) with geldings overrepresented (Pickles et al., 2014; Roberts, 2019). No breed predisposition has been identified (Pickles et al., 2014; Roberts, 2019).

The condition has a prevalence of 4.6%–6.2% in the UK (Ross et al., 2018) and has been demonstrated to be due to sensitisation of the infraorbital branch of the trigeminal nerve, resulting in a lower threshold for activation (Aleman et al., 2013, 2014). It is unclear why this sensitisation occurs. While demyelination of the nerve has been seen in humans with a similar condition, this demyelination has not been demonstrated in the horse suggesting a functional rather than structural change to the nerve (Roberts et al., 2017). In addition, in contrast to humans who are typically unilaterally affected, horses are typically affected bilaterally (Aleman et al., 2013, 2014; Roberts, 2019; Roberts et al., 2017).

Diagnosis

Diagnosis of trigeminal-mediated headshaking is usually made by exclusion of other causes of headshaking (dental issues, sinusitis, otitis interna, guttural pouch mycosis and EPM) (Roberts, 2019). Since confirmation of the involvement of the trigeminal nerve, effects of local analgesia have been explored. Bilateral local analgesia of the rostral portion of the infraorbital nerve is not useful for diagnosis (Mair, 1999; Newton et al., 2000), however, bilateral local analgesia of the posterior ethmoidal nerve resolved headshaking clinical signs in 85% of horses (Roberts et al., 2013).

Gastrointestinal cause of poor performance

Equine gastric ulcer syndrome

Presentation

Equine gastric ulcer syndrome (EGUS) can be a cause of poor performance in equine athletes (Franklin et al., 2008; Sykes et al., 2015, 2019). It can be further categorised into equine squamous gastric disease (ESGD) and equine glandular gastric disease (EGGD) based on the anatomic region affected (Sykes et al., 2015). Thoroughbred racehorses diagnosed with EGGD have been found to perform below expectation (Sykes et al., 2019). In addition, Thoroughbred racehorses with ESGD have been shown to have decreased performance associated with the presence of gastric ulcers, independent of severity or number of ulcers (Sykes et al., 2015). Equine gastric ulcer syndrome is common in equine athletes with a prevalence of 17%–58% in sport horses and 80%–100% in racehorses in training (Sykes et al., 2015).

Common signs of glandular or squamous gastric disease include episodes of colic, which can be recurrent, bruxism, weight loss and reduced appetites (Sykes et al., 2015). Girthiness can also be a sign of gastric ulceration; in one study of horses that showed girthiness while being tacked, gastric ulceration was the most common

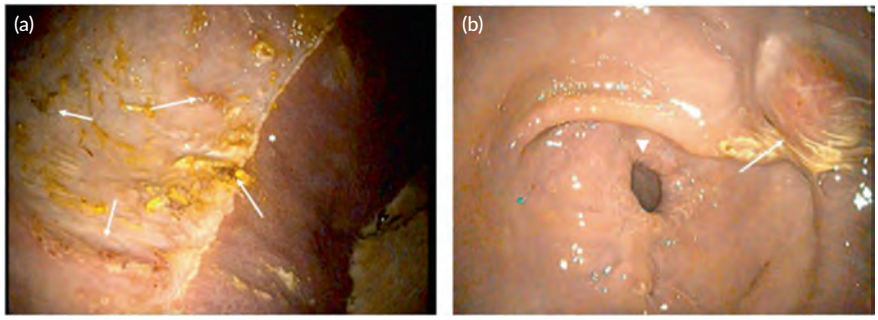

FIGURE 6 Examples of gastroscopy images from horses with gastric ulceration. (a) Grade 4 equine squamous gastric disease with coalescing erosions (arrows) in a striated pattern extending from the margo plicatus (asterisk) with associated hyperkeratosis. (b) Equine glandular gastric disease with raised and haemorrhagic ulcers (arrow) present near the pyloric opening (arrowhead).

TABLE 4 Grading scale for equine squamous gastric disease[a]

Grade 0: The epithelium is intact and there is no appearance of hyperkeratosis
Grade 1: The mucosa is intact, but there are areas of hyperkeratosis
Grade 2: Small, single or multifocal lesions
Grade 3: Large single or extensive superficial lesions
Grade 4: Extensive lesions with areas of apparent deep ulceration

[a]Sykes et al. (2015).

finding (Millares-Ramirez & Le Jeune, 2019). In addition, changes in behaviour, including nervousness, aggression and self-mutilation have been reported in association with gastric ulceration (Sykes et al., 2015). While EGUS can be the cause for poor performance in some horses, it is also possible that there is another factor in play that is both causing EGUS and poor performance (e.g. axial spine pain). Therefore, it is important to determine the likely underlying cause for EGUS when the condition is diagnosed.

Diagnosis

Definitive diagnosis is made by gastroscopy. Complete evaluation of the pylorus and proximal duodenum is necessary for diagnosis of glandular gastric disease (Figure 6) (Sykes et al., 2015). There is no relationship between presence of ESGD and EGGD; therefore, the presence or absence of one is not predictive of the presence or absence of the other (Sykes et al., 2015). Diagnosing EGUS should be followed by determining the cause of EGUS in that particular animal. A grading scale has been established for ESGD (Table 4). Current grading recommendations for EGGD include descriptors of presence/absence, anatomical location, distribution and appearance of lesions (Sykes et al., 2015).

CONCLUSIONS

Diagnosis of poor performance and gait deficits in the equine athlete can be challenging. While lameness of orthopaedic origin should be evaluated in these patients, diagnostics focused on non-orthopaedic causes should also be taken into consideration.

ETHICAL APPROVAL

As a review article, there is no requirement for ethical review and approval. No original experimental data relating to client-owned animals was presented in this manuscript.

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