EXERTIONAL HEAT ILLNESS IN THOROUGHBRED RACEHORSES: OBSERVATIONS AND TREATMENT IN THE FIELD  by Meg Brownlow p. 13
Chlamyphilia causes pain and distress.

Antibiotics will eventually work but ... wouldn’t it be better to prevent it?

Maybe it’s time to break the cycle.

“Cats with Chlamyphilia infection can experience quite intense and uncomfortable ocular inflammation. With no obvious downsides, practitioners should seriously consider vaccinating against Chlamyphilia routinely, particularly in younger cats.”

Prof T Graffyfield-Jones, VetMed PhD DipECVIM MRCVS, University of Bristol, 2013
FROM THE DIRECTOR

Welcome to the first edition of C&T for 2014. You may notice some changes in style and layout of this edition as our new marketing team has worked hard to provide a fresh appearance to enhance the fantastic content from our contributors.

Lis Churchill and Richard Malik continue as the main editors, while the design and layout have been transformed by our new marketing manager Iнес Borovic and design officer Monique Coffey. I think they have done a great job working under pressure to meet our production deadlines and should be congratulated on their results.

As is often the case with C&T, we have a mixture of articles across a broad range of species, from macropods to horses and goats, to dogs and cats. Many of these articles are quite extensive and we appreciate the time and effort it takes to produce articles of such high quality, often supported by great photos and video clips, which are accessible by viewing the ebook version of the C&T. This edition has really made me reflect once again on the wonderful diversity that a veterinary career provides and the profession.

This edition has really made me reflect once again on the wonder of life-long learning. We have all had our own journeys and paths taken by members of the profession. Tom Hungerford’s words about education has equipped them with the tools to pursue whatever path they choose. Tom Hungerford’s words about the variety of paths followed by members of the profession.

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10 - 11 Apr Feline Tales Melbourne
4 May Small Animal Dentistry Adelaide
1 Jun Dermatology Canberra
16 - 19 Jun Cradle to Grave - Small Animal Health and Wellbeing Conference Melbourne
29 Jun ecoCPD: Radiology Adelaide
15 - 18 Sep Canine and Feline Internal Medicine Conference Honolulu, USA
19 Oct Small Animal Dentistry Port Macquarie

WORKSHOPS

29/30 Mar Basic Echocardiography Sydney
19 Jul Hip & Stifle Sydney
20 Jul Bone Plating Sydney
9 Aug Feline Surgery Sydney
10 Aug Canine Surgery Sydney
16 Aug Feline Surgery QLD
17 Aug Canine Surgery QLD
26 Sep Basic Echocardiography Melbourne
27 Sep Advanced Echocardiography* Melbourne
8/9 Nov Basic Echocardiography Cairns

* Prior learning will be required to attend this workshop.

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22 May Backyard Chooks Part 2
5 Jun Pancreatitis in cats
26 Jun IMHA and how to prevent pulmonary thromboembolism
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7 Aug Blue tongues, turtles and frogs
21 Aug Management of CHF in dogs with mitral valve disease
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DOES AUSTRALIA BREED COMPANION ANIMALS ETHICALLY?

WEBINARS

Dr Robert Dixon (1921-2011) Champion for Animal Welfare

Robert Dixon graduated from the University of Sydney in 1973 with a BSc(Wet) degree before completing his BVSc degree in 1974. Robert completed his PhD in 1980 in New Zealand and returned to the University of Sydney in 1985 to take up an academic appointment within the Faculty of Veterinary Science. For many years Robert held the Faculty position of Sub-Dean Animal Welfare as well as serving on the University Animal Ethics Committee. Robert expressed his friends and colleagues with his determination, passion, positive attitude and sense of humour as he battled with ill-health throughout his life. Robert’s email signature finished with a quote from Mohandas Gandhi – “The greatness of a nation and its moral progress can be judged by the way its animals are treated.

To view past Robert Dixon Memorial Symposia please visit www.cve.edu.au/animalwelfare
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• QUEENSLAND UNIVERSITY: Tracey Gowen
• UNIVERSITY OF MELBOURNE: Kathy Luk
• UNIVERSITY OF SYDNEY: Duncan Pearce

Established in 1969, the C&T is the brainchild of Dr Tom Hungerford, first Director of the PGF (1968–1987), who wanted a forum for unencumbered and unedited material. Tom wanted to get the clinicians writing...

Tom wanted to get the clinicians writing...

...not the academic correctitudes, not the theoretical niceties, not the super correct platitudes that have passed the panel of review... not what he/she should have done, BUT WHAT HE/SHE DID, right or wrong, the full detail, revealing the actual “blood and dung and guts” of real practice as it happened, when tired, at night, in the rain in the paddock, poor lighting, no other vet to help."

Paul graduated from the Faculty of Veterinary Science at Sydney University in 1989 with first class honours. The son of a veterinarian, Paul was particularly close to his mother Christie and his brothers Con and Ian. Paul ended up taking over his father’s practice in Casula, buying him out in 1986 and continuing to run the hospital till 2011. This was a substantial undertaking for a recent graduate. Paul gave himself the brief of transforming the practice into a progressive one that focused on the needs of the clients and patients, whilst simultaneously upgrading the plant and facilities, to ensure that the veterinary care was of the highest quality. Through hard work, determination and significant natural ability, Paul was highly successful in this venture. He was greatly aided by his business partner and first wife, Miriam Meek. Together, they made a formidable and compassionate veterinary team. Other veterinarians who shared Paul’s vision of veterinary practice soon joined him. Over the years Paul opened branch practices in neighbouring suburbs with Casula remaining the main treating hospital and focal point.

Paul was one of that endangered species, the omnicompetent clinician, comfortable with each and every aspect of small animal practice. His passion, however, was surgery, a discipline for which he showed both great aptitude and ability. Paul bought the best available range of surgery instrumentation, especially for the practice of his favoured orthopaedics. He undertook advanced training in orthopaedic surgery through courses, workshops and independent study.

He completed his MACVSc in Small Animal Surgery by examination at the first attempt, and subsequently became an active member of that Chapter of the College, serving as its Treasurer (2004–2013). MACVSc-examiner and an active and dynamic member of its organising committee. Paul embraced the concept of life-long learning. He kept abreast of the latest developments in veterinary knowledge by constantly reading journals, attending seminars and courses, and undertaking cutting edge workshops. He would become excited by new techniques and new technology, and his enthusiasm was infectious.

Paul spent his whole career serving the people and pets of south-western Sydney. It’s a complex part of Sydney, with a multicultural mix of people widely varying socio-economic status and personal values. Paul and his team took all this into their stride, dealing most effectively with people from all walks of life. This speaks volumes for his ability to relate to different people, be a cogent and effective communicator and an advocate for his patients. While offering outstanding medical and surgical services to his patients and clients, Paul managed to create a thriving business enterprise. It was a matter of routine for Paul to conduct major surgeries on patients whose owners had strong emotional commitment, but without the accompanying ability to pay. It didn’t matter to Paul and his team—they would more often than not just give it a go, and hope the people would eventually give him some recompense. Clearly, his try came from helping the animal. Paul also had the notion of what he called “a learning case” – where if the owner didn’t have the ability to pay, but the case represented a surgical or medical challenge which made the undertaking worthwhile, he would proceed to test and hone his surgical skills, while permitting him to develop or master a new technique. There is no doubt that after 10 years in practice he was routinely performing both orthopaedic and soft tissue procedures that most other practitioners would refer to a specialist surgeon, but at a fraction of the price of a referral surgeon. This meant a lot to his clients in the Liverpool area.

As an owner and manager, Paul was demanding of his staff who he expected to live up to his high but
practicable standards. Paul built enduring relationships with his team over the years, including his practice manager Liana Stieamandri who worked with Paul for over 20 years. It was because he set the bar so high, but at the same time was incredibly supportive and respectful, that he was a fantastic mentor for associates, who were willing to put in the hard yards. It was not possible for Paul to continue full-time practice after he became ill, but when in remission, he would return to the practice, and especially the operating theatre. Around this time Paul found additional ways to give something back to the profession by taking an active role in the management of the Centre for Veterinary Education (CVE; formerly the Postgraduate Foundation in Veterinary Science or PGV) and the Faculty of Veterinary Science, at the University of Sydney. Paul became a member of the CVE Council and challenged everybody with his take on the future of our rapidly changing profession, especially in relation to trends in practice and the use of technology. He also worked for the CVE as a consultant, and helped chair conferences and symposia, as well as being a sounding board for the Director, particularly during restructure of the organisation in 2013 through a change management process. In 2012 Paul returned to the Veterinary Faculty as a Tutor in Anatomy, and there was never a more effective demonstrator in this discipline than Paul, teaching students the importance of structure and function from the perspectives of both surgery and diagnostic imaging. Paul’s intellectual curiosity and quiet confidence and competence also greatly buoyed the spirits of his anatomy colleagues. He was a driving force and welcome presence in the teaching team. He also helped out by doing sessional work at the Veterinary Teaching Hospital, mainly on weekends, where as usual, he was an engaging, challenging and inspirational teacher.

In the years following his diagnosis and subsequent treatments Paul was inspired to volunteer his professional skills. He taught surgical practices pro bono for a short time in Indonesia with a grassroots organisation that provide care to street dogs in Bali. Paul also worked with a homelessness service to provide free veterinary consultations to locals in the Kings Cross area. Paul found this work incredibly rewarding, as he was always touched by the care people showed their animals despite their economic situations. There was a great deal more to Paul than veterinary science, as he was one of those individuals who worked and played hard, in equal measure. He was passionate about swimming, cycling, eating, cooking, coffee and music. He read widely, in all genres, had wide-ranging and eclectic tastes in cinema and fiction, and had a deep understanding of history, politics and world affairs. It’s hard to know how he managed to squeeze all these interests into his life, considering the long hours he put in at Casula Veterinary Hospital and Elizabeth Drive Animal Hospital. Paul had an extraordinary large circle of acquaintances, from academics to bohemians, covering every social demographic imaginable. During his long battle with cancer, these friendships were deepened and strengthened, and special credit goes to his wife Kate who supported him through everything with her extraordinary love, strength of will and ability to make the most out of everything, and move forward. He also was greatly supported by his older brothers Con and Ian, and indeed his illness brought them all closer together. Paul was one of those people to make strong and enduring friendships, and he will leave a substantial legacy. His team at Casula continue to practice veterinary medicine with the philosophy he instilled in them, and Agnes Chiu is a deserving yet reluctant protégé, and very proud to accept the baton from Paul. Penny Tisdall, who started with Paul as a new graduate, is now the senior surgical specialist in Adelaide, and no doubt her career trajectory and moral compass had a lot to do with the formative years she spent at Casula.

Paul was a very special human being who enriched the lives of others. He was a man of many talents and interests who cared deeply about people, pets (including his own menagerie of cats and dogs) and life. Paul always said, ‘It’s about making a difference’ and what a difference he had made to everyone he touched. Paul is survived by his wife Kate, his brothers Con and Ian, his sister-in-law Christina and by his two nieces Stephanie and Anna. Vale Paul Gotts-Graham.

Richard Malik, Agnes Chiu, Corinna Klupiec, Wallace Lewis, Penny Tisdall, Kate DeMaane and Paul J Ccanfield

Compiled at the Currumbin Sanctuary Wildlife Hospital by Mimi Dona © 2010

This series is the result of collaboration between Mimi Dona & Dr Michael Pyna of Currumbin Wildlife Sanctuary Veterinary Hospital. Film clips courtesy of Lincoln Williams, fotomedia (www.fotomedia.com.au). Non CVE members can access these flashcards and videos at www.cve.edu.au/candt2013

PART 4: WILDLIFE FLASHCARD SERIES

MACROPODS

Macropods

Be aware:

• Can inflict a nasty bite, scratch and have a powerful kick that can cause significant trauma.

• Adult macropods are difficult to restrain manually – should be chemically restrained for transport/ treatment.

• All pouch emerging joeys and adults should be assessed for myopathy. Adult macropods do not cope with being in captivity and succumb to myopathy very quickly, especially following trauma-related incidences.

• Female Macropods have a pouch; always check for joeys. Never pull a Joey off the teat; remove very gently or cut the teat off the bottle and pin to the inside of a cotton pouch liner.

• Unlumed joeys require regular feeding (every 2 to 3 hours) and until they reach pouch emergence stimulation for defecation and urination.

• Marsupial young are born very under-developed and until the stage of development that we are able to successfully hand rear young joeys, they are considered to be ‘unviable’. Consult a trained/experienced person for assistance with identification and confirmation on viability.

• Native marsupials don’t get tick paralyses.

Handling

• Take care not to induce myopathy when attempting to capture or restrain macropods.

• Medium to large macropods need to be chemically restrained; due to the risk they should only be caught by someone experienced/trained. To restrain a large macropod that is on the ground unable to get up, use several people to go in from behind, sharing the weight to hold down the tail and shoulder area, stopping it from turning. Cover the head with a towel and stay away from the legs. Mobile and injured macropods will require a licensed person with a dart/ tranquilizer.

• For small macropods, grab the base of the tail near the rump and lift off the ground facing the legs away from you and away from others. Support the chest with the other hand and face the legs away from you and others. To place in a bag (pouch) have someone hold in development that we are able to successfully hand rear young joeys, they are considered to be ‘unviable’. Consult a trained/experienced person for assistance with identification and confirmation on viability.

• Marsupial young are born very under-developed and until the stage of development that we are able to successfully hand rear young joeys, they are considered to be ‘unviable’. Consult a trained/experienced person for assistance with identification and confirmation on viability.
Housing the sick or injured macropod

- Preferred enclosure temperature for Adults is 26°C. Orphaned joeys just furred to furred 28°C to 30°C and 32°C for unfurried.
- Pinkies can be placed in a cotton pouch in a plastic aquarium lined with towels and must be given heat – just furred/furred 28°C to 32°C and unfurried 32°C. Ideally they should be housed in a Vetarco® or Humidi crib. This must be monitored with an indoor/outdoor thermometer.
- To reduce stress, place furred orphans in a cotton pouch and then in a make shift pouch using a pillowcase and coat hanger. Place towels underneath and inside for additional comfort and support. Always provide a quiet place during the day and a safe closed room in case they get out. Alternatively the cotton pouch can be placed in a plastic carry cage lined with towels or small blankets.
- Large joeys can be housed temporarily on a soft bed post-operatively or if unable to move and in a recumbent state. Hand raised joeys once standing can temporarily be housed in a room (free of clutter) or outside pen.

**Emergency diet**

- Only offer food once rehydrated.
- If you are familiar with the species, grasses, native herbs and vegetation (be careful where collected avoid sprayed/contaminated areas) are ideal for just-furred and onwards.
- Orphans can be given water and glucodin initially for first two feeds, then suitable milk replacer (Wombaroo® Kangaroo Milk Replacer, Brolar® M 100 or Wombatmilk® Kangaroo Milk Replacer).
- The lateral caudal tail vein is the preferred route for intravenous access.

**Assessment under anaesthetic**

- **Gaseous**
  - Use an anaesthetic mask at 5% induction, can take up to 5 minutes (very stressful with wild macropods; best to use suggested injectable agent)
  - Maintain using a mask on isoflurane at 1.5 to 2% with an oxygen flow rate of 1 L/min.
- **Anaesthetic agents**
  - **Zoletil**
    - Small to medium – 10mg/kg – (I/M)
    - Medium to large – 5mg/kg – (I/M)
  - **Alfaxan CD RTU 3 mg/kg – (I/M)**
  - Due to the difficulty with intubation of Small Macropods and IPF in case of apnoea, gaseous anaesthesia is recommended.

**Intubation**

- Intubation is difficult in macropods and respiration under anaesthesia is reliable. Intubation is generally reserved for when oral/racial surgery is required and a facemask is not possible.
- Cuffed endotracheal tube or catheter tip, insert the endotracheal tube with the aid of an anaesthetic spray and tie in with shoelaces.

**Recovery**

- Be careful of uncontained larger macropods when waking up from anaesthetic as they can wake up very quickly.
- Use a Bair hugger® or heat mat and room temperature to maintain the patient’s core body temperature throughout the procedure, using a cloacal thermometer to monitor. With joeys, Vetarco® or Humidi cribs are ideal during post-operative recovery.

**Myopathy – poor prognosis**

- Vitamin E Selenium can be used as a preventative treatment
  - Mild cases or as a preventative 1mL/50kg SID for 3 days (I/M)
  - Moderate cases (bad orphaning) 1mL/30kg SID for 3 days (I/M)
  - Severe cases (dog attack etc) 1mL/20kg SID for 3 days (I/M)
  - Pamrin
    - 1mL per 10 kg for mild sedation and 1mL per 5kg for heavy sedation.
  - Fluid therapy

**Fluid Therapy**

- It is important to remember to warm the fluids being administered. Using 0.9% sodium chloride, dose the patient at 5% of its bodyweight. Fluid therapy can be administered subcutaneously or standard IV infusion rates. Syringe pumps are ideal to use in orphans.

**Preferred routes of drug administration**

- Subcutaneous – administered in loose skin at lateral neck/chest, side of abdomen or over thigh area.
- Oral – given via a syringe.
- Intramuscular – administered to dorsal lumbar muscles, cranial and caudal thigh, upper arm, gluteal.
- Intravenous – lateral caudal tail vein is preferred or cephalic, saphenous.

**Euthanasia methods**

- Injection of sodium pentobarbitone can be administered either by intravenous, intracardiac or intraperitoneal routes.
- If administering by intracardiac or intraperitoneal, macropods must be anaesthetised first. Pinkies do not always reach appropriate levels of anaesthesia in a gas chamber, Alfaxan® I/M is recommended.

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Authors' views are not necessarily those of the CVE
On Monday 07.11.12 my colleague Dr Jim Phelan and I were attending a native fauna exhibit to capture a young male swamp wallaby (Wallabia bicolor) prior to castrating him and we were asked to check a young white female Red Neck wallaby (Macropus rufogriseus) (Fig. 1). She had a swelling over the caudal aspect of the right maxilla and swollen eyelids of the right eye (Fig. 2). The swelling had been present for no more than 2 days over the weekend. The attendant had been feeding the joey formula daily on week days and she is a keenly observant carer. My initial thought was ‘Hope this is bruising caused by a collision with a fence or enclosure equipment but since this unusual animal is the subject of a naming competition among the local primary school kids, / bit it is lumpy jaw!’ We have treated ‘lumpy jaw’ in a variety of wallaby species and eastern Grey kanga ros without great success. Because we were fully occupied with capturing and transporting the animal to be castrated, we did not have time to examine the joey closely. I asked the carer to ring me the following day hoping that the swelling might subside if it had been due to bruising. There was also significant swelling of the buccal gingiva. Radiographs showed a lack of bone density at the base of the loose molar. The molar was extracted without difficulty and the alveolus was swabbed for bacterial culture and susceptibility testing. Results of the culture were obtained from Vetnostics on 11.11.12. Anaerobic bacteria had been cultured. Susceptibility testing was not done and the laboratory commented that Anaerobes are predictably susceptible to Metronidazole®. Alternative agents include clindamycin and Augmentin®.

There are ample reports in the literature and on the internet that outline various treatment regimes for this condition. Various bacteria have been cited as primary infectious agents in this condition, how they invade the tissue and how a variety of secondary infectious agents relish the opportunity to invade and cause intractable diarrhoea. Our second choice was clindamycin (11mg/kg bid, Dalacin C Phosphate Injection 500mg/30mL, Pfizer Australia) and Clavulox® 10 mg/Kg sid (140mg/mL Amoxicillin Trihydrate & 35 mg/mL, Clavulanic Acid, Pfizer Animal Health) and this was started on 16.11.12. The carer was concerned that the antibiotics would change the gut flora and cause diarrhoea. We explained that we also were concerned about this possibility but that the likely outcome of not administering an effective antibiotic course would be one deceased joey. We had previously instructed the carer, who had completed an animal care course at TAFE, on how to administer the injectable antibiotics.

This joey had been housed with her mother and other wallabies in an open air enclosure. Her diet had been supplemented by bottle feeding Biolac M 200 (2 scoops equivalent to 24 g powder/100mL warm water) formula once daily and was no trouble for the carer to handle. We decided to separate the joey from her mother for the course of the treatment so that the carer could take her home on weekends and also for a 3 week period when she took annual leave on the North Coast. There was some stress involved in separating the animals and confusing the joey indoors and we administered Modocate® (Flufenazaine decanoate 50 mg in 2 mL, Bristol Myers Squibb Aust. P/L) at a dose of 2.5 mg/kg, I.M. on 16.11.12. The joey tolerated the injections and appeared relaxed with her carer on the numerous occasions when I examined her. There were a few days when the joey did have pasty faeces but she never stopped drinking her formula and continued to gain weight. That was an encouraging sign. The swelling over the maxilla fluctuated in size and on 27.11.12 we again anaesthetised the joey to examine her mouth. The second molar was loose and was removed. On the same day the carer left with the joey on her annual holidays. We were concerned that the antibiotic course was ineffective but decided to prolong the course. She was supplied with 3 weeks of Clavulox® and Clindamycin® in syringes. On 17.12.12 I examined the joey on site and administered a second dose of Modocate and supplied the carer with more Clindamycin®. We arranged to examine her mouth while anaesthetised and did so at our clinic on 19.12.12. At that time the swelling had gone and the remaining teeth were firm and there was no apparent inflammation. The Clavulox® and Clindamycin® were continued for a further 2 weeks. The joey has been returned to the enclosure with her mother and several other wallabies and has had no recurrence as of 13.05.13. The winner of the naming competition chose ‘Lily’ for her name, I would have chosen ‘Lucky’.

A point of interest in this case includes the length of the course of the appropriate antibiotics to achieve a cure. If presented with an animal with lumpy jaw the carers / owners must be advised of the labor intensive process, the cost of the antibiotics and the possibility of repeated anaesthetics to monitor and treat infected bony tissue. Even at our ‘friendly’ rates the cost of this exercise was substantial.

Figure 1. Female white red necked wallaby.

Figure 2. Note swelling over the caudal aspect of the right maxilla and swollen eyelids of the right eye.

Figure 3. Examination of the teeth was conducted after the anaesthetic was administered.

Figure 4. At caring for Lily.
Another consideration in this case is that the opposing molars to those that were extracted may elongate or develop sharp spurs because they may not be ground down during mastication. These molars may need to be rasped in the future.

We have experienced poor outcomes in the past in treating this condition and have researched the various treatment regimes that have been recommended. This animal responded to the antibiotic and surgical intervention we have described.

This is a good article regarding the difficulties encountered in attempting to treat lumpy jaw in macropods.

In the young wallaby, the first 2 ‘molars’ are actually premolars which will eventually be followed by 4 molars so perhaps it would be better to just refer to these teeth as cheek teeth to avoid confusion. The Red-necked Wallaby undergoes molar progression as it ages with the caudal molars pushing the arcades forward as they erupt (in fact, this is used for ageing purposes). This usually results in the shedding of the more rostral teeth.

The process of odontoclastic root resorption allows these teeth to be lost and the inflammatory responses in the vicinity of the tooth roots is a perfect environment for bacterial proliferation especially of anaerobic bacteria such as Actinomyces spp. etc. Radiographs of this young wallaby show the presence of erupting molars which are causing some displacement of the teeth already present in the arcades. In herbivorous animals rotation or malposition of any teeth in a tightly packed arcade allows food trapping and decomposition (this is the form that periodontal disease takes in these species) with proliferation of anaerobes especially. Unfortunately for macropods, infection with these anaerobes can spread to the adjacent bony structures and lead to lumpy jaw.

Treatment of choice for these infections is good debridement, where possible, followed by sodium iodide injections usually with good results. Unfortunately sodium iodide frequently has untoward reactions in macropods hence other therapies are needed such as diltiazem. Chlorhexidine oral washing after thorough surgical debridement has also been used with success in wallabies that are amenable to the frequent restraint required for this type of treatment regime. These animals can be trained to accept a flavoured chlorhexidine relatively quickly (Mercado®; Vetplus).

As pointed out in the article, cost of treatment is always an issue with these and euthanasia is an option. The macropod teeth are brachydont type of treatment. These animals can be trained to accept a flavoured chlorhexidine relatively quickly (Mercado®; Vetplus).

Data on the incidence of exertional heat illness (EHI) in thoroughbred racehorses is unavailable but during the summer months in eastern Australia when the ambient temperature and humidity are high, horses suffering from EHI manifesting as a heat stress/heat stroke syndrome are not uncommon.

Appropriate management of these horses is critical as EHI represents a medical emergency. This article attempts to define and describe the spectrum of EHI syndromes and to provide a rationale for treatment protocols in the field.

Introduction

The thoroughbred racehorse is an exceptional athlete, galloping at very high work intensities at speeds of about 70 kilometres per hour over relatively short distances, with some races completed in less than a minute. To achieve this, the heart rate peaks at 250 beats per minute, cardiac output may rise to 450 litres a minute and oxygen consumption is from 160 to 200 millilitres of oxygen per kilogram per minute (Sharp, 2012; Young, 2003; Hodgson and Forman, 2014). It has been calculated (Hodgson, 2014) that heat production under such circumstances can reach 450 kcal/ minute, and possibly elevate core body temperature by about 1°C/minute. Unless the heat stored during exercise is dissipated the body temperature will continue to rise to a hypothetical 42°C, which has dire consequences for the animal concerned because heat is toxic to cells and initiates a complex, progressive array of pathophysiology (Bouchama and Knochel, 2002).

In moderate environmental conditions (for example, temperature 20 to 25°C and humidity below 50%) metabolic heat from strenuous exercise is dissipated by radiation and convection to the atmosphere and through evaporative cooling by sweating. However, when the skin temperature and ambient temperature are equal, evaporative cooling is the only avenue for heat loss. In conditions where there is high ambient humidity, the water vapor pressure gradient from skin to air is decreased, thus limiting the ability of the body to lose heat by sweating and posing a serious threat to thermoregulation (McCutchcheon and Geor, 2004).

While the physiology of thermoregulation in the horse is comprehensively described in the literature (for example, Guthrie and Lund, 1998; Hodgson, 2014; Hinchcliff et al., 2004), Hodgson et al., (1994), there is scant information on the clinical syndromes of thermoregulation observed in thoroughbred racehorses, whose performances are characterized by short bursts of strenuous, largely anaerobic activity at high intensities (Gerard et al., 2014). This is in sharp contrast to the endurance horse, whose heat exhaustion syndromes have been extremely well documented but are essentially quite different, endurance activity being by nature aerobic, protracted and submaximal in nature, with dehydration a key feature (Lucke and Hall 1980; Landon Fidley et al., 2009).

In the capacity of a race day official veterinarian for Racing New South Wales, the regulatory racing body in New South Wales (NSW), Australia, the author has over a period of twenty years observed and treated horses presenting with EHI syndromes. Unquestionably, such cases constitute a real medical emergency and are extremely challenging because of the narrow ‘window’ (Yuval et al., 2004) of opportunity for recognition and effective treatment. The highest priority therapeutic objective is to reduce core body temperature to a safe level within thirty minutes if possible, but certainly in no longer than sixty minutes, as recommended by Casa et al., 2007 in humans.

Exertional Heat Illness in Thoroughbred Racehorses: Observations and Treatment in the Field

Meg Brownlow

The author would like to acknowledge the influence of Professor David Hutchins in learning to appreciate the value of simple clinical observations.

The value of simple clinical observations.
This paper defines the syndrome of EHI in the thoroughbred horse, describes the spectrum of clinical signs, and presents the rationale for treatment in the field.

**Description of Exertional Heat Illness (EHI) in the Thoroughbred Race Horse**

EHI is represented by a continuum of clinical signs along a common pathway from the milder forms of EHI through to death. The mechanism of evapaporation heat loss through sweating becomes relatively inefficient at controlling body temperature. The wet-bulb globe temperature (WBGT°C) is by far the most widely accepted index of heat stress used throughout the world. It takes into account air temperature, humidity and wind velocity to provide a single reading, and there are well-established limits to help users interpret readings and thus predict the risk of thermal injury. An increase in WBGT°C from 21°C to 28°C indicates extreme risk. Levels of WBGT°C above 28°C indicate very high risk of thermal injury, and thus predict the risk of thermal injury (Yaglou and Minard, 1957; Casa and Roberts, 2003; Budd, 2008). The concept of WBGT was introduced in the early 1960s in an attempt to control heat illness in training camps of the United States Armed Services. Since that time has been adopted by sporting regulatory bodies world-wide, the US Occupational Health authorities and the American College of Sports Medicine. For humans, WGBT levels of 21 to 25°C indicate extreme risk (Sports medicine Australia, 2013; Binkley et al., 2002). Schroeter and colleagues (1998) used the WBGT to quantify environmental heat loads during the three-day event competitions at the Atlanta Olympics. Racing NSW, the regulatory body for horse racing in New South Wales, Australia, has adopted policy recommendations concerning racing in hot weather based on WBGT levels (Racing NSW, 2009). Above a certain level, WBGT 26°C rise to 28°C there are changes to race-day programming and procedures, the provision of strategically placed horses, quantities of ice made available and an extra veterinarian assigned to monitor post race recovery and to treat horses with signs of EHI if necessary. In the author’s experience the WBGT has been useful in predicting the occurrence of EHI; once a level of at least 24°C was reached it was highly likely that on that day we would encounter an increased number of horses with thermoregulatory problems after racing. It should be pointed out, however, that it is a misconception to assume that EHI will only occur on hot days. This author has seen cases of EHI on relatively cool days, and Roberts (2006) has reported heat-related illnesses in humans at an air temperature of only 9.4°C but with relatively high humidity. In my experience, the clinical continuum of EHI, in order of increasing severity, includes horses that present as:

(a) **Level 1** – Very ‘hot’ and distressed (see Fig. 2). There are increased rates of respiration (>60-100 breaths per minute) with widely dilated nostrils and heaving thoracic excursions. The heart rate is always elevated (>150 beats per minute) and the animal is usually sweating profusely. Most importantly the skin is extremely hot to the touch.

(b) **Level 2** – Irritability: these animals demonstrate unusual behaviors such as kicking out in a random fashion (which may be confused with a colic episode) and are becoming uncooperative (see Fig. 2).

(c) **Level 3** – Altered mentation toward depression and incoordination (wobbly) may be evident. Horses in this group can become unmanageable, plunging and stopping haphazardly with wide-based strides and taking meticulous steps (see Fig. 1).

(d) **Level 4** – The final stage, ‘heat stroke’, is where the horse demonstrates substantial levels of central nervous system dysfunction (encephalopathy), throws itself down or falls repeatedly and is a risk to itself and its handlers. This latter group will progress towards death unless treated rapidly and effectively (see Fig. 4).

### Table 1. Exertional Heat Illness Syndromes in Thoroughbred Race Horses

<table>
<thead>
<tr>
<th>Level</th>
<th>Clinical Signs</th>
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</thead>
<tbody>
<tr>
<td>1. <strong>HOT</strong> horses</td>
<td>Normal mentation</td>
</tr>
<tr>
<td>2. <strong>HOT</strong> horses + Irritability</td>
<td>Altered mentation</td>
</tr>
<tr>
<td>3. <strong>HOT</strong> horses + altered mentation + incoordination + ‘wobbly’</td>
<td>Incoordination</td>
</tr>
<tr>
<td>4. <strong>HOT</strong> horses + gross signs of central nervous system dysfunction + falling over – ‘dangerous’</td>
<td>Death</td>
</tr>
</tbody>
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### Table 2. Treatment Protocols for Various Levels of Exertional Heat Illness

#### Level 1: ‘HOT’ horses
- Need to be effectively cooled with copious amounts of cold (iced) water by hosing or bucketing and scraping the heated water off the skin.
- Important to monitor heart and respiratory rates and skin temperature.

#### Level 2: ‘HOT’ horses + irritability
- Need to be effectively cooled as above.
- Provide sedation and inhibit serotonergic activity.

#### Level 3: ‘HOT’ horses + altered mentation + incoordination ‘wobbly’
- Need to be effectively cooled as above.
- Provide sedation and inhibit serotonergic activity.
- Provide flunixin meglumine to control SIRS.
- Provide neoprogen and stabilize cell membranes.
- Address hydration status – may need to give intravenous fluids intravenously.

#### Level 4: ‘HOT’ horses + gross signs of central nervous system dysfunction + falling over – ‘dangerous’
- Need to be effectively cooled as above.
- Provide sedation and inhibit serotonergic activity.
- Provide flunixin meglumine to control SIRS.
- Provide neoprogen.
- Address hydration status – may need to give intravenous fluids.
core body temperature elevation. It is also important to emphasize that a diagnosis of EHI should not rely solely on the clinical finding of an elevated rectal temperature because this may not be observed due to the lag period of heat build-up prior race, often a faceted endotherm in nature, and the risk involved in actually taking a temperature from an animal with instability and altered mentation. It is not considered necessary or a priority to establish hyperthermia before instituting treatment.

**TREATMENT PROTOCOLS FOR EXERTIONAL HEAT ILLNESS**

Effective and rapid cooling

It is considered an absolute priority that horses with EHI are recognized early, and this requires the active involvement of those who are familiar with the variations in clinical signs described above. Horses whose heart and respiratory rates remain elevated post-race cannot be targeted and presented to the treatment group for evaluation. Ideally, the treatment area should contain a wet room where horses can be hosed and where ice-water is readily available. An air-conditioning unit or large fan is also advisable.

Effective cooling strategies are the cornerstone of treatment. Casa and colleagues (2007; 2010) have investigated a wide variety of cooling modalities, concluding that ice-cold water immersion is the gold standard for treatment of exertional heatstroke in humans. The physical characteristics of water make it vastly superior to air as a cooling medium. Flow of blood to the skin is increased to accelerate heat transfer to the environment and if this is effective the body core will cool without consequence. This redistribution involves a decrease in blood flow to the gut, and if this is prolonged gut ischemia will result, stimulating oxidative and nitrosative stress. The tight junctions of intestinal epithelial cells increase their permeability and allow the influx of bacterial endotoxin, which then gains access to the systemic circulation (Dokladny et al., 2007). In a most interesting study Donovan and colleagues (2007), using a high speed treadmill, exercised horses to absolute fatigue and found that the brief but strenuous exercise induced endotoxemia and a systemic inflammatory response characterized by elevations in cytokines and prostaglandins and thromboxane which produce many of the clinical signs of endotoxemia in horses (Moore et al., 1998; Semard et al., 1987; Bottoms et al., 1981). In the author’s experience, horses with a history of administration of flunixin at therapeutic dose rates improve clinically within ten to twenty minutes.

The pathophysiology of the EHI pathway and implications for other treatment modalities

Research in humans in the last decade (Epstein and Roberts, 2011; Leon and Helwig, 2010; Bouchama and Knochel, 2010) has provided new insights into the pathophysiology of heat stroke and suggested an integrated view, depicting a common pathway from heat stress to heat stroke (see Fig. 5). Stress is important for treatment because it explains the progressive nature of the illness and therefore the need for all phases of EHI to be treated as aggressively as possible. There is convincing evidence that the severity of the illness will be determined by the critical thermal maximum, a term which attempts to quantify the level and duration of elevation in cell temperature that will initiate and potentiate progressive tissue injury (Shapiro et al., 1973; Byun et al., 1978).

Brieﬂy, from the model of heat stroke based on observations in humans (Bouchama and Knochel, 2002; Leon and Helwig, 2010) and experimental models of heat stroke in laboratory animals, it is suggested that during strenuous exercise there is an elevation of core temperature which stimulates physiological changes. Flow of blood to the skin is increased to accelerate heat transfer to the environment and if this is effective the body core will cool without consequence. This redistribution involves a decrease in blood flow to the gut, and if this is prolonged gut ischemia will result, stimulating oxidative and nitrosative stress. The tight junctions of intestinal epithelial cells increase their permeability and allow the influx of bacterial endotoxin, which then gains access to the systemic circulation (Dokladny et al., 2007; Lambert et al., 2002). In heat stressed horses endotoxin from the gut enters the circulation at a core temperature of 40°C and its concentration increases as the core temperature rises (Galligham et al., 1987). The immune system responds to endotoxemia by activation of the systemic inflammatory response syndrome (SIRS) with increased production of cytokines, interleukin-1 and other immune modulators including prostaglandins. This will cause haemodynamic instability, cardiovascular collapse and shock. In concert with the above, hyperthermia also causes a vasodilation of the cutaneous blood vessels, initiating coagulation pathways that lead to microvascular thrombosis and disseminated intravascular coagulation (DIC) (Sohal et al., 1984). The SIRS and coagulation pathways interact to cause multi-organ system failure and the progression from heat stress to heat stroke and death (Leon and Helwig, 2010).

To extrapolate this heat stroke model to horses, it is well documented that horses are susceptible to the syndrome with increased production of cytokines, interleukin-1 and other immune modulators including prostaglandins. This will cause haemodynamic instability, cardiovascular collapse and shock. In concert with the above, hyperthermia also causes a vasodilation of the cutaneous blood vessels, initiating coagulation pathways that lead to microvascular thrombosis and disseminated intravascular coagulation (DIC) (Sohal et al., 1984). The SIRS and coagulation pathways interact to cause multi-organ system failure and the progression from heat stress to heat stroke and death (Leon and Helwig, 2010).

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is a key driver of the exhausted horse syndrome (McCulloh and Gies, 2004; Hodgson, 2014). There is considerable individual variation amongst horses in the degree of sweating and consequent fluid loss and electrolyte perturbations, such as genetic predisposition, temperament, acclimatization to hot weather, ambient levels of temperature and humidity, diet, exercise, race duration and intensity, and individual motivation of each horse to race, which all play a role in determining thermoregulatory dysfunctions. Ineffective however of whether dehydration is a primary or secondary factor, management of dehydration must be included as part of the treatment protocol.

Central Nervous System Dysfunction Rationale for Treatment

The most challenging aspect of EHI syndromes in thoroughbred racehorses is a management approach for the emerging symptoms of central nervous system dysfunction and the risk this poses to the people trying to treat these horses.

Cerebral pathophysiology and pathology

It is well documented that the human brain, compared with other organs, is especially vulnerable to hyperthermia, and the severity of the injury depends on the level and duration of overheating (Nielsen and Nybo, 2003). In humans, hallmark signs of the progression of heat stroke illness include alterations to mental status such as dizziness, confusion, delirium, combativeness, collapse, seizures and coma (Nybo, 2007). The pathophysiological changes (see Fig. 6) responsible for this symptomatology are, firstly, reduction in cerebral blood flow leading to cerebral ischaemia, and changes to blood-brain barrier permeability allowing leakage of serum proteins, resulting in vasogenic brain oedema (Sharma and Hoopes, 2003). The latter is crucial in determining the extent of cell injury and consequent brain pathology because swelling of the brain within the rigid cranium has serious consequences. It is associated in humans with severe headache, the appearance of normal reflexes, alterations to consciousness (Yaqub and Ali Deeb, 1998) and the associated pathological picture is one of neuronal degeneration, particularly in the cerebellum where the Purkinje cell layer is most affected (Learon and Helvig, 2015; Binkley et al., 2002). It is worthy of note that during rapid cooling, about 30% of human patients in the heat stroke category experience permanent neurological impairments that may be related to interactions between temperature alterations and mental status such as dizziness, confusion, delirium, combativeness, collapse, seizures and coma (Argaud et al., 2007; Demattei et al., 1998).

Therapies – glucocorticoids

Therapies that address the central nervous system dysfunction include the use of therapeutic doses of glucocorticoids such as dexamethasone which has been shown to be beneficial in the treatment of brain and spinal cord injury in man, experimental cerebral ischaemia in animals and cerebral injury and trauma in horses (Hall, 1992; Behrmann et al., 1994; Feary et al., 2007; Reed, 1994). More recently Liu and colleagues (2000) experimentally investigated whether intravenous dexamethasone could protect against the heatstroke-induced cerebral ischaemia and neuronal damage in the rat heat stroke model. They used heat exposure before heat exposure or 80 minutes after the start of heat exposure and found dexamethasone reduced the heatstroke-induced arterial hypotension, the elevated intercellular levels, cerebral ischemia and neuronal degeneration, and resulted in prolonged survival. Administration of the drug before heat exposure rather than later gave superior results. The authors concluded that dexamethasone was an important modulator of the cerebrovascular and cytokine responses and thereby provided neuroprotective effects in the heat stroke model that they employed.

Synthetic glucocorticoids such as dexamethasone have potent anti-inflammatory and immunosuppressive effects and are widely used in the treatment of inflammatory disorders. There are general effects such as stabilization of cellular and lysosomal membranes and decreasing vascular permeability, as well as more specific effects aimed at modulation of the key chemical mediators of the inflammatory cascade (Amsterdam et al., 2002; Zink, 2004). Detomidine provides related sedative and related sedative and anaphylactic effects in horses with a rapid onset of action but of longer duration than dexamethasone (Grzegorczyk and Taylor, 1998; Vanis, 1998). It is beyond the scope of this paper to review the physiology of alpha-2 adrenergceptors but briefly they are located in the central nervous system and virtually every peripheral tissue. The alpha-2 agonists xylazine, detomidine medetomidine and dexmedetomidine stimulate presynaptic and postsynaptic alpha-2 adrenergceptors in the CNS, resulting in decreased levels of norepinephrine and dopamine and producing the clinical effects of sedation, analgesia and muscle relaxation which is the basis of their therapeutic efficacy. These alpha-2 agonists are selective but can also activate alpha-1 adrenergceptors to a small extent which may play a part in clinical effects but only activation of alpha-2 adrenergceptors produces sedation. At clinical doses the more selective a drug is towards the alpha-2 adrenergceptors the more potent it is as a sedative. The ratio of alpha-2 to alpha-1 selectivity has been classified as: medetomidine (160:1), detomidine (260:1), and xylazine (160:1); Virtanen and MacDonald, 1985; Virtanen et al., 1988; Virtanen and Nyman, 1985.

An in-depth study (MacDonald et al., 1988) of the neurochemical effects of medetomidine (a methylated derivative of detomidine) found that it depressed the turnover of noradrenaline and dopamine, and also significantly lowered serotonin levels. In view of this, it is suggested that detomidine might also cause some reduction in serotonin level. This is relevant because Shams and Dey (1989) have implicated elevated serotonin level as a key driver in the cerebral pathophysiology associated with the heat stress/heat stroke progression. It has been well documented that serotonin is one of the most powerful vasoactiveamines, capable of causing vasoconstriction of cerebral vessels, impairing the integrity of the blood-brain barrier, increasing cerebral vessel permeability and leading directly to cerebral oedema and neuronal injury (Winkler et al., 1995). These findings were supported by Kao and Lin (1996), who reported that when the cerebral serotonin system was antagonized experimentally in rats, heatstroke-induced ischaemic damage to the brain was reduced and the rats had increased survival compared with the untreated control group.
The equine veterinarian should be ready for the occurrence of heat stress on any race day especially during the summer months, which means having ice always available and ready access to the necessary medications. Emergency medicine is all about being prepared and time is the utmost importance for heat-affected animals. Staff in charge of horses at race meetings must be educated to recognize the behavior associated with heat stress in horses and be able to present the horse to the on-duty veterinarian as soon as possible. In an era when the welfare of racing animals is increasingly exposed to public scrutiny the racecourse veterinarian needs to be well informed and well prepared.

The author wishes to acknowledge all those who have worked tirelessly to treat heat-stressed horses on race day and who have contributed in some way to the observations in this article. They are: Craig Suann, Melissa Kay, Sue McMaster, Kyle Smallwood, Carol Griffiths, Pat Coazi and Natasha Pesce, and I also acknowledge the input from all of the fine racetrack veterinarians that we work with on a regular basis.

A very special thank you must go to Stephen Page for his most enthusiastic encouragement and editorial assistance to Mark Bradley for some of the excellent photographs and to the outstanding equine veterinarian Emily Streickle for intellectual discussions, rumination and debate on this most fascinating subject.

**References**


CVO Question: Flunixin is widely used in horses, whereas it’s a very tricky drug in dogs and cats. Would other newer generation NSAIDs work in horses in this setting? It’s just with gut ischaemia due to hyperthermia a COX2 selective NSAID may be safer for the gut, but maybe it would not work as well. Your comments? The non-steroidal anti-inflammatory drugs (NSAIDs) inhibit prostaglandins, which are central to the generation of the deleterious effects of the inflammatory cascade. Following an exogenous stimulus such as inflammation, or in my cases heat stress, cell membranes are damaged and phospholipid is liberated to form arachidonic acid. Cyclooxygenase (COX) is an enzyme that catalyses the conversion of arachidonic acid into prostaglandins, which play a significant role in both health and disease of the gastrointestinal tract, renal, skeletal and circulatory systems. There are two distinct forms of cyclooxygenase isoenzymes, referred to as COX1 and COX2.

COX1 is often referred to as the ‘housekeeping enzyme’ and is believed to have an essential role in the maintenance of healthy tissues, including gastric mucosal protection, renal function and vascular homeostasis. Inhibition of COX1 often elicits gastrointestinal toxicity in animals and humans, manifested by bleeding, ulceration, perforations and perforations. There appear, however, to be significant interspecies differences in both the level of COX1 and the ratio of COX1/COX2 expression in various tissues. This is most apparent in the gastrointestinal tract, so that extrapolation of toxicity effects between species from cats to dogs to horses may be misleading (Rad 2005). Apparently the cat is most sensitive, followed by the rat and dog, which may partly explain the overt sensitivity of this species to sub-therapeutic doses of the nonselective NSAIDs such as flunixin (Radzi and Khan 2006). Toxic effects of non-selective NSAIDs have been reported in horses (Macay et al., 1983), particularly for phenylbutazone. The literature has suggested, however, that providing the drug is used at clinical dose rates such problems are relatively uncommon. According to Mowat and colleagues (2001), flunixin administered at 3 times the recommended dose for 10 days resulted in few adverse clinical signs; however, some cases of peritonitis have been reported in ponies and foals. The COX2 isoforms, on the other hand, is highly induced by pro-inflammatory mediators in the setting of inflammation, injury, and pain and inhibition of COX2 accounts for most and possibly all of the therapeutic effects of the non-selective NSAIDs. Consequently, there has been an intensive search to identify and develop drugs with selectivity for inhibition of COX2. Flunixin meglumine is a non-selective COX inhibitor but according to Hsu and Kangsamy (2013) this drug shows greater COX2 inhibitory effects than COX1 in horses, in contrast to dogs where flunixin appears to exhibit preferential COX1 inhibitory effects. Flunixin was introduced into equine practice in the 1970s and since then has gained widespread popularity in the treatment of musculoskeletal disorders, for visceral pain with colics, and suppression of endotoxaemia, which has been well documented in the scientific literature. In a recent survey of 6,911 equine practitioners in America, Hubbell and colleagues (2010) reported that phenylbutazone and flunixin were the most commonly used NSAIDs. Flunixin remains one of the most useful and popular drugs for the equine veterinarian. Meloxicam is a NSAID with selective COX2 activity (Bretta et al., 2005), which may be a useful alternative to flunixin for the equine practitioner (Llum Funnell - 100ml $35; Norbrook Flunixin - 100ml $35). Meloxicam is marketed for horses as an intraavenous injection at 2-3 times the price of flunixin, containing 20mg/mL (Randall Meloxicam; Boehringer Vetoquinol - 100ml $135) with the recommended dose rates of 0.1-0.2mg/kg IV once daily, range of 0.05-0.15mg/kg IV once daily, depending on the horse’s weight.


CVA Question 2: With dogs, simultaneous use of dexamethasone and flunixin is avoided for fear of stomach ulcers. Is simultaneous use contraindicated in horses? Would you use flunixin for one grade, and dexamethasone for another? When treating exertional heat stressed (EHS)/heat stroke horses with a combination of flunixin and dexamethasone we are using a single one-off treatment to avert a potentially life-threatening situation. Nevertheless, the ramifications of that treatment need to be considered and the horses monitored post-treatment for any adverse consequences. Concurrent therapy with corticosteroid/NSAID combinations should only be used when medically necessary and where it is considered that the benefits of treatment outweigh the potential risk.

If veterinarians are concerned about using both drugs a choice can be made based on the level of symptoms. Anecdotal evidence from trainers who have had horses severely affected (Level 4) suggests that some of these animals may never fully recover in terms of their neurological functioning. The case in people with heat stroke is similar; Sharma (2007) describes that those who survive a severe heat stroke episode are often crippled with lifetime disabilities and may exhibit profound cognitive, sensory and motor dysfunction akin to premature neurodegeneration.

If substantial central nervous system dysfunction is apparent in heat stressed horses, neuroprotection with dexamethasone is warranted. Dextromethorin may also be neuroprotective by reversing to some extent the serotonin-induced alteration to the blood-brain barrier and progression to cerebral oedema. Finidyn is considered essential to halt the progression of the inflammatory cascade although dexamethasone will also achieve this to some extent.

The author has treated many horses with the combination of dexamethasone and finadine and is not aware of any adverse effects that would suggest the severity of gastrointestinal ulceration that has been described in small animals.

References


CVA Question 3: When I treat dogs locked in cars, I tend to sedate them with low doses of acepromazine and an opioid, the rationale being that the peripheral vasodilatation will be helpful when you go on to use cold water for cooling. Is this protocol of any value in the horse? I realize that acepromazine alone may be insufficient sedation for controlling the horse as per your article.

To answer your question:-
1. Our horses with EHI are already vasodilated (they have raced) and are often sweating profusely although the sweat may not be evaporating because of the high levels of humidity. We have to pay particular attention to our cooling techniques so that we don’t overcool and shut down the peripheral vasodilation. This requires constant monitoring of the skin temperature by touch, and observation of the network of dilated vessels of the skin – see photos.

2. In horses, acepromazine is not the best tranquilizer in terms of management, and can make horses more difficult to handle under some circumstances. For sedation in heat stress cases, xylazine or detomidine are far superior in terms of reliable manageability, and it’s interesting to observe that even when these drugs are administered at the higher dose rates the EHI horses don’t appear obviously sedated, i.e. head down and sedated stance. Might this be due to the pain management in the brain?

3. Many of the physiological and biological responses differ between exercise-induced hyperthermia (my heat stressed horses) and passive hyperthermia (your dog locked in a car). Jimenez and colleagues (2007) experimentally put one group of people in a climatic chamber to reach a rectal temperature of 38°C in 120 minutes while a second group exercised on a treadmill at 65% VO2max (submaximal) in controlled conditions to achieve a rectal temperature of 39°C after 120 minutes. Although many parameters measured, they found substantial elevations of catecholamines, which they thought were responsible for marked mobilisation of blood leukocytes and leukocyte subsets, in the exercise group only. The major hormone released during passive heating was prolactin, and it is hypothesised that this different hormonal response may have a role in a different immune response from that seen in the exercise model. The most interesting finding was that plasma volume was better preserved during exercise than during passive heat exposure. So, you need to give more fluids to dogs locked in the car than I do to my horses with EHI!

References


CVA Question 4: Finally, in dogs with heat stroke – cool IV fluids and ice water enemas are thought to be helpful for cooling the core. It’s easier to give fluids fast to a dog than to a horse, but giving some cold fluid into the rectum after sedation might be possible?

Giving a rectal enema to a horse at the racetrack might be a bit frightening to say the least – even after sedation with detomidine. Personally, I feel that the potential risks would greatly outweigh the benefits. You must remember, it is not a practice environment – sometimes it is difficult to even give intravenous fluids at the races because of the logistics and the time constraints. There is a race every 35 minutes and on a hot day we might have waves of horses that need attention, or at least supervised cooling, after every race. People actually available to help are few and at country tracks, with only one vet to do everything, the situation becomes even worse.

References
A CHRONIC VIRAL DISEASE OF GOATS

That we need to eradicate:
WHAT DISEASE DOES THIS TO GOATS?

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There is NO treatment
and NO vaccine.

It is mentioned in the National Goat Health Statement that should go with each National Vendor Declarations (NVD) and in the National Kid Rearing Plan.

But, it is not Johne’s Disease nor gastro-intestinal parasitism (although wasting is a common sign and these goats could have these conditions as well because their resistance is reduced).

FROM THIS

TO THIS, 12 MONTHS LATER

Figure 1. Prior to contracting the disease.

Figure 2a.
The disease is **CAPRINE ARTHRITIS ENCEPHALITIS (CAE)**

CAE is caused by a slow virus which is a member of the retrovirus family (like HIV-AIDS). It is from the subgroup, lentivirus, the same as the sheep disease Maedi-Visna (also called Ovine Progressive Pneumonia), which is exotic to Australia. MV/OPP and CAE are so similar that it is suggested both must be controlled at the same time for either eradication program to succeed. CAE viruses are attached to monocytes and macrophages. CAE can also be referred to as ‘big knees’, caprine retrovirus or caprine lentivirus.

Large numbers of viruses are in the inflamed brain, spinal cord, lung, joints, and mammary gland cells of CAE positive goats. Smaller numbers are in the uterus, lung, liver, spleen, lymph nodes, lining of the vessels of the brain, synovium, intestines, kidneys & thyroid.

Transmission is via bodily fluids. Milk and colostrum are the main sources of infection. Horizontal transmission between dry goats is less likely, but still possible. There is a very high risk if goats are milking or kidding. A single tiny intra-mammary dose can transmit CAE, so CAE positive goats should be milked last and milking machines well maintained. Overseas, sheep have acted as carriers.

The key message to goat producers is **NEVER feed pooled milk to goat kids** (unless it is pasteurised first). Clinical signs take one of these following forms:

- Chronic arthritis which starts as a peri-arthritis and progresses to deformed limbs and wasting
- ‘Hard Udder’ – udder feels like a smooth stone under the skin with only a small amount of milk produced
- Chronic interstitial pneumonia with wasting and difficulty breathing
- Nervous signs (generally in kids)

Clinical signs can be very slow to show up and initially they can be very mild e.g. just swollen carpus joints as shown in next photo. Signs increase with increased viral load in a herd.

Some, but not all Australian states, have CAE freedom accreditation schemes and goat herd owners should be encouraged to take part and only buy new animals from accredited herds.

**Milk and colostrum are the main sources of infection.**

Further Information


Figure 3. Illustrates why CAE is sometimes referred to as ‘Big knees’.

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**Grateful thanks to both Scott Reid and Sandra – Scott for supplying the great goat image featured in our ebook which inspired Sandra to write these articles specifically for the C&T Series.**

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**The key message to goat producers is NEVER feed pooled milk to goat kids.**

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Mark (pictured above) with one of Tasmania’s endangered Wedge-tailed Eagles

Mark is a born and bred Brisbane boy who graduated from the University of Queensland in 2000. As a self confessed meteorological refugee, he fled the balmy sub-tropics for temperate Tasmania. Three years in mixed practice in the ‘north-west’ followed by the ubiquitous 2 year sojourn in the UK, led him further south to Hobart. Mark has been practicing in Kingston, just south of Hobart, for 8 years and his interests lie mainly in small animal medicine. Exploring Tasmania’s beautiful places with his family and running provides the yin to his veterinary yang.

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‘Jax’ a 3-year-old male described Tonkinese indoor/outdoor cat presented to us in the middle of summer with acute onset of inappetance, lethargy and tachypnea. Physical examination was unremarkable except mild dehydration and high temperature (40°C). Tachypnea was not noted at examination despite the owners reporting it at home. My general approach to fever (40˚C). Tachypnea was not noted at examination despite the owners reporting it at home. My general approach to fever

Mark was going to need a thoracotomy, but before this I thought a chest CT and FIV/FeLV testing was prudent. Jax represented 8 days later for a dry cough, weight loss, inappetance and tachypnea. He improved after the medications prescribed previously but never got back to normal. Physical examination was unremarkable (temperature was normal) except tachypnoea of 60 breaths per minute with mild abdominal effort. Chest radiographs were taken and revealed a radiopaque mass in the right side of the thorax (~50x20mm), some radiofrequency caudal induration (possible pneumothorax) and some loss of contrast ventrally (see fig. 1 and 2). Image quality was not great due to a moving patient. A quick ultrasound of the right side thorax revealed no obvious pleural fluid and I could not visualise the mass. Pulse oximeter of the tongue revealed 88% Po2 saturation.

Jax was sent to the local after hours emergency centre where he was placed on oxygen, given pain relief, IV fluids, mitazapine and Clavulox/clindamycin. His oxygen saturation improved to 91% (off oxygen supplementation) during the night. Jax was anemic with a PCV of 23.1. Mark was stable, a thoracic ultrasonic was planned.

Ultrasound revealed a 20x30mm mass on the right caudal dorsal thorax. Milky air bubbles in the mass suggested a lung based mass. IDEXX FBN2 cytology revealed no microorganisms, no neoplastic cells, but reactive lymphocytes and haemosiderophages (macrophages with ingested red blood cells) indicating pathological bleeding and mixed inflammatory reaction.

Jax was going to need a thoracotomy, but before this I thought a chest CT and FIV/FeLV testing was prudent. Jax was retrovirus negative and his CT results revealed a bilateral pneumothorax, atelectasis of the right middle and caudal lung lobes with a RIGHT CAUDAL LOBAR PNEUMONIA BULLA. Enlarged pulmonary arteries with pulmonary hypertension suspected.

A thoracotomy and pneumonectomy was performed by a referral surgeon soon after. The right side middle, accessory and caudal lung lobes were removed due to consolidation/atelectasis. The cranial lobe was partially inflated (see CT images) and was left. A chest drain was placed and removed 48 hours after a good recovery.

Culture from the affected tissue grew a few colonies of Actinobacter spp. Histopathology showed pyogranulomatous pneumonia with plant material. It was unsure whether this was a grass seed or grass blade although the former seems much more likely. Jax went on appropriate antibiotics for 2 weeks. He recovered well.
from his surgery and had chest radiographs 2 weeks later (see Fig. 5 and 6) which showed recovery and PCV 3 weeks post surgery which was normal.

The right side of the thorax in carnivores has ~ 60% of the total lung tissue (a cranial, middle, caudal and accessory lobe) whereas the left side has ~ 40% (a cranially and caudally divided cranial lobe and a caudal lobe). Speaking with Richard Malik, he commented that in veterinary literature there is good evidence of cats living on 40% of functioning lung tissue. Thus the entire right side or left side can be removed and enable survival (be it with a non-athletic patient)! Also with the possibility that remaining lung may ‘expand’ into the space deficit and become more efficient at gas transfer.

My lesson learned here is to radiograph all cats with any hint of tachypnea – especially if the owner has noted it at home. The threshold of 30 breaths per minute or greater should not be seen in a healthy non-stressed cat. I would like to thank Chris Alltree, Sean Muir and Richard Malik for helping Jax and myself.

Figure 1: Left lateral radiograph prior to CT and ultrasound guided FNAB. Note radio-opaque mass (bulla) dorsally-caudally.

Figure 2: DV radiograph prior to CT and ultrasound guided FNAB. Note early pneumothorax signs (radiolucency) in the caudal right side thorax.

Figure 3: Thorax ventro-dorsal with contrast. ‘Note the radio-opaque mass (pulmonary bulla) in the right side thorax with some functional cranial lung lobe and large pneumothorax. Mini pneumothorax seen on the left side as well caudally’.

Figure 4: Thorax transverse with contrast. ‘Again the radio-opaque mass (pulmonary bulla) can be seen in the dorsal right thorax (cardiac silhouette ventral) with associated pneumothorax’.

Figure 5: Left lateral radiograph 2 weeks post pneumonectomy

Figure 6: VD radiograph 2 weeks post pneumonectomy

Elizabeth graduated with honours from the University of Sydney in 2007. She started working in a Sydney metropolitan small animal practice from 2007-2011, where she was particularly involved with treating hyperthyroidism in cats using radioactive iodine therapy. She joined the Small Animal Specialist Hospital in 2011, completing a one year rotating internship, followed by a medicine internship and then commenced her residency in small animal internal medicine in January 2013. Elizabeth has also worked with the Department of Primary Industries during the Equine Influenza outbreak and with her local government as a member of the Companion Animal Advisory Committee.

ABSTRACT

An 8-year-old male neutered Weimeraner dog presented for a focal swelling on the palmar aspect of the left thoracic limb. There was no lameness associated with the swelling and the dog was otherwise well. The swelling was immediately proximal to the accessory carpal bone on the caudal aspect of the limb, and appeared to affect the tendon of the flexor carpi ulnaris muscle (Fig. 1).

The affected area was firm on palpation, but not painful. The carpal joint had, perhaps, a slightly reduced range of motion (on flexion). The dog was sent home with instructions to enforce strict rest and to administer metacam (0.1 mg/kg orally with food) 1 for one week.

As the swelling did not improve with NSAID therapy (Fig. 2), the dog was admitted for radiographic and ultrasonographic studies. Dorsocentral and mediolateral radiographs of the carpi were obtained, and demonstrated a small focal radiopaque lesion, surrounded by soft tissue swelling, on the caudal aspect of the carpus (Fig. 3).

An ultrasound examination was performed to further characterise the cause of the radiopaque area within the soft tissue swelling (Fig. 4). There was loss of the normal pattern of linear, parallel collagen fibres within the flexor carpi ulnaris tendon, the architecture being replaced by a disorganised mixture of hyperechoic and hypoechoic densities possibly reflecting haemorrhage and/or oedema. A focal fusiform lesion (approx. 5 mm x 2 mm) was evident in the middle of the tendon (Fig. 4a and 4b – black arrows), with distal acoustic shadowing (Fig. 4a and 4b – yellow arrows). Fine needle aspiration of the swelling was performed but there was poor exfoliation and the specimen obtained was non-diagnostic.
Based on the physical findings (swelling of flexor carpi ulnaris tendon), radiographic and sonographic findings, we made a diagnosis of traumatic tendonopathy. Based on the experiences of the remarkably similar case recently reported by Sue Kuan and colleagues (2007) in the Australian Veterinary Journal, we elected to use a topical ‘roll-on’ formulation containing flumethasone (0.05mg/g) and dimethyl-sulfoxide (600mg/g) and continuation of strict rest. Within two applications, the swelling had disappeared. Topical therapy was continued for one week in total. The dog remained well for several months, although the problem recurred in the same location after a bout of vigorous exercise, this time associated with mild lameness. Again, application of the roll-on DMSO/corticoid formulation rapidly resulted in clinical improvement.

This case was presented for investigation of a soft tissue swelling affecting the flexor carpi ulnaris tendon in one limb of an aged male Weimeraner. With the benefit of hindsight, this was an easy ‘pattern recognition’ diagnosis to make, because the ‘illness script’ for flexor carpi ulnaris tendonopathy (Kuan et al 2007) is especially characteristic (Gladwell 2005, Sanders 2009). Indeed, the inclusion of Chris Tan in the list of the authors was contingent on his ability to recall this particular ‘illness script’ when we sent him a photo of the patient’s leg as an e-mail attachment! No doubt Graeme Allan would have likewise been able to diagnose the condition in the ‘blink of an eye’ when provided with a photo of the patient, a radiograph of the limb, or a sonogram of the affected tendon (radiologists can use pattern recognition, often only using shades of grey!) (Gladwell 2005).

During the initial investigation of this patient, the primary clinicians (Thrift and Malik) were not aware of this entity. Neither of us has a strong interest in orthopaedic condition of dogs, and although we likely cited Dr Kuan’s paper in the AVJ, it didn’t ‘stick’ in our brains. Stated another way – we didn’t have an accessible ‘illness script’ for this somewhat obscure musculoskeletal condition. So instead, we initially had to rely on a problem-orientated approach using ‘hypothetical-deductive reasoning’. Our differential diagnosis for the firm swelling was a degenerative or traumatic disorder (e.g. tendonopathy, hematoma), focal abscess, foreign body granuloma, neoplasm (soft tissue sarcoma, mast cell tumour, etc). To be honest, tendonopathy wasn’t really on our list! Indeed, once we detected the mineralized density in the centre of the anatomic region affected, our principal reason for using sonography was to make sure there was not a foreign body granuloma present, or a localised abscess around a radio-opaque foreign body.

It was just as well we sought expert advice about this case following the initial ‘work-up’, because after palpation, radiology, sonography and needle aspiration, further avenues of investigation might have proved to be extremely expensive (helical computed tomography, magnetic resonance imaging, scintigraphy) or invasive (exploration and biopsy).

Truthfully, had the primary clinicians been aware of this condition when the dog was first examined, it is quite likely that radiography, ultrasonography and fine needle aspirate biopsy might have been circumvented, and instead the patient may have been given a trial of the topical DMSO/corticoid preparation. This would have saved the owner a great deal of money. The lesson to be learnt here is that quick consultation with an expert early in the work-up can improve the efficiency and speed of the diagnostic process. The value of consulting an expert clinician when confronted by a case a bit out of the ordinary is therefore emphasised. This might involve a phone call, or an e-mail, rather than a formal referral consultation.

Flexor carpi tendonopathy has been well documented in racing Greyhounds, although remarkably, most of the information is in conference proceedings and textbook chapters, rather than in peer-reviewed journal articles. This is a limitation of the use of evidence-based medicine (EBM) in veterinary medicine – much of the best information for certain conditions appears in sources at the bottom of the EBM pyramid! Presumably the tendonopathy develops as a consequence of the increased stress placed on this tendon with intense repetitive activity (Denny, 2004). The recent report of this tendonopathy in a Weimeraner, (Kuan et al. 2007), suggests that non-racing dogs subjected to repetitive percussive trauma during normal but vigorous exercise can also develop this condition.

This case differs from that of Kuan et al (2007) in a number of respects. In our patient, the swelling did not result in significant lameness. In relation to therapy, a padded foreglobe splint was not applied in this case to immobilize the limb as a preliminary step in therapy. Denny (2004) suggests that complete immobilisation in the four to six week period after tendon damage is vital for tendon healing as movement significantly impacts on the development of fibroplasia and fibrous union. However, in the case documented by Kuan et al (2007), lameness returned immediately after the splint was removed, but there was subsequently a remarkably prompt and complete response to topical DMSO/corticoid roll-on. Our patient likewise improved astonishingly well after topical therapy, suggesting immobilisation is not necessary, provided the dog has restricted exercise. The rationale of using the DMSO/flumethasone roll-on formulation, is that the DMSO carries the corticoid rapidly through the skin and into the soft tissues in and around the damaged tendon.

It is interesting that in both this case report and the report by Kuan et al (2007), the two respective patients were members of racing Greyhounds and suggests that this condition may be more common in practice than the current peer-reviewed literature would indicate. In an attempt to gather more information on the prevalence of this problem in this and other breeds, a questionnaire is presented at the end of this article.

2The concept of mineralized tendonopathy did not occur to us until we had re-read Sue Kuan’s paper after email correspondence with Chris Tan, although we were aware of the calcifying tendonopathy affecting the biceps muscle tendon of large-breed dogs.
SURVEY

We are interested in determining the commonness of flexor carpi ulnaris tendonopathy in non-Greyhound breeds. Please complete each question below if you have observed a dog present with signs consistent with Flexor Carpi Ulnaris tendonopathy (FCUT).

Veterinarian’s name, Clinic and phone number or e-mail address:

The breed of dog(s) affected:

Was the left front leg, right front leg, or BOTH front legs affected?

L / R / both (circle)

Was diagnostic imaging (radiography/ultrasonography/CT/MR) used to confirm or support your diagnosis?

Yes / No (circle)

If yes, please discuss the method used and briefly describe your findings:

Was surgery or needle aspirates performed to collect a biopsy specimen?

Yes / No (circle)

If yes, please indicate the findings:

Please discuss any treatments (including strict rest, splints or medication) and whether or not they achieved clinical resolution:

Please Fax to 02 9363 1238 or email to elizabethralph85@gmail.com

References


Acknowledgements

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Authors’ views are not necessarily those of the CVE

CVE Control & Therapy Series – Issue 274 March 2014

FELINE TIPS

C&T NO. 5381

Dr Kim Kendall Cat Vet

I SPEND MY LIFE TRYING NOT TO PUT E-COLLARS ON CATS

(I ONLY DO IT EVERY 10 YEARS OR SO…)

So, the Air Muzzle is for grooming cats/ restraining them (takes the fear out of handling them and everyone calms down). It’s easy to slip on the cat’s head even when it is being fussy. At least the first time – haven’t had a chance to retry it on the same cat!

The other forms of Elizabethan-collar (E-collar) are to try to reduce the stress inherent in stopping a cat from licking itself. Licking releases endorphins in a cat, and some research at the Waltham Centre showed that just putting an E-collar on a cat was enough to induce stress-related pruritis. E-collars are NEVER the answer for itching cats, and only a very temporary answer for surgery wound protection.

DUAL USE AIRMUZZLE/ OXYGEN CAGE

The airmuzzle is pretty self-explanatory once you have one in your hands and has an adaptor that turns it into an oxygen mask.

The adaptor bit should be removed when using is as a muzzle, as the cats are easier to monitor if you can see them.

The key is – take the front ‘oxygen attachment’ section out, so it is a see-through system. Open the back section wide (it goes very wide) and make sure the velcro is attached only on the purple section – so you can connect the back section together quickly. Obviously you have to stop the cat going backwards, but then most cats will be so busy looking at the contraption that it is pretty easy to slip over their heads (back section first). Quickly velcro to neck size (small). There’s no chance of choking the cat. Then everyone has only to watch for claws. Wrap cat if needed.

The unit is very light, easy to clean and does not jam, even when you are in a bit of a rush. Try it on a nice, compliant cat first. Because they can breathe, see, and make noises, most cats will be so busy looking at the contraption that it is pretty easy to slip over their heads (back section first). Quickly velcro to neck size (small). There’s no chance of choking the cat. Then everyone has only to watch for claws. Wrap cat if needed.

The unit is very light, easy to clean and does not jam, even when you are in a bit of a rush. Try it on a nice, compliant cat first. Because they can breathe, see, and make noises, most cats give up the fight.

Saves fingers, time and deep bites!

I bought mine direct from USA (about $130 landed) and there are cheaper ‘home use’ versions. (Unfortunately I don’t think they’re available here in Australia yet.) It is designed to be turned into an ‘oxygen cage’ – the 1cm hole in the front would have some endotracheal tube fitting that would work, along with tape!

Authors’ views are not necessarily those of the CVE

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AND SURGICAL WRAPS AND COATS

The surgical wrap and little coat were mainly because the dermatologists have identified that atopic itching is reduced by removing skin contact with allergens (airborne especially). It is surprising what a cat will tolerate wearing. The coat needs to come off intermittently to allow the cat to groom itself (and obviously the cat should be well-washed before putting it on!), but they will just lick the coat rather than pull it off if they are comfortable.

The Siamese in the first pictures, of course, defied all the rules – pulled out stitches AND staples out of the surgical wound along his thorax (lipoma removal – should not have bothered) surgical wound along his thorax (lipoma removal – should not have bothered)

And then chewed out the staples! I do a lot of stapling – quick and easy and no more painful than local. So we had to resort to this – the white body wrap is a surgical wrap (stretchy bodysuit with armholes, velcro to hold it around, and I used Comfort wrap on top of it. And a ‘puff collar’ – blow air into a little plastic tyre tube inside the blue outer to make it fit. I think the collar attachment is superfluous, but strapped it on anyway!

So far so good!

Given the drive to create cat-friendly and dog-friendly clinic items, plus the law in some states requiring the use of muzzles in our patients, I wondered if anyone knew of or thought whether more ‘human visually friendly’ dog and cat muzzles were useful/available or could be designed? I ask because of a fairly active discussion on a vet global e-list I am on where even some vets struggle with the concept of using a muzzle and to my horror and sadness, seem to accept themselves and their staff being bitten as a badge of honour…

As a way forward, I was intrigued when 2 good colleagues revealed they ask for a ‘nose warmer’ or ‘party hat’ when asking the nurse to get a muzzle – a clever change of concept. I tried the ‘nose warmer’ term on a few clients and it went down well.

To develop that, perhaps the makers of say, the Mikki leather and the plastic cloth type muzzles could consider making muzzles other than in black – perhaps bright pink and calming green, or some cute clouds and field scenes (no pics of other animals with big eyes staring back up at the dog). This would really help de-stress the owner and, in changing the visual, change the mood in the room. And make the consultation overall safer and less stressful for pet, vet and owner.

I warn the owner that the cat looks like Frankenstein temporarily but will be fine. I soon figures out it can’t bite, but you can still see the cat’s face. I rarely use anything – and the ‘burrito wrap’ as Dr Sophia Yin calls it is a defacto muzzle: www.drsophiayin.com/sh

The ‘air muzzle’ is quite useful for cats… or anything?

“i didn’t know it was compulsory to muzzle dogs…” or anything?

Sophia Yin calls it is a defacto muzzle: www.drsophiayin.com/sh

I didn’t know it was compulsory to muzzle dogs… or anything?

For a ‘nose warmer’ or ‘party hat’ when asking the nurse to get a muzzle – a clever change of concept. I tried the ‘nose warmer’ term on a few clients and it went down well.

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EYELID STAPLING FOR CORNEAL LESIONS

I use local anaesthetic into the eye, debride with cotton bud, pack with Orbenin (CATTLE) Eye Ointment, then staple close. After checking 3 days later, I decided that this cat needed restapling.

I have done several now and the cats are remarkable unreactive (even Burmese leave their eyelids alone).

There is no need for an e-collar – just pinch up the eyelids between your fingers, staple with a 35W staple and it went down well.

To develop that, perhaps the makers of say, the Mikki leather and the plastic cloth type muzzles could consider making muzzles other than in black – perhaps bright pink and calming green, or some cute clouds and field scenes (no pics of other animals with big eyes staring back up at the dog). This would really help de-stress the owner and, in changing the visual, change the mood in the room. And make the consultation overall safer and less stressful for pet, vet and owner.

I warn the owner that the cat looks like Frankenstein temporarily but will be fine.
A 3-year-old, female spayed Bengal cat had gone missing for about a week before her owners found her back home with her right forelimb caught in the flea collar. The owners noticed a large wound under the cat’s right forelimb before her owners found her back home due to disuse was considered as the cat had her right forelimb caught in the flea collar for up to a week and was unable to move it. It is also possible that an inflammation of the tendons or musculature around the elbow joint could have resulted in some stiffness as well.

Clinical Presentation and History

A large laceration wound on right axilla was large, exudative and had a putrid odour. There was a significant amount of granulation tissue and the wound looked like it was healing. On presentation looked like a classic case of localised tetanus. In this case, the main differential diagnoses considered for lameness or a neuromuscular junction cause. To an orthopedic, muscular, neurological diagnoses would be to ascertain if there was no paw positioning reflex but a neuromuscular junction cause. To an orthopedic, muscular, neurological diagnoses would be to ascertain if there was no paw positioning reflex but a neuromuscular junction cause.

Initial Physical Examination Findings: 8 May 2013

On presentation, the cat was agitated and slightly aggressive, making a thorough examination difficult. Based on distance examination, she was bright, alert, responsive and still had a good body condition score (4/9). Her right forelimb was caught in her flea collar at the level of her axilla, resulting in a laceration. The plan was to anesthetise the cat to dislodge the flea collar, assess and manage the wound and conduct a thorough physical examination.

The right forelimb was monoparetic, the right elbow was non-weight bearing on full extension and carpal flexion after a traumatic injury (Fig. 1.3) is a poster child for localised tetanus, although it can occur in the hindlimbs or tail as well. Thus, the diagnosis of localised tetanus was made in this case based on the strongly suggestive history and presenting signs.

Tetanus is caused by a neurotoxin from the anaerobic spore-forming bacteria, Clostridium tetani. It occurs due to localisation of tetanus spores from the environment into a wound and under anaerobic conditions often created by necrotic tissue, converts to a toxin-producing form (Riso & Gétai 2003). The toxin, tetanosporin, binds to presynaptic site of inhibitory neurons, blocks the release of inhibitory neurotransmitters and results in constant activation of motor units, appearing as localised or generalised muscle spasms (Riso & Gétai 2003; Langner et al. 2011). Cats have an innate resistance to this toxin, related to the difficulty in tetanosporin penetrating and binding to nervous tissue so it does not occur commonly and when it does, its effects

Localised Tetanus in a Bengal Cat

C&T No. 5382
Joyce Lauw
BVSc (Year V, University of Sydney)
Chris McClelland
BVSc (Hons)

The cat weighed 3.6kg and was sedated with a combination of 0.3mg/kg methadone, 0.025mg/kg atropine and 0.05mg/kg acepromazine (0.6mg/kg MAA) subcutaneously and induced with 5.5mg/kg (2mL) alfaxan intravenously. She was intubated with a 3.5mm endotracheal tube and maintained on inhalational isoflurane/oxygen anaesthetic.

The right forelimb was caught in her flea collar and it could be extended easily. The cat recovered well from anaesthesia but seemed to be knuckling on her right carpus although further investigation was required to determine if it was due to proprioceptive deficit or a musculoskeletal problem. The right forelimb was monoparetic, there was no paw positioning reflex but there was a deep pain response was present. All other limbs were normal.

Problem List

- Temporary joint stiffness of the elbow
- Large laceration wound on right axilla
- Mononeuropathy of right forelimb
- Stiff extended right elbow
- Flexed right carpus

Differential Diagnosis and Assessment

The right forelimb was caught in her flea collar when the limb got caught in it. However, this does not help to explain the stiffness in the right elbow and flexion of the right carpus. The first step to formulating and narrowing down the differential diagnoses would be to ascertain if lameness of the right forelimb was due to an orthopedic, muscular, neurological or a neuromuscular junction cause.

Radial nerve injury
Fractured or dislocated shoulder
Other localised nerve, muscle, ligament or tendon damage
Localised tetanus

Temporary joint stiffness of the elbow due to disuse was considered as the cat had her right forelimb caught in the flea collar for up to a week and was unable to move it. It is also possible that an inflammation of the tendons or musculature around the elbow joint could have resulted in some stiffness as well.

Brachialplexus avulsion was a concern but the wound did not seem extensive enough to have caused injury to the brachialplexus. That would usually cause a flaccid paralysis and loss of sensation of the affected limb instead of rigidity (Garosi 2012). Radial nerve injury was also considered but it typically presents with both the elbow and carpus locked in flexion (Garosi 2012). Although no obvious fractures or dislocations could be palpated, radiographs are required to rule out any orthopedic injuries.

Diagnosing localised tetanus in cats can be quite the challenge if one has not seen a case or heard of it before as it is a relatively uncommon disease with unusual presenting signs. However, this syndrome has hallmark presenting signs, making them almost pathognomonic so we hope that this short report would enable other veterinarians to easily recognise these signs and make a quicker diagnosis in future cases. As we were unfamiliar with this syndrome and the presenting signs were not that pronounced initially, we considered and tried to rule out the above differential diagnoses first. A conclusive diagnosis was not made by this point of time and it was decided to treat the cat conservatively and reassess in a few days.

Initial Conservative Treatment: 8 May 2013

The cat was treated with 1.1mg/kg (0.3mL) oral Metacam®/Meloxicam and strict cage rest as part of conservative treatment and was due for a revisit in 2 to 3 days time for repeat wound cleaning and reassessment under sedation.

Review and Re-assessment: 11 May 2013

The cat was represented for reassessment and wound cleaning under sedation. The cat had been hiding a lot at home, but had been eating, drinking, urinating and defecating normally. The cat was quiet, alert and responsive but the extremity rigidity in her right forelimb had gotten worse and was non-weight bearing on the affected limb.

She was sedated with 0.18mg, Domitor®/Medetomidine and 0.15mg methadone combined intramuscularly. Radiographs were taken of the elbow joint under sedation to rule out any skeletal problems like fractures, dislocations or osteoarthritis (Fig. 1.1-1.2). The radiographs were normal on both lateral and cranio-caudal views. Fig. 1.1 also shows the extent of the soft tissue wound in the area of the axilla. There was some discharge around the axilla wound edges, which was cleaned with chlorhexidine solution. Otherwise, the wound seemed to be granulating well. The right forelimb was now worse than before, with the elbow locked in full extension and the carpus remaining in flexion. There was limited movement in the shoulder and severe extensor rigidity in the elbow, which could not be manipulated or flexed at all (Fig. 1.3).

Interpretation and Diagnosis: Localised Tetanus

After worsening of the extensor rigidity and ruling out other orthopedic involvement, the cat’s current presentation looked like a classic case of localised tetanus. A cat that develops extreme localised forelimb rigidity, elbow extension and carpal flexion after a traumatic injury (Fig. 1.3) is a poster child for localised tetanus, although it can occur in the hindlimbs or tail as well.
are localised (Riso & Gelati 2003; Langner et al. 2011). Binding of the toxin to presynaptic sites is irreversible and recovery is slow as it depends on sprouting of new axonal terminals (Riso & Gelati 2003).

Potential Diagnostic Tests

To obtain a definitive diagnosis, collection and isolation of Clostridium tetani can be done from the site of infection but this is often challenging as anaerobic bacteria can be difficult to culture and a negative result does not rule out this diagnosis (Baral et al. 2002). Electromyography (EMG) can be helpful in investigating peripheral nerve injury or neurological involvement in cases of occult lameness where an orthopedic cause cannot be identified and can also be helpful in diagnosing tetanus (Baral et al. 2002; Garosi 2012). An EMG would demonstrate a characteristic persistent motor unit activity in cases of tetanus and serum tetanus antibody titres may also be useful but these options are not widely available (Baral et al. 2002).

Actual Treatment Plan

As C. tetani is an anaerobic bacterium, a suitable antibiotic must have a good anaerobic spectrum of activity. Metronidazole is bacteraicidal against most anaerobes including C. tetani and can attain effective therapeutic concentrations even in anaerobic tissues (Greene 1998). Metronidazole has also been shown to have superior effects compared to penicillin G and tetracycline in clinical and experimental tetanus cases (Baral et al. 2000). Metronidazole is often used with a broad spectrum antibiotic (eg. amoxicillin clavulinate) to improve anaerobic spectrum and provide additional cover for any other bacteria present.

14mg/kg (60mg) Metronidazole tablets and 14mg/kg (60mg) Amoxicillin-Clavulinate tablets were prescribed twice daily for 2 weeks. The owners were also instructed to continue with wound management at home, cleaning the wound with chlorhexidine solution and keeping it dry. The cat was to continue with 0.125mg/kg (0.3ml) oral Metacam®/meloxicam for pain relief and anti-inflammatory effects for 2 more weeks before coming back for a recheck and reassessment.

Potential Treatment Options

Other potential treatment options include tetanus antitoxin and muscle relaxants such as diazepam or midazolam. Tetanus antitoxin only works to neutralise any circulating toxins, it cannot dislocate the toxin that has already been bound and thus it is unlikely to be useful if signs are localised and not progressing (Baral et al. 2002). Administering the antitoxin does have its risks and may trigger an anaphylactic reaction (Riso & Gelati 2003). Benzodiazepines may help reduce discomfort from the muscle rigidity throughout the long recovery period but their effects have been inconsistent and variable in cats with localised tetanus (Baral et al. 2000).

Outcome

The cat’s wound granulated and healed completely within 3 weeks. Her owners kept her under strict confinement and monitored her progress. She completed her 3 week course of antibiotics and was not given any more as she was becoming a challenge to medicate orally at home. Her limb remained tetanic until 7 weeks post-injury, when her owners noticed that it was starting to free up. The cat continued to improve rapidly over the next 2 weeks and eventually regained full function of her affected limb approximately 9 - 10 weeks post-injury.

Conclusion

Localised tetanus in cats is a relatively uncommon disease with peculiar presenting signs, which makes diagnosis difficult. The purpose of this case report is to help readers recognise this syndrome, enable early diagnosis and administration of prompt treatment in similar cases seen in practice in the future. This is important as localised tetanus can actually progress to generalised tetanus if left untreated (Baral et al. 2002). With appropriate treatment, the prognosis for cats with this disease is very good although it may take up to 2 - 4 months for complete resolution (Baral et al. 2002; Riso & Gelati 2003).

References

Langner, KFA, Schenk, HC, Leithauser, C, Thaler, M & Simon, D 2011, Short Communications: Localised tetanus in a cat, Veterinary Record, Vol 169, no. 5, pp. 126

VENLAFAXINE (EFFEXOR) INGESTED BY A CAT: A FEEL-GOOD STORY

CAT NO. 5383

Dr Anne Fawcett

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PO Box 300
Cooma, NSW 2630

References

Venlafaxine is a popular human anti- depressant medication marketed as Effexor. It is also indicated for treatment of generalised anxiety disorder, social anxiety disorder and panic disorder in humans. I had no idea it was toxic to cats (or that cats would seek these pills out) until we had a case recently. This case is very mild, but I thought it worth writing about as there really isn’t much literature on this subject and it is possible that Australian vets are seeing these cases or will come across them in the near future.

A few years ago venlafaxine made it to number 3 on the ASPCA’s top 10 toxins of cats (permethrin insecticides sit pretty at number 1, but other insecticides, glastil, oximes, anticoagulant rodenticides and amphetaamines follow).

In this case I had a Sunday morning phone call from an owner whose cat ‘Elle’, had eaten her Effexor tablet. The owner had set out the tablet with a fish oil capsule and the room to make breakfast and returned to find the tablet missing and tooth marks in the fish oil capsule.

I had not heard of this agent before but a quick internet search revealed it is toxic to cats so we recommended bringing the cat down immediately. On admission the 2-10 mg/kg dose results in excellent bioavailability (RCS 3/5, 5kg) appeared normal albeit with bilateral mydriasis. On physical examination the cat had a very quiet demeanour, HR 220, pinks mucous membranes with normal capillary refill (<1 second), T 38.4°C. There were very mild muscle fasciculations in the flanks and over the hindlimbs. The cat was not on other medications and had enjoyed good health. The mydriasis, muscle fasciculations and tachycardia were all thought to be due to the venlafaxine (although the stress of being caught and transported to the vet may have played a role in these signs).

A call to the NSW Poisons Information Centre found that in humans, venlafaxine overdose is associated with delayed seizures (up to 16 hours post dosage) and cardiorespiratory depression.

Bloods were drawn for a baseline biochemistry panel and electrolytes, all of which were within normal ranges. An IV catheter was placed and Hartmann’s administered at 10ml/kg/hr.

An attempt was made to induce vomiting by giving xylazine (at this stage, probably 30 minutes following ingestion) IM to no avail (why does it never happen when you want it to?). In fact, this produced sedation so a reversal agent, atipamezole was administered. The muscle fasciculations disappeared at this point.

When the cat was sitting up we decided to administer charcoal 1g/kg PO to mop up any drug left in the stomach.

A search on Web of Science revealed no peer reviewed case reports or case series on this toxin in cats. A search on VIN revealed some discussion threads and links to general articles available online, although no reports of any fatalities associated with this drug in cats that I could locate.

I learned that venlafaxine is a bicyclic antidepressant which causes a huge increase in CNS serotonin and noradrenaline levels and a small increase in dopamine levels. In the US it is available in tablets and capsules at doses of 25, 37.5, 50, 75, 100 and 150mg.

It is available in 2 formulations: immediate and slow release. Onset of signs is usually within 8 hours of ingestion but the slow or extended release formulation may be associated with onset of clinical signs up to 72 hours following ingestion.

It is well absorbed orally and highly protein bound. All sources I could locate online commented on how appealing the capsules seem to be to cats though didn’t explain why.

Activated charcoal is effective, but dosing should be repeated at 4-6 hours if the sustained release formulation was ingested, as it was in this case.

According to US Toxicologist and VIN consultant Tina Werner, mild depression can be seen with ingestion of as little as 1mg/kg PO, with other signs including mydriasis, vomiting, tachycardia, ataxia, agitation, tremors and seizures being potentially observed.

The recommended treatment is similar to treatment for overdose of selective serotonin reuptake inhibitors (SSRIs), notably:

• Emesis or gastric lavage in asymptomatic animals
• Blood pressure monitoring and IV fluid administration (fluids don’t seem to increase excretion but they support BP and maintain renal function)
• Acamprosate for agitation
• Methocarbamol for muscle fasciculations or tremors

Authors’ views are not necessarily those of the CVE
• Midazolam or barbiturates or Kepax® for seizures

• Cyproheptadine can be used to treat agitation which may be a sign of serotonin syndrome, dose is 2-4mg PO per cat; while there is no evidence, multiple sources recommend crushing the tablet in saline and administering per rectum if PO administration is not possible due to erosion or seizing.

After I posted about the case on VIN, Dr Wismer advised that if the cat is simply sedated and atactic, IV fluids are the most appropriate treatment. Gastric lavage is not needed. Dr Wismer was not aware of case fatalities in cats exposed to this dose.

The call in this case maintained normal blood pressure on IVF and improved overnight with mydriasis resolving and blood pressure on IVF and improved.

Gastric lavage is not needed. Dr Wismer advised that if the cat is viscous from sedation or atactic, IV fluids are the most appropriate treatment.

Two unrelated Persian kittens were presented in March 24th and April 30th, 2013 for routine vaccinations. Both kittens were owned by 2 different breeders in Victoria who had never shared breeding lines.

The kittens presented with dorsal cranial ridging affecting the frontal bones as a symmetrical deformation. The clinical examinations of both kittens were otherwise normal, with no visible cranial nerve deficits, or cognitive or behavioural dysfunction.

Kitten 1 had been raised on a mixture of Whiskas dry kitten food and home-made meat recipe, and the other exclusively on Royal Canin Kitten dry food. No evidence existed of in utero exposure to any drugs during the pregnancies.

Radiographic and CT analysis2 of one kitten revealed premature lateral suture closure with dorsal deviation of the skull, similar clinically to craniosynostosis in humans.

In humans, there are a variety of syndromes which underlie the pathogenesis of craniosynostosis, such as Saethre-Chotzen syndrome, Apert Syndrome or Pfeiffer Syndrome, and all human cases have a genetic basis (transcription factor TWIST and fibroblast growth factor receptor genes FGFR1, 2, 3) underlying the clinical presentation which includes craniofacial defects, open fontanelles, Chiari malformations, visual impairments and cerebral hypertension6. Human craniosynostosis can also arise as a congenital defect due to in utero exposure to mutagens such as cyclophosphamide, nitrosatable drugs or processed meats7. Surgical intervention via craniotomy is usually curative of the cranial defect in human patients.

No feline specific DNA tests are available for screening of patients with Craniosynostosis: Molecular Strategy. American Journal of Medical Genetics 120A:470-473


While it is rare for cats to willingly ingest medications, cats seem to readily eat ventraline. It is not uncommon for the ASPCA Animal Poison Control Center to receive phone calls about cats that have eaten several capsules. Cats can become symptomatic at low doses but usually respond very well to therapy. Length of treatment will vary depending on when the animal presents (can you decontaminate?) and the formulation of the medication (immediate vs extended release). Treatment time can be 24-72 hours. Ventraline binds well to activated charcoal and charcoal administration can stop any signs from occurring. Due to the costs involved with a long treatment time, there has been some interest in using intravenous lipid therapy to decrease treatment time. Ventraline is lipidsoluble and lipids have been used for treatment of overdoses in human medicine. As an aside, ventraline will give a false positive for PCP on many over-the-counter urine drug screens.

Figure 1. The pill packet brought in by the owner.

Figure 2. The patient, Elke, with mydriasis.

Figure 3. Elke 24 hours later.

Figure 4. The pill packet brought in by the owner.

Figure 5. The pill packet brought in by the owner.

Figure 6. The pill packet brought in by the owner.

Figure 7. The pill packet brought in by the owner.

Figure 8. The pill packet brought in by the owner.

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Figure 43. The pill packet brought in by the owner.

Figure 44. The pill packet brought in by the owner.
Emesis
Apart from the described (Perspective 101, – Rhian Cope & Rosalind Dalefield) potential complications of inducing emesis, contra-indications to getting the patient to vomit with Metaldehyde poisoning include its inherent gastric irritability and the potential for aspiration in patients who may develop a depressed level of consciousness or seizures. Perhaps an argument could be made to induce emesis in a peracute ingestion (<30-60 minutes) of a large volume of Metaldehyde in a clinically normal dog under controlled conditions (veterinary clinic) where rapid airway access and control can be ensured if needed.

Gastric Lavage

Despite the eloquently detailed short-comings and potential complications of gastric lavage detailed in Perspective 101, the above massive ingestion of a highly toxic substance (say 7.5 gram) as Metaldehyde which is absorbed over all the GIT, and which is enterohepatically recycled warrants strong consideration for gastric lavage and at least single dose activated charcoal treatment (SDAC). In fact, this is a situation where I feel careful enterogastroic lavage (lavage of the whole GIT) is indicated, especially as these patients are often presented with clinical signs severe enough to necessitate anaesthesia and endotracheal intubation anyway. Multiple dose activated charcoal (MDAC) if the patient remains adequately airway protected has a good chance of enhancing elimination of Metaldehyde, in my view, due to its long half-life and its enterohepatic recycling.

I like Dr Ava Firth’s comments in VECC C&T no. 5386 (Issue 273, Dec 2013) and the scientific debate which the recent publication of the Perspective No. 103, – Rhian Cope & Rosalind Dalefield) has engendered. I am of the opinion that enterogastric lavage (lavage of the GIT) should be used in conjunction with activated charcoal treatment (SDAC), but I do feel that the use of the latter must be carefully considered.

Further comment from Terry King on Perspective 101 (Issue 273, Dec 2013)

A Toxicologist’s Perspective 101 by Drs Rhian Cope & Rosalind Dalefield (AC) is the most comprehensive and logical review and commentary I’ve read on SDAC, MDAC, Cathartics & Enemas; Gastric Lavage, Emesis, and WBIL. As with most of the Control & Therapy Series, I enjoyed reading it immensely, albeit squirming often at the thought of how wrong I’ve been in my treatment of many poisons over the years. I applaud researchers and scientists working towards providing us with evidence-based medical facts, and am sorry that I won’t be around long enough to see all these questions answered. The authors of Perspective 101 will have to forgive us practitioners for being slow to get up-to-date with the science (P. 60. ‘The reaction from the NV veterinary profession’) – we are still trying to get our heads around why a healthy dog starved for 12 hours for an elective surgical procedure can vomit up a whole load of gastric contents when the evidence tells us that the canine stomach empties within 2-4 hours!

Cardiac troponins are biomarkers. Biomarkers are naturally occurring molecules, genes or characteristics by which a process, disease etc. can be diagnosed and/or monitored. Cardiac troponins consist of three proteins known as Tn-C, Tn-I and Tn-T. They form a complex that interacts with tropomyosin in the contractile apparatus of cardiac muscle and are released after myocardial damage. In human medicine, troponins are used as markers in patients suspected of having a myocardial infarction due to coronary artery thrombosis, the most commonly occurring human cardiac disease. Troponins are also increased in small animals with myocardial injury so troponins are potentially useful in the diagnosis of cardiac disease. Troponin I can be used in conjunction with ECG after trauma to help identify animals with myocardial contusions that are at risk of arrhythmias.

Cardiac troponins are sensitive and specific indicators of myocardial injury; however, they do not tell us anything about the mechanism of that injury. In humans, elevated troponin levels are seen in a number of non-cardiac diseases including chronic renal failure (due to decreased clearance), cerebrovascular accidents (myocardial injury secondary to massive catecholamine release either locally in the myocardium or systemically), pulmonary embolism (cardiac shock?), chronic obstructive pulmonary disease (pulmonary hypertension), hypoxia, increased right ventricular afterload and acute critical illness (cysteines). Stewart exercise and direct myocardial trauma will also increase troponin levels in the absence of cardiac disease.

Cardiac biomarkers other than troponins are also being investigated in small animals. ProBNP is the precursor of the Brain-type natriuretic peptide (BNP), a natriuretic hormone released in response to neuroendocrine activation, hypoxia and stretch of heart muscle. The function of BNP is to balance the actions of the renin-angiotensin-aldosterone system (RAAS) promoting natriuresis and vasodilation. When proBNP is released, it is cleaved into an active BNP portion and inactive NT-proBNP portion. BNP has a short half-life in circulation, while the NT-proBNP fragment is stable and therefore easier to assay. N-terminal pro-atrial natriuretic peptide (NT-proAPN) is the precursor of ANP. Higher atrial pressures stimulate the production and release of ANP. Its role is similar to that of BNP viz. to promote natriuresis and diuresis. It is used in humans to predict disease severity and mortality, to monitor response to treatment and identify patients with asymptomatic cardiac disease.

Potential utility of cardiac biomarkers in veterinary medicine:
1. Distinguishing cardiac from non-cardiac causes of respiratory disease
2. Detection of occult cardiac disease
3. Staging of cardiac disease
4. Assessing response to therapy for cardiac disease
5. Prognosis in cardiac disease

IDEXX laboratories offer testing for levels of NT-proBNP. The IDEXX website recommends the Cardiopet® proBNP Test for all dogs presenting with murmurs and additional clinical signs that may or may not be attributable to cardiac disease, and to screen for occult hypertrophic cardiomyopathy in cats with or without clinical signs. Vetrosciences Laboratory offers TN-1 testing. No diagnostic guidelines are available with this test at the time of writing.
What is the evidence? Most papers on this subject present their findings in terms of sensitivity and specificity. A highly sensitive test is one that picks up all possible positives, though may also pick up a few negatives on the way i.e. it has some false positives. With 100% sensitivity you can be sure that all animals with the condition being screened for will be picked up. A highly specific test will only pick up those animals that are truly positive. The downside is that some positive animals may not be detected. With 100% specificity, you can be sure that all positive animals definitely have the condition being screened for. Another way results are presented, and one that is perhaps more intuitive, is to describe test cut-off values in terms of positive predictive values (PPV) or negative predictive values (NPV). If a test has a 95% PPV at a specified cut-off level, any animal over this level has a 96% chance of actually having the condition being screened for. Alternatively, a cut-off of 0.2 ng/mL is considered abnormal in an otherwise healthy cat although 3 of the 20 cats with HCM had values below this level. Cats with CHF also had significantly higher Tn-I levels than those that had never had CHF or whose clinical signs were well controlled at the time of testing.

Will cardiac troponins help us detect occult cardiac disease? A possible alternative: detecting occult cardiac disease in cats and dogs may allow earlier treatment, delaying the development of clinical signs and death in some patients. A 2004 study attempted to establish a reference intervals for Tn-I in normal dogs and those with cardiac disease. Healthy dogs had a median Tn-I of 0.03 ng/mL (range 0.01 - 0.15 ng/mL), which was significantly lower than dogs with previously diagnosed cardiomyopathy (median 14 ng/mL; range 0.03 – 1.88 ng/mL), mitral valve disease (median 11 ng/mL; range 0.01 – 9.53 ng/mL) and subaortic stenosis (median 0.08 ng/mL; range 0.01 – 0.94 ng/mL). Again, there is significant overlap in the groups meaning that Tn-I is not a stand-alone test for the diagnosis of cardiac disease although very high levels are suggestive of cardiac disease. A study of 90 dogs and 14 cats found that Tn-I is elevated in dogs and cats with azotaemic renal failure and with other non-cardiac diseases. Tn-I is cleared from the body via renal excretion but increased levels were not correlated with and could not be corrected for the severity of azotaemia. This study casts doubt on the utility of Tn-I to diagnose cardiac disease in patients with co-morbidities.

Twenty cats with hypertrophic cardiomyopathy had higher Tn-I levels than 23 clinically healthy cats. A cut-off value of > 0.157 ng/mL was established as abnormal in an otherwise healthy cat although 3 of the 20 cats with HCM had values below this level. Cats with CHF also had significantly higher Tn-I levels than those that had never had CHF or whose clinical signs were well controlled at the time of testing.

Will cardiac troponins help us stage cardiac disease? The use of Tn-I to assess severity of disease and prognosis in 120 dogs with a heart murmur or an enlarged cardiac silhouette on thoracic radiography was investigated. Dogs were divided into 3 groups based on initial Tn-I levels. Dogs in the group with Normal Tn-I levels (< 0.15 ng/mL) had no or stable cardiac disease, were younger and had a significantly increased survival time. In the groups with elevated Tn-I levels, no significant difference between levels in dogs that died and those that survived was found so a single sample did not seem sufficient to provide prognostic information.
Will cardiac troponins help us assess response to therapy for cardiac disease?

Currently, there are limited ways to assess response to therapy other than the owner’s assessment of quality of life. While this is certainly important, an objective assessment of improvement that allows us to give an accurate prognosis is needed. In the above study, dogs with above normal Tn-I levels that decreased significantly within 2 months of initiating therapy had increased survival times compared to those whose levels did not decrease in response to treatment.

Will cardiac troponins help us predict a prognosis in animals with cardiac disease?

Mitrval valve disease is the most commonly diagnosed cardiac disease in dogs. It has a variable rate of progression and some dogs with a murmur never go on to develop heart failure. A method of differentiating dogs at risk of cardiac disease from those who were likely to remain asymptomatic would be beneficial to owners and veterinarians alike.

A study that looked at whether levels of NT-proBNP would predict mortality in dogs with mitral valve disease found that both NT-proBNP and left ventricular end-diastolic diameter are independent predictors of survival.12 Dogs with NT-proBNP > 78.5 pmol/L had the greatest chance of dying of cardiac disease in the year following diagnosis, and for every 100 pmol increase in NT-proBNP the hazard of all causes of mortality increased 1.07 times.

In another paper, dogs with mitral valve disease and Tn-1 levels over 0.2 ng/mL had a shorter survival time than dogs with Tn-1 levels < 0.2 ng/mL.4 In a third paper, levels of high-sensitivity troponin I (hsTnI) were found to be prognostic in 202 dogs with degenerative mitral valve disease. This paper did not find that NT-proBNP was prognostic. Dogs with hsTnI > 0.025ng/mL had shorter survival times. The rates of increase of NT-proBNP and hsTn-I over time were also associated with likelihood of dying of cardiac-related disease.

My conclusions

While cardiac biomarkers are existing, non-invasive and promising, there are overlaps between the levels found in healthy animals vs those with cardiac disease, and in cardiac respiratory disease vs non-cardiac respiratory disease dogs and cats for each marker tested. In addition to this, animals with systemic illness may have elevations in troponins unrelated to cardiac disease.

The different reporting methods and various cut-off points used to achieve statistical significance in the above papers also make drawing clinical conclusions difficult.

So yes, troponins and other biomarkers might help to distinguish cardiac from non-cardiac respiratory disease, especially if the animal has a loud murmur. But in an emergency situation, clinical examination, traditional imaging modalities +/- response to treatment are still of greater utility.

References

1. Ranini A, Carini. Tropoerin elevations in conditions other than acute coronary syndromes. VHRM. 2011 Sep;997.


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