Centre For Veterinary Education

Celebrating our Leaders

2015 TG Hungerford Award

Celebrating 50 years of service to the veterinary profession in 2015

Follow Tom Hungerford’s ‘goanna track to success’...
Chlamydia 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 (Chlamydia felis) infection can experience quite intense and uncomfortable ocular inflammation. With no obvious downsides, practitioners should seriously consider vaccinating against Chlamyphilia (Chlamydia felis) routinely, particularly in younger cats.”

Prof T Graaff-Jones, BVetMed PhD DECVIM MMRCVS, University of Bristol, 2013
FROM THE DIRECTOR

The striking cover in black & gold of our first C&T for 2015 attracted a lot of attention and even one comment that it looked like every copy should have been accompanied by a bottle of Johnnie Walker Black Label Scotch whiskey!

Our 50th year continues to proceed well, with strong attendance at recent events in Launceston, Adelaide, Sydney and Brisbane. Each year we strive to provide interesting programmes across Australia, to support our members in particular, and the veterinary profession, in general with varied programmes offering choice and flexibility.

In April, we held a two day pharmacology seminar in Sydney, in association with the Pharmacology Chapter of Australian and New Zealand College of veterinary Scientists (ANZCVS). At the end of each day there was a panel discussion where unanswered questions were posed and some lively debate took place. One of the more engaged members of the audience has written two articles for this edition. Ani Seavers is one of our regular contributors and in this publication continues to challenge all vets to think more deeply about everything they do in day-to-day practice.

A dinner was held in Sydney after the pharmacology seminar for the presentation of the TG Hungerford award to Dr Stephen Page for his contribution to continuing veterinary education over many years. Jill Maddison (a classmate of Stephen at Vet School) spoke warmly about how fitting Stephen was as a recipient of this award. The award was presented by Prof Rosanne Taylor, the Dean of the Faculty of Veterinary Science at the University of Sydney. Stephen then gave a humorous and entertaining reply. Jill’s more formal citation is published in this C&T, along with Stephen’s considered reply. If you want to view the more light-hearted recording of this event, I strongly recommend you view it in the eBook.

This June C&T contains an eclectic collection of articles ranging from horses and goats, to dogs and cats, as well as three great perspectives. Many of the articles have been written by regular C&T contributors, but we continue to encourage others in the profession to send us articles. There must be at least one interesting case waiting to be written up in everyone’s files?

As Stephen Page stated in his acceptance of his award, forums like C&T provide a rich source of clinical anecdotes, which have been accompanied by a bottle of Johnnie Walker Black Label Scotch whiskey!

As Stephen Page stated in his acceptance of his award, forums like C&T provide a rich source of clinical anecdotes, which have been defined anecdote as “a brief account of any curious or interesting observations and topics for valuable research.”

In 1992, as EBM was emerging, Pickering wrote a great opinion piece in the BMJ entitled “The neglectful treatment of the medical anecdote”. He defined anecdote as “a brief account of any curious or interesting incident” (does that sound familiar?) and concluded that “that habit of curiosity, of critical regard and appraisal of their work, is being lost” – to the detriment of clinicians.” See page 6 for the full article.

Dr Hugh White, The Director of the Centre for Veterinary Education

2015 | Celebrating 50 Years

Major Conferences

Soft Tissue Surgery Conference  *Also see workshop Monday 15 – Thursday 18 June  |  Melbourne
Clinical Dermatology Conference
Monday 14 – Friday 18 September | Port Douglas

Seminars

Ophthalmology Seminar – Theory and Practice
Saturday 25 July | Sydney  Also see workshop
Canine Internal Medicine Seminar
Sunday 11 October | Port Macquarie
ECOCPD – Small Animal Radiology Seminar
Saturday 31 October | Melbourne

Hands-on Workshops

‘Soft Tissue Surgery: Wound Management Workshop
Friday 19 June – Melbourne

Basic Echocardiography Workshop
Saturday 18 & Sunday 19 July | Brisbane
Ophthalmology Workshop – Theory and Practice
Saturday 26 July | Sydney

Basic Echocardiography Workshop
Saturday 25 & Sunday 26 September | Melbourne

Stress Free Surgery Workshop
Saturday 17 October | Sydney

Diagnostic Ultrasound Workshop
Friday 6 November | Sydney

TimeOnline – Online CPD

Practical and Advanced Dentistry
Monday 6 June – Sunday 5 July | Online
Reptile Medicine
Monday 22 June – Sunday 19 July | Online

Feline Anaesthesia
Monday 13 July – Sunday 9 August | Online
Avian
Monday 27 July – Sunday 24 August | Online
Demyelinating ECGs
Monday 10 August – Tuesday 13 September | Online
Anesthetic Complications
Monday 24 August – Tuesday 27 September | Online

Feline Emergencies
Monday 7 September – Sunday 4 October | Online
Small Animal Behaviour
Monday 21 September – Sunday 18 October | Online
Wildlife
Monday 12 October – Sunday 8 November | Online

Respiratory Failure
Monday 26 October – Sunday 22 November | Online

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Diseases in Wild Birds
Tuesday 30 June | Online
Bovine Anaemia, Jaundice and Red Urine: is it Thalassaemia?
Tuesday 28 July | Online

Hindlimb Lameness – Seeing the Trees
Tuesday 24 August | Online

Sudden Death – Investigating Causes in Cattle
Tuesday 29 September | Online

Pharmacology – Popular Veterinary Nutritional Supplements
Tuesday 27 October | Online

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September

2016 Early Bird Ends
2016 DE Super Early Bird Ends
School holidays (NSW)
PodcastPLUS – Free to CVE Members
Follow Tom Hungerford’s ‘goanna track to success’...

The CAT is the brainchild of Dr Tom Hungerford, one of the founders of the PSP* (established in 1965) and the first Director (1968-1987), who wanted a forum for uncensored and unedited material.

...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.

The first CAT, contributed by Dr R M Kibble from Kurring-gai Animal Hospital, Turramurra North, NSW was on ‘Intermittency – Ulcerative Conditions’ and was issued on 29 April 1969. CAT Members are reminded that this and other CATs’ Perspectives, Proceedings and veterinary publications are available to CVE members through the CVE Library. Contact cve.library@sydney.edu.au or call us on +61 2 9351 2799 if you’ve forgotten your Username and Password for access.

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Your financial support sponsors the production of the C&T Series in high quality colour print format as well as the complementary digital eBook version (facilitating the inclusion of film clips, downloads, rollovers and enlarging of images) and postage costs.

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...and more C&T articles and Perspectives are needed.

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WINNERS

MAJOR PRIZE

Entitled the recipient to one year’s free membership of the CVE: RESTRINGENT OF HORSES IN THE PADDOCK OR PORTABLE CRUSH by Nick Scott

CVE PUBLICATION PRIZE WINNER

Entitled the recipient to a CVE proceedings of their choice: www.vetbookshop.com

FELINE PLASMA CELL PODODERMATITIS – A PATHOLOGIST’S EYE VIEW by Melanie Dobromylskyj

And in March 2015 Issue 276, apology for the omission: ACHOMEGALY IN A MALE DOG by Shelley Wilshire

*The Post Graduate Foundation in Veterinary Science of The University of Sydney (PGF) was renamed The CVE in 2008.

Citation prepared by Jill Maddison, Richard Malik, Aine Seavers and Anne Fawcett. Delivered by Jill Maddison.

Stephen graduated from the University of Sydney BVSc with First Class honours in 1980. Following graduation he was an intern then resident at the Rural Veterinary Centre Camden, culminating in the award of Master of Veterinary Clinical Studies. He then embarked on a life-long career pathway in industry which culminated in his term as head of research in the Asia Pacific Region for Pfizer Animal Health. Throughout his career, Stephen has made enormous contributions to both small and large animal veterinary pharmacology in Australia, Europe and the USA. He was instrumental in bringing selamectin successfully to the market in Australia whilst achieving a Masters of Applied Science in Environmental Toxicology. His work is characterised by exceptional scientific rigor and breadth, but this work does not form the only basis of this nomination.

In addition to his industry roles, and later a number of different private consultancies, Stephen has been dedicated to fostering the disciplines of veterinary pharmacology and therapeutics within Australia. He has worked especially effectively with the Australian College of Veterinary Scientists (ANCVS) and its Pharmacology Chapter, being an examiner and head examiner in this subject for many years. He regularly presents at Science Week. The Pharmacology Chapter is unique in providing detailed notes and study guidelines for ANCVS membership (MACVSc) candidates. Stephen was instrumental in developing this resource, by badge-fag and co-opting interested members of the chapter. Likewise, he was pivotal in the development of the ‘Block Course’ to assist MACVSc candidates to prepare for their examination.

Stephen made a seminal contribution to arguably the most popular veterinary pharmaceutical resource available, in concert with Dr Jill Maddison and Professor David Church. Critically, this text is clinically focused and based not just on drugs and body systems, but also on clinical problems. Highly readable and a great seller internationally, it’s onto its second edition.

Together with Associate Professor Paul Mills, he played a critical role in obtaining a $250,000 grant from the Australian Learning and Teaching Council for the development of a national pharmacology curriculum, the Veterinary Clinical Pharmacology Network, a portfolio of clinical case scenarios illustrating key pharmacological principles. The Australasian Veterinary Schools and Dr Amanda Craig from the School of Pharmacy at Canberra University will be involved in its implementation.

Stephen is an unassuming, extremely hard working quiet achiever and a truly original thinker. He cuts directly to the heart of an issue and provides extraordinary insight and scholarship. His innate personal humility, tolerance of human error, and juggling of responsibilities with skill, are key to his success.

Stephen is head of the Therapeutics Advisory Committee of the Australian Veterinary Association (AVA), an onerous role and largely pro bono, which plays an absolutely vital role in interfacing between drug manufacturers, veterinarians in the field and government regulators. Tiredless.

Stephen is extremely effective in terms of: contacts; insight into the pharmaceutical industry and the veterinary profession; his ability to think strategically and wisely about difficult issues; his diplomacy; and finally his ability to be effective and to bring about change. Permeating the intoxaction of cats, an emergency feline intoxication problem in Australia recently was attributable to the availability of these products without advice at supermarkets and pet barns. Stephen provided not only intellectual and moral support to colleagues concerned with this issue but also the intellectual and theoretical frameworks to tackle the problem. An AVA task force subsequently researched the advertising, marketing and labelling of these products and published an editorialized lead article in the Journal of Feline Medicine and Surgery (Appendices 1 & 2).

Stephen, typically, took this further by networking with key opinion leaders in Europe, and making a detailed submission to the USA Environmental Protection Agency in relation to this issue. Stephen contributes to the Control and Therapy Series, teaches part time in a number of Masters Programs offered by the Faculty of Veterinary Science of the University of Sydney and develops close interaction with veterinary colleagues in developing nations. He frequently assists practitioners in his own time by conducting high quality, in-depth and extensive literature reviews to assist in solving clinical problems. He consistently encourages colleagues to take a balanced, scientific approach to often emotive issues.

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TGW HUNGERFORD AWARD ACCEPTANCE SPEECH

Stephen Page

To be the recipient of such a prestigious award is a massive honour for me and a huge contrast to being medically and legally pressed in his inessential thirst for knowledge and its dissemination and a great role model for me (and many others). It is always a privilege to be able to help others and a very rewarding experience. To be recognized by my colleagues is absolutely humbling. However, I feel that my work is only just beginning, building on many years of conversations, research and unique experiences. I continue to have a trusted voice as a veterinarian, I feel like the fourth Prince of Serendip, travelling from one unexpected and exciting frontier to another, discovering new things along the way. I was helped and influenced enormously in my formative undergraduate days by Farrow and David Watson for small animals, David Hutchins for horses, Bob Love for pigs - all so clear in their thinking, all helped lay foundations that could then be tested when as an intern and resident I was responsible for facilitating the learning of students, of all us separated by so little experience. I quickly learned that questions from enquiring minds are an invaluable and limitless source of cognitive stimulation. Finding answers to questions has been my lifelong quest.

I have an insatiable thirst for knowledge and can never rest sharing what I learn with others. And I am continually surprised how many people just love to find out new things too. I no longer need information either, so many times new and curious links eventually lead me in new directions. Fortunately the garage I use to store the massive pile of information I hoard was sold and a new owner has taken an interest. I hope they appreciate the ‘just in time’ information, the annotated bookshelves, the computer files and the ever-growing video collection.

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I cultivate an interest in adverse drug reactions while in industry which led to increasing interest in tissue residues in livestock, contaminants in feed and the various impacts of poisonous plants and other environmental toxins. When the University of Sydney was preparing to begin an online Masters course in Veterinary Public Health Management I was asked to help facilitate the modules on hazards to human and animal health and on food safety. More interaction with students from all over the world eventuated as did many opportunities to expand my own knowledge horizon. Students working with FAQ later invited me to work in Nepal providing training on risk analysis and to work in Thailand and Japan on food safety risk analysis. Being paid to work in Kathmandu, Chiang Mai and Tokyo was an exceptional experience and helped expand my veterinary network.

I continue to meet extraordinary people along the way. Just when I think I have reached a plateau, a new challenge emerges. Richard Malais’ call to action to prevent permethrin intoxication of cats beckoned me to join the team that was forming - and a new and wonderful adventure commenced. CVE was able to survey vets and a mammoth hidden cause of deaths was revealed. Actions were taken, the results were published, international collaborations were established and legislative changes made. The issue was increased and hopefully many feline lives were saved.

My veterinary path has been far from linear. I never plan ahead, things just happen, and I continue to observe and wonder and find inspiration from colleagues and students.

My path started before the launch of evidence based medicine (EBM) and has continued throughout my veterinary career. Despite many witnesses a number of changes. By the way, how was diagnosis and treatment undertaken before EBM - surely it was not evidence free? Human and veterinary medicine had been successfully helping somebody else deal with illness – only a perception of success? (Human perception or risk, framing and susceptibility to logical fallacies is another interest, but that might be a topic for another day.)

This brings me to Control & Therapy, another great initiative from Tom that has been collecting practice wisdom for 50 years, a magnificent repository with an international audience.

But surely the content of CAT is all anecdote? Amazing that few people question the validity, particularly during the age of EBM. In 1992, as EBM was emerging, Pickering wrote a great opinion piece in the BMJ entitled ‘The neglect of therapeutic trial: the evidence of anecdote’ and the message of that optimism and the reality of any curious or interesting incident (does that sound familiar) and concluded that ‘that habit of curiosity, of critical regard and appraisal of their work, is being lost’ – to the detriment of clinicians. Almost 20 years later, anecdotal evidence was being emphasized – Nunn (2011) wrote that ‘not all anecdotes are worthless and not all randomised controlled trials (RCTs) are gold’ and in his opinion, evidence derived from anecdotes in medicine is more than a little ahead of EBM.

Why but was there thus interest and in recognition of the value of anecdotes, those individual observations, those clinical vignettes? Those who understand the safety of drugs is concerned was brought about in great part by the couple of sentences written by Bill McBride in the Lancet in 1961 about his personal observations of a new embryopathy associated with the use of thalidomide by the mother – no RCT, just an individual’s clinical observations and intuition. Aronson is a heavyweight in the world of adverse drug reactions and always has something interesting to say. He wrote that ‘anecdotal case reports contribute about one third of the published literature on adverse drug reactions and interactions’ and that ‘each suspected adverse reaction should be reported in detail and reactions should be reported in such a way that the pattern of reactions need to be recognized’ (Aronson 2005). He realised that a single case should be reported, but that interpretation required many cases – but each can add to our understanding of a pattern to be recognized.

The transformation of how the safety of drugs is considered was the role of anecdotes must be acknowledged, studied and utilized. ‘The core competency of excellent physicians across the centuries is practical wisdom, what Aristotle called pronoia, the ability to determine what is possible and proper to each unique clinical situation’ was noted by LeBlond (2013) who went on to say that ‘the increasing emphasis on narrowly restricted expertise and technical proficiency as the foundation for medical care can obscure the realization that objective knowledge and technical competence are sufficient for clinical excellence’, ‘without an equal emphasis on mindful reflection, thoughtful understanding, making ethical and moral decisions, practice patterns will not be replaced by technicians,’ and finally, ‘we must encourage and mentor learners to strive for practical wisdom what is the core competency of all excellent physicians’. So how could I have not stumbled on this stuff during my formative undergraduate days? ’

I have been collecting practice wisdom for 50 years, a miniature practice wisdom pyramid in the past, there has always been another pyramid, only now coming into focus, the phronesis hierarchy, without which medical or veterinary patients will not benefit ideally and yet today the pyramid is still the phronesis hierarchy pyramid in the past, there has always been another.

But what is phronesis? To EBM (McMaster University in Ontario) noted that ‘if evidence-based health care is to meet its potential, the important role of phronesis must be acknowledged and worked’. phronesis that C&T has played and continues to play such a highly valuable role. Well ahead of its time, very prescient.

It has been and continues to be a privilege to be involved in control and therapy. It is an honour to be asked to be the recipient of the TG Hungerford Award – especially when my own learning has benefited at least as much but probably more than that of my colleagues and students. It was a privilege to receive the award from Prof Rosanne Taylor who was a student of mine – albeit briefly and many years ago. I hope she can only remember the good things I as struggled with teaching as a newly graduated resident veterinarian. We have had many challenges and exciting times and every new project is built on the accumulated experiences of the past, all enhanced by the interactions, collaborations and network of colleagues and friends.

References

View the video of the Award ceremony at www.cve.edu.au/tghungerfordaward

Note: Stephen has provided a comprehensive reference list which, due to space constraints, is available in the eBook.
LETTER TO THE EDITOR:
CREDIT WHERE CREDIT IS DUE FOR PET INSURERS

Dear Editor,

Subsequent to my article Pet Insurance – Problems And Potential in C&T (Issue 276, Sep 2014) we can report some pleasing progress with pet insurance.


Encouragingly, insurers are now participating in veterinary conferences where they can interact directly with veterinarians for advice or feedback. Several major insurers are developing electronic online claims processes and a trial is about to commence. This should help reduce theцион and frustrating problem of loss of claim forms in the post (or more likely during processing). We hope insurers will also be receptive to simplification of paper insurance claim forms and claims processes - personally I am still mystified as to why detail on vaccination status is required for every claim when the vast majority of claims are not related to preventable infectious disease. The costs of completion of forms are significant to vets and any simplification would be welcome. Many industries face rapid growth and change. One has only to look at the airline industry moving from travel agents and paper tickets to online sales, check in via Apps, mobile phones or online, frequent flyer programs and simplification and any changes are welcome. Many industries face rapid growth and change. One only has to look at the airline industry moving from travel agents and paper tickets to online sales, check in via Apps, mobile phones or online, frequent flyer programs and any change is welcome. Many industries face rapid growth and change.

One key objective was that, when requested, insurers offer review of claims by an Australian registered vet who is fluent in English and medical terminology. One major insurer, Petsure, has developed a vet hotline, allowing vets to automate communication processes without having to battle through call centres.

Insurers are actively seeking feedback from vets. On the subject of denied claims I have been asked them to review their policies on cover for parasitic conditions. Some insurers deny claims on any parasitic disease – few would object when there is a preventative available – such as for heartworm disease or hookworm. There is an obligation on the owner to maintain basic healthcare object when there is a preventative available – such as for heartworm disease or hookworm. There is an obligation on the owner to maintain basic healthcare.

We hope insurers will also be receptive to simplification of paper insurance claim forms and claims processes - personally I am still mystified as to why detail on vaccination status is required for every claim when the vast majority of claims are not related to preventable infectious disease. The costs of completion of forms are significant to vets and any simplification would be welcome. Many industries face rapid growth and change. One only has to look at the airline industry moving from travel agents and paper tickets to online sales, check in via Apps, mobile phones or online, frequent flyer programs and any change is welcome. Many industries face rapid growth and change.

Following on from C&T No. 5416 'How Much Do You Know About Human Tick-Related Diseases?', Clinical Associate Professor Sheryl van Nunen has sent us the link to the program, which recently featured on the ABC’s Catalyst show, highlighting the dangers of ticks to humans.

WATCH NOW in the eBook or go to: www.abc.net.au/catalyst/stories/4177191.htm and please share with your friends and colleagues.

Thank You.

Photo courtesy of Anne Fawcett

Ovarian granulosa cell tumours (GCTs) account for more than 85% of all tumours of the reproductive tract in mares and 2.5% of all equine neoplasia. Typically these tumours are benign and unilateral in nature with the contralateral ovary commonly small and inactive. GCTs are hormonally active and produce variable amounts of inhibin, oestradiol and testosterone. It is therefore common for mares to present with behavioural abnormalities including aggressiveness, stallion-like behaviour, prolonged anoestrous, or nymphomaniacal behaviour. However, most mares with GCTs present with anovulatory anoestrous. GCTs display no breed predilection and have been reported in horses, ponies, mules, donkeys, mares, stallions, and geldings. The definition of anovulatory anoestrous depends on the stage of the oestrous cycle and stage of pregnancy. However, in the early stages of pregnancy, the definition of anovulatory anoestrous depends on the stage of the oestrous cycle and stage of pregnancy. However, in the early stages of pregnancy, the definition of anovulatory anoestrous depends on the stage of the oestrous cycle and stage of pregnancy.

Anti-Müllerian Hormone – A New Test for the Diagnosis of Granulosa Cell Tumours in Mares

Matamata Veterinary Services Ltd, Matamata, New Zealand
C&T NO. 5465
Jasmin Hyatt, Dave Hanlon, Fiona Hollinshead
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Ovarian granulosa cell tumours (GCTs) account for more than 85% of all tumours of the reproductive tract in mares and 2.5% of all equine neoplasia. Typically these tumours are benign and unilateral in nature with the contralateral ovary commonly small and inactive. GCTs are hormonally active and produce variable amounts of inhibin, oestradiol and testosterone. It is therefore common for mares to present with behavioural abnormalities including aggressiveness, stallion-like behaviour, prolonged anoestrous, or nymphomaniacal behaviour. However, most mares with GCTs present with anovulatory anoestrous. GCTs display no breed predilection and have been reported in horses, ponies, mules, donkeys, mares, stallions, and geldings. The hormonally active nature of GCTs allows endocrine analysis to be used successfully as a diagnostic aid. The current standard ‘GCT panel’ quantifies serum concentrations of inhibin, testosterone and progesterone. Increased serum concentrations of inhibin (>7ng/mL) and/or testosterone (>50-100pg/mL), combined with decreased circulating progesterone (<1ng/mL) are suggestive of a GCT. However, serum concentrations of these 3 hormones can fluctuate depending on the stage of the oestrous cycle and stage of pregnancy. In addition, in the early stages of tumour development, levels of all 3 hormones can be within the normal range. Anti-Müllerian Hormone (AMH) is a homodimeric glycoprotein produced by the granulosa cells of ovarian follicles that has a role in the regulation of follicular development. AMH is highly conserved across species and has been found in all domestic mammals tested so far (horses, cattle, dogs, cats, sheep and goats). Immunohistochemistry has previously shown that AMH is expressed in granulosa cells of normal equine ovaries as well as in GCTs. Serum AMH as a biomarker has been investigated as a more sensitive diagnostic aid than previously measured hormones in equine GCTs.

In horses, serum AMH concentrations were first characterised by Almeida et al. (2011) using a heterologous enzyme linked
immunoassay (ELISA) in normal cyclic mares, ovariotomised mares and mares previously diagnosed with a GCT. There were no significant differences in circulating serum AMH concentrations between normal cyclic mares and pregnant mares, thus negating any confounding factors encountered by inhibin, progesterone and testosterone analysis. The concentration of AMH in ovariotomised mares was equal to or less than the limit of detection of the assay, confirming the ovarian localisation of the hormone. AMH concentrations were increased in all mares with histologically confirmed GCTs. This demonstrates significantly increased sensitivity when compared to inhibin and testosterone analysis, with levels elevated in only 73% and 45% of GCT cases respectively if these hormones are measured individually. Ball et al. (2013) conducted further studies, concluding that AMH, when measured in combination with testosterone and inhibin, achieves a sensitivity of 100%.

Catterbury Health Laboratories (Christchurch, NZ; E. info@cch.co.nz) and Vetpath Laboratories (Perth, WA; Vetpath.reception@vetpath.com.au) currently perform the only AMH assays available for clinical use in Australasia. Though the assays are designed for humans (NZ), and dogs and cats (WA), the manufacturers report that they also cross-react with AMH from other species including the horse. Though each assay may not have full measurement (cross sensitivity) of the AMH from other species it appears adequate for differentiating the elevated AMH observed in mares with GCTs compared to healthy mares. Both laboratories are currently in the process of establishing individual reference intervals for the mare and therefore it is useful to provide a clinical history with your submission, especially if you have confirmed the diagnosis of a GCT by histology. It is also important to contact the relevant laboratory for specific submission requirements. Note that results are reported as AMH pmol/L, to convert to ng/mL [the unit often quoted in veterinary publications] divide the pmol/L result by 7.14.

REFERENCEs

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10 June 2015

When examined rectally or vaginally it is surprising that very few horses will actually kick, but when they do the results can be devastating and even fatal for the person behind them. Unlike cattle, rather than kick back with one hind leg most horses will kick up both legs and ‘fly off with both barrels’ which can generate significant force.

Without a properly designed horse crush there is considerable danger, but makeshift arrangements like 2 hay bales or reaching over a gate can cause a false sense of security and be even more dangerous. Poorly designed ‘crushes’ can be even worse; in fact, one of the worst kicks I have ever received was when the handler allowed a mare to jump forward and kick over the flimsy rails that had been arranged behind her.

Likewise, use of the client’s serving hobbles is not much of a solution as they are only placed around the mare’s hocks and still allow a decent kick to be delivered.

Unfortunately, those of us who are in part-time equine practice are likely to have this problem more than those who work on large studs etc. Some operators favour a single sideline which places a large strap across the neck and then a strap is hooked to the hind pastern and the leg is dragged forward almost off the ground and a special quick release buckle attaches this line to the neck strap.

I used this apparatus for a while but found it placed the horse in an unnatural position, particularly for rectal examinations, and was very cumbersome to stow and then get out of the vehicle. The paired sidelines as pictured, with a strap going from front to rear or hind pastern to hind pastern on each side, have served me very well, and sometimes I call them my portable crush.

They were made for me by Goulburn saddler, Bill Duttikins‘ in the late 1970s and since then have been placed under severe pressure by some badly behaved horses. After all this time, some of the buckles are getting a bit stretched but the straps are still perfectly serviceable.

Bill made the straps out of a basically treated leather which stretched at all and only very occasionally gets any leather pressure by some badly behaved horses. After all this time, some of the buckles are getting a bit stretched but the straps are still perfectly serviceable.

The Major prize Winner is entitled to a year’s free membership to the CVE.

The two major sponsors of the CVE are also offering special prizes.

The major prize Winner is entitled to a year’s free membership to the CVE.
Again the straps are about 3 cms wide. The centre piece is 18 cms long and contains a piece of steel sewn into the leather to keep the knees apart at the correct standing position.

The short piece to the buckle is 12.5 cms long while the long free strap to wrap around the legs is 108 cms.

You will notice that my long strap is wrapped twice around the mare’s leg as my straps are always inclined to slip down below the knees. This is because my straps are made with 2 steel rings for the straps to go through. I have seen other hobbles work much better when the rings are replaced by “D”s which are shaped like a square.

The attached photos show a mare prepared for a post foaling uterine examination. Usually I would not use the knee hobbles as well unless she was very excitable.

The last 2 photos show how the sidelines are buckled back to themselves and how the knee hobbles are threaded onto the legs.

*Details Goulburn Saddlery, 37 Verner St, Goulburn NSW 2580. T. (02) 4821 2838

**NOTE FROM HUGH WHITE**

This excellent C&T from Nick Scott on horse restraint will strike a chord in the hearts of every older vet. I am sure there are many vets in mixed practice who are still confronted by the need to conduct an internal examination on a horse where there are inadequate facilities. Thirty to forty years ago, workplace health and safety was regarded as a common-sense matter, but this did not stop many of us from placing our lives at risk by doing internal exams on horses around stable doors or gates, in horse floats or in cattle yards or races. Over the years, I personally know colleagues who suffered life-threatening injuries including ruptured livers and spleens when conducting these procedures, and others who have received severe head injuries. While OH&S guidelines may advise ‘ideal’ workplace facilities, vets will still be presented with horses on hobby farms or commercial farms where there are inadequate facilities. Nick has described restraints which may be viewed as old-fashioned by some, but in the absence of ideal facilities, could save a life.

**FURTHER COMMENT FROM DAVID JOHNSON, PACIFIC VETCARE, COFFS HARBOUR**

This is a great article by Nick, but my concern is that the equipment is pretty difficult and specialised to use unless used by someone with competent horse-handling skills. (A bit like describing how to put on a saddle correctly – sounds easy but actually difficult to do properly unless you know what you’re doing.) In an ideal world, each practice should have specific policies and procedures on handling horses; what equipment, sedation, handlers and when and where rectal examinations of horses can be performed. For example in our practice the policy is no rectal exam can be performed unless the horse is in an approved crush.

**REPLY TO DAVID JOHNSON FROM NICK SCOTT**

I would concur with David that without these straps I would be unwilling to perform any examination [not just rectals] to the caudal end of a horse unless it is in a proper crush.

However I would take issue that my straps could only be used by someone with ‘competent horse handling skills.’ I would submit that we should have or study handling skills before trying to examine or treat ANY species.

The difficulty is that these days a large proportion of mixed rural practices have such a large caseload of small animals that many of our new graduates are not getting the chance to learn the skills in handling large animals which they need to operate safely and effectively.
LEG WOUNDS IN HORSES

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Go to the eBook version of this issue to read 5 other C&Ts about wound management in horses including C&T No. 5168, Proud Flesh.

Honey in wound management, C&T No. 4313
Management of a forelimb wound in a mare, C&T No. 4618
Large flesh wounds, C&T no. 4663
Proud flesh, C&T No. 5168, Mar 2011, Issue 262
Wound Management in the horse, C&T No. 5169, Sept 2011, Issue 264, reply to Proud Flesh (C&T No. 5168, Mar 2011, Issue 262)

Lilian Wong and Jason Andrew wrote an excellent C&T No. 5168 Proud Flesh which refers to an AVJ article by Andrew Dart et al. I certainly do not intend to try and refute any of their recommendations.

The only comment I would like to make is that I find in a general practice situation, successful treatment of these large wounds depends very much on whether the owners have the necessary time, dedication, horsemanship skills and finances to devote to the several months of treatment and bandaging that some of these wounds require.

Secondly, the temperament and co-operation of the horse in tolerating these treatments can make a huge difference to the treatment regime and the outcome.

Many times, once the wound has a reasonable granulation bed, I have advised to just cover the wound with limestone powder (microfined limestone, not the slaked lime) and clean all the dried scabs off once or twice a week.

This can give quite a reasonable result but of course then the wound is at the mercy of various other ‘advisors’ who want to put weird and wonderful potions on the wound at least daily and irritate it and cause more damage.

However, one of the most frustrating wounds to treat is when the flexor tendon sheath below the carpus or hock is damaged, sometimes by quite a small wound. Often this wound may not even be apparent at the initial examination.

There is usually no actual damage to the tendon but the rupture of the sheath will cause a massive outpouring of sheath fluid which looks like copious synovial fluid. Exuberant granulation tissue masses around the wound but it will not seal under a bandage and every time the wound is exposed you will see a large gelatinous clot of serous fluid which I feel is pathognomonic. Of course, the tendon sheath will be susceptible to penetrating infection which could spread right down into the hoof so you must keep the wound covered and give high doses of systemic antibiotics.

Once I am sure that infection is controlled, I have found firm pressure applied to the wound will gradually allow the granulation tissue to spread in and fill the defect in the tendon sheath. Those of us who only do mixed horse practice often do not have the confidence to apply a full leg cast, particularly when the horse is quite a distance from the clinic, making supervision difficult. In the past, I have fashioned a ‘half leg cast’ out of a product like Hexcelite or Vetlite or Askina cast. Once that cast sets, I then get the owners to redress every 2 or 3 days with an appropriate ointment then a strip of cotton wool and gauze roll or Combine dressing on the wound. Then the cast is applied over this and then strapped onto the leg with Elastoplast.

Figure 1. The wound in late November, soon after injury, with the initial infection controlled.

Figure 2. Wound on 16 December 2013 showing the protruding proud flesh.

Figure 3. In late December 2013, after the splints had been applied for over a week.

Figure 4. The wound on 7 January 2014.

Figure 5. The wound on 26 January 2014.
Recently, I had a case with one of these wounds; the horse was 28kms from town and considerable costs had already been incurred with antibiotics, bandages, revisits etc. On my visit just before Christmas, in desperation I gave them 2 pieces of PVC pipe which I had saved for calf splints. I advised the owners to tape those together and strap over the wound as a stop-gap measure until I could get out with some casting material. The RESULTS WERE SO GOOD that we didn’t ever get to make the cast up and you hopefully can see from the attached pictures that the wound healed very quickly under the splint and bandages, once the tendon wound sealed.

However, I must say that a huge factor in the successful treatment was that these owners were particularly dedicated in following my advice in changing bandages, cleaning the wound and giving antibiotics etc. Also, the horse was an excellent patient and tolerated injections and treatments very well.

To summarise my clinical notes, I first visited ‘Ned’ on 13.11.13; he had been freed from a fence entanglement 3 days before. The off hind leg was now severely swollen and lame with the flexor tendons actually exposed and seeping about half-way between the fetlock and tarsus. Luckily, there seemed to be good warmth in the lower leg so it seemed that the wire had not damaged the major blood vessels.

Ned is a big horse (estimated weight 695 kgs) so the wounds were cleaned as much as possible and poulticed for 2 days then bandaged with Flamazine, cotton wool and gauze roll, and Elastoplast. Initially, the owner and her very efficient family changed the dressings daily then every second day etc as the infection slowly improved.

On the day I gave him 48 mLs of Gentam (100mg per mL) i/v and prescribed 50 mLs of Propercillin be given i/m TWICE daily. Tetanus prophylaxis was also given.

By the 15/11 they reported a big improvement so the twice daily penicillin was given till the 16th, then 50 mLs was given once daily until the 25/11.

When I revisited on 22/11/13 he had improved and was only just lame at the walk. Pieces of skin were now sloughing from the wire encirclement at the front of the leg. The tendon sheath was pouring fluid but it all looked clear so I was very hopeful that the penicillin was controlling the infection.

From the 25/11 I advised to keep bandaging but to now give Benacillin 50 mLs once every 48 hours. I felt it was essential to keep up some antibiotic cover while the tendon sheath was still open and discharging.

They continued with this, giving me regular phone updates until I revisited on the 16/12/13. The lameness was going well but the wound was still leaking copious fluid and had not really closed in at all.

As we were flat out with the pre-Christmas rush, I could not decide when I would be able to revise to make a half cast so I supplied the owners with the splint and explained how to place it over the cotton wool and gauze then strap it onto the leg with Elastoplast.

The owners reported good improvement with this regime and when I re-examined the leg on 7/1/14 the wound had shrunk significantly with just a small amount of tendon discharge coming from the medial side, so they started placing the splint more on that side rather than directly at the back of the leg. After that, I suggested it should be safe to leave the dressing on for 3 days as the discharges onto the dressing were much less. I advised to continue with Benacillin every third day till all discharge had stopped.

Please note the PVC splints I used were made from 90 mm storm water pipe. I also try to keep some emergency splints of 150mm sewer grade pipe which is thicker and more suitable for splints on adult animals. The annoying thing about making these splints is that when they are split down the middle the resulting semicircle is too narrow to fit on the leg. One must split the pipe into thirds to allow the splint to be flat enough to be strapped onto the leg.

The final photos show the splints on their own, then taped together; they each were 18.5 cms long by 8 cms wide. Unfortunately we don’t have any pictures of the leg when Ned first presented on 13/11/13. Fig 1 was after a week or 10 days, the initial infection is settling down well but you can see proud flesh forming around the edges while the gap in the middle where the tendon is discharging is actually larger than at the initial visit.

Figure 6A & B. The PVC splints were made from 90 mm storm water pipe.
This cat was diagnosed with a mycobacterial infection of the conjunctiva by histopathology (and acid-fast staining). The causative agent was identified via PCR as the novel species provisionally named *Mycobacterium* sp. ‘Tarwin’ that causes disease in cats typically geographically located in the eastern suburbs of Melbourne, the Mornington Peninsula and the western part of Gippsland, Victoria. (Fyfe, J. A., et al. Molecular characterization of a novel fastidious mycobacterium causing lepromatous lesions of the skin, subcutis, cornea, and conjunctiva of cats living in Victoria, Australia. J Clin Micro 2008;46: 618-626.) Lesions caused by *Mycobacterium* sp. ‘Tarwin’ are mostly found on the head (including the eyelids, conjunctiva and/or cornea, muzzle/lips, gingiva) and front limbs. This species has not been shown to cause widespread cutaneous or systemic disease.

Definitive treatment guidelines for *Mycobacterium* sp. ‘Tarwin’ and indeed the other fastidious mycobacteria that cause feline leprosy are yet to be established, because the antibiotic susceptibility of these organisms is largely unknown due to the fact that they are unculturable via current mycobacteriological methods. Clarithromycin combined with rifampicin (with or without clofazamine), ideally with surgical resection of lesions, appears to be the most efficacious treatment regimen. As with other mycobacterial infections, it is recommended that medical therapy be continued for at least 1-2 months past resolution of clinical signs or following resection of all visible lesions.

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Figure 1. Swollen conjunctiva lateral canthus. Note discrete paler colour mass upper eyelid which appeared lymphoid in nature.

Figure 2. Swollen conjunctiva lower eyelid near third eyelid.

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FROM THE ISFM FORUM – FELINE-MEDICINE FIP & STEM CELL THERAPY

C&T NO. 5469
Rachel Payne


QUESTION

We have a 4-month-old kitten with FIP (actually our head vet’s kitten) and have read some articles discussing using mesenchymal stem cells for FIP. Do we stem cells here and I’m wondering if anyone had any experience in using stem cells in these types of cases? It is the wet form. IV injection or intraperitoneal as well?

Any input would be very much appreciated.

REPLIES

Reply No. 1
Richard Malik, CVE
Stem cell therapy is very interesting – I think there is promising evidence that it is helpful for degenerative joint disease – where the cells set up show as anti-inflammatory cytokine factories. This makes sense, and there is increasing evidence for it. There is a paper by Dow group for its use in renal disease, and another in Quimby’s in the JSAP concerning cardiomyopathy in dogs where it was not helpful.

But why in FIP? There needs to be either EVIDENCE or at least a conceptual reason for trying it.

Response from Rachel
Thanks for your input, Sam.

We have in-house stem cell therapy set up (have had it since the beginning of 2012) and have so far done 13 dogs (no cats, but this is the next step!).

We have had huge success with stem cell therapy in dogs for degenerative joint disease. We have to choose our candidates very carefully and they are often those that have exhausted other routes of treatment. The clients that have taken the leap with us and stem cells as it is new for us, too! have been very happy with the results. I was a bit of a skeptic as well, but after seeing the response these dogs have had to it (nearly all of the clients expressing that their dogs are a bit of a skeptic as well, but after seeing the response these dogs have had to it)

It is pretty expensive but, for the right client and the right dog, such a good investment.

We have had a couple of potential renal failure cats as candidate; however, have not performed stem cell therapy on one as of yet.

My boss had stem cell therapy on his knee and I think this personal experience has made him even more in favour of it.

DELIBERATE BEFORE YOU MEDIATE – PART 2

C&T NO. 5470
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What about drug interactions? Be really careful using tramadol combined with drugs like Clonicepal® (clomipramine hydrochloride), Reconcile- (Fluoxetine hydrochloride) Pivofridodrogel that you don’t induce distressing serotonin syndrome. Also, take care with Ondansetron?

What about analgesic efficacy? Tramadol is a centrally-acting analogue of codeine (more on codeine later) with 2 modes of action. Tramadol and the O-d metabolite have a higher affinity for Mu and Delta receptors than Kappa opioid receptors and also inhibit norepinephrine and serotonin uptake in CNS.

VCA VSmall Anim Pract. 2013 Sep;43(5):1109-25. Pharmacokinetic studies in dogs have shown that dogs do not produce OCM as a substantial metabolite after tramadol administration, however, they do produce OCM. Therefore tramadol is not expected to have substantial opioid effects after tramadol administration. Plasma concentrations of tramadol after administration of 15 mg/kg by mouth to dogs were slightly less than the plasma concentrations achieved in humans administered 100 mg single doses, but the concentration of OCM was 10 times less in dogs compared with humans. The elimination half-life of tramadol in dogs is more rapid (1.1 hours) compared with humans (5.6 hours). The production of OCM, which has an elimination half-life of 3.6 hours in dogs, may produce some opioid effects in dogs. Repeated doses of tramadol either decreased drug absorption or enhanced presystemic metabolism of tramadol in dogs, in which a 60% to 70% decrease in tramadol plasma concentrations resulted after just 8 days of treatment (20 mg/kg by mouth).

In humans, tramadol can trigger spectacular orthostatic or positional hypotension. Does this occur in dogs? Again, no-one I know using it had blood pressure machines on the patient. Is the MI metabolite the cause and hence postural or standing hypotension not an issue in dogs? Again, no-one I have asked can answer this. Is analgesia mired where the dog simply cannot remain standing so lies ‘calmly’ up? As an esteemed colleague wondered aloud to me: Is this similar to how we once duped ourselves that ACP worked in thunderstorm phobia when all that happened was the poor dog lay there paralysed and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and terrified? Is the post-op tramadol dog not moving because as soon as it attempts to stand its blood pressure crashes so it lies there supposedly pain-free but, in fact, in pain and...
just someone who fits the analgesic to the patient and the work, things must be bad! The evidence that is available suggests that much higher dose you will get constipation and, if so, in an IVDD case the evidence for its use? Given all that, why then is codeine given to dogs with IVDD? Is it an effective analgesic in these cases? Is it fair to give a dog analgesia when, to my mind, the evidence is not yet there; there are, however, several real concerns but no real historicalcline as yet. The evidence that is available suggests that much higher evidence for its use? I initially get derisory looks from others when I declare I don’t use tramadol as if I am a bad doctor and practicing good medicine, evidence-based medicine. Their decision quickly changes to concern and self-doubt when I raise my concerns regarding the investigative due-process vets need to apply as a default process to any new medication procedure so as ensure a 360 view of any concept, good or bad. Overseas, the drug has now been changed to an equivalent of S8 due to its highly additive properties – not something I want to start home-prescribing for chronic pain if the opportunity for human self-abuse is in. The feline vets have done a globally remarkable job in the last 15 years emphasising that Cats are not shrunken Dogs. It’s now the turn of the canine vets to step up to the plate and emphasise that Dogs are not oversized Cats. Moving on to Codeine. Not all opioids are equal to all pain targets. In people, codeine is metabolised to morphine. As many vets know only too well from their own migraines – not all opioids are equal. When a migraine escapes so that codeine or morphine know only too well from their own migraines – not all opioids are equal. When a migraine escapes so that codeine or morphine only, about 7% (20mg/kg) is given in combination with other analgesics so sole efficacy wasn’t determined by Fior et al. What we have been told about it or it is, in fact, one of the safest… Why? First, let’s all acknowledge the elephant in the room. This drug has been, and continues to be used effectively and affordably for decades in millions of cases of urinary incontinence, right up to the present day. This drug is LICENCED for use in Australia for oral use in small animals so we are not using it off-label or out of species when used. We do not use it. Allegedly, this drug can cause bone marrow suppression. It does – but you have to give it every day! Yes, it’s a powerful hormone and we do need to respect it in every aspect. From handling to when and to what patient it is dispensed. But – has any practitioner ever seen bone marrow suppression with this drug when used for canine incontinence? And, if, so did that adverse drug reaction occur using the 5mg high doses or with the 1mg daily a 3 days then every 3rd day until condition ceases regime? I ask because when the very big push came about 15-20yrs ago for ‘Good vets’ to use synthetic, expensive, hormone analogues (around the time of the new drugs’ launches), I canvassed several international vet forums. Yet – NOT ONE VET has ever seen the issue I followed up this year with another 30 or 40 results. The only time an adverse drug reaction issue was seen was in 2 cases where 20x oral dose was given repeatedly by a human medic or an off-label mis-mis-use of injection was not only given but repeated. Otherwise, no adverse drug reactions. Lappin 1969 and Barnsli 1983 recommended it as the drug of choice for incontinence in dogs as 0.1-1mg/kg for 3-5 days and as required thereafter. So, why the scare mongering? The ‘practice based evidence suggests Stilboestrol® used wisely at low doses must be or the T of the safest drugs on our shelves but we are “sold” the opposite and told to use more expensive alternatives. Yes these alternatives carry their own risks – Pseudoef and Propalin both have hypertension concerns. So why are we lambasted for using Stilboestrol®? What harm, cost, power statistics does anyone who pushes the anti-Stilboestrol® warning have, that this shows to be so dangerous as to be avoided; especially when the stats for other drugs are much worse but don’t have the same bad press. Stephen Page did a superb job (no surprises there) in reviewing this topic for the AVA in AVU 1991. Page’s conclusions” need to be taught to every vet student as a given from the clinical front-line experience of practitioner-based evidence validates his statements close to a century later. I quote: “DES has certain advantages in being orally bioavailable with a short half-life. Short courses administered outside the luteal phase have not been associated with adverse side effects.” Given that 99% of the incontinence we see in is desoxed females, then we need that protocol a 20mg/kg amount of angst we self- infest when dispensing this medication. Incontinence will lead to euthanasia of a family pet in far greater numbers than the statistical risk of an adverse drug reaction from Stilboestrol®. All my clients are given the 4 options for incontinence control; all explained, success rates and risks explored, daily dosings outlined and the client chooses what they can afford and wish to use. About 90% choose Stilboestrol®, about 9% choose the highly effective but costly Propalin and the remaining 1% pick a selection of other meds/surgery options benign neglect. In 30 years, I can’t think of another drug that has given me less trouble – yet using it makes me a “Bad Vet’. PPIs Proton pump inhibitors continued My views on these are expressed in first part of my DE&MY article, so I have been now asked what I do use. That would be cautious low doses of Ranitidine (Zantac) which, despite the non-need for care in renal cases, will however get many renal uraemic patients eating again. It would appear to target the uraemic gastritis and nausea superably and brings quick and great relief to the dog and cat. You do need to check blood pressure and also do an oculor check as glaucoma at high, long doses has been recorded. Also, I’m finding it superb for the canine reflux/back/going drinking water cases that are controlled cheaply on just this drug for years. Dose changes between species, and in rabbits you need to use a higher dose. Rabbits 4-6 mg/kg bid-tid Guinea pigs 5 mg/kg bid Dogs 2 mg/kg bid-qid Cats 3.5 mg/kg bid Activity® and Bravoect® single packs Let’s end on a positive note. The definition of good business practice is to find and exploit a niche good or service that the opposition isn’t catering for, and predators. MGD seems to have embraced this theory whilst listening to what we, as front-line vets, need and they produce their products in single packs. Many vets seem to be unaware that our right to prescribe and our privilege does not mean we only use branded SIs – i.e. you can’t sell SS Comforts etc single out of their inner packaging. Whilst some might think they will never be caught doing so – the reprehensible, unethical action of splitting an Avantix pack may lead to the suffering and death of a cat against which you don’t have a legal leg to stand on. That said, it is also so
frightening for front desk staff to spend valuable time advising a walk-in-client on the correct flea control only for that person to walk back out without a purchase as they ‘only want one’ and ‘the vet’s put down the roof’ will split the pack for them. Since Activity4 has come on the market in single packs, we have been able to successfully complete every enquiry at our front desk. So, whilst Bayer’s Advantage range remains my preferred flea product, Activity4 is now a valid in our vet’s flea service. I am equally pleased to hear MSD’s B-Duo® tick product will also be in a single, non-scored, one animal single dose packing thus eliminating stress and lost time at front desk. Thank you MSD for listening to what vets want BEFORE you apply for registration sizes and dose regimes from thyroid medication to parasitic control.

But, best of all practitioners, use the best piece of equipment you own – Your own Voice, which follows this article here. Bayer’s blood testing will be covered in Part 3 BBVM.

REFERENCES


3. Tramadol has become a controlled substance in the UK and in the USA. a.[No authors listed]. 2014 Jun, Ketamine and tramadol recrystallized. Vet Rec 174(25):566. doi:10.1136/vr.100982.


CONCLUSION AND RECOMMENDATIONS

The CAT article by Aine has raised an extremely important point applicable to many drugs that have not been systematically tested for use in animals, especially those drugs with significant PK differences compared with the species for which the drug is primarily indicated – in this case humans. The literature describing the use of tramadol in dogs is growing and suggests that there are circumstances where tramadol could be used safely and effectively. But which circumstances are these and how does one determine what benefits and what risks? Certainly there are situations where tramadol should not be used or if used must be done with great caution and vigilance. In the end, the growing literature needs to be critically appraised and those situations where it can contribute positively to canine health and welfare clearly described. A review of tramadol would make an excellent Cochrane-type review – but who is going to perform this important task?

Note: Stephen has provided a comprehensive reference list which, due to space constraints, is available in the eBook.
INVITED COMMENTARY COURTESY OF:

Tanya Stephens
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For the practitioner there are some excellent EBVM resources available and the Royal College of Veterinary Surgeons is at the forefront with the RCVS Knowledge EBVM Network. The EBVM Toolkit from RCVS Knowledge is designed to help busy practitioners answer a clinical question with the best available evidence. www.knowledge.rcvs.org.uk/evidence-based-veterinary-medicine/ebvm-toolkit.

VETS WORLDWIDE ARE INVITED TO BE PART OF THE EBVM NETWORK.

These resources, enable clinicians to easily access systematic reviews without needing to undertake their own. I really like BestBets for vets and check it often for the latest reviews as these are regularly updated.

Skeptvet which is produced by the US based EBVM Association has a wealth of information and you can join the EBVMA to keep up to date and join in discussions. This group is very much practitioner focussed.

Always keep in mind, of course, ‘clinical expertise’ in EBVM decision making and don’t forget the client, the important third party in ethical decision making, and the one who pays the bills!

For more resources of interest, non veterinary organisations promoting EBVM include the UK based group Sense about Science and the Australian group Friends of Science in Medicine, which has over 1000 members including Gustavo Nossal and Peter Doherty.

INVITED COMMENTARY COURTESY OF:

Richard L’Estrange
BVSc MANZCVS (Vet Pharm)
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I would like to thank the CVE for very kindly inviting me to comment on C&I #5470, submitted by Dr Aine Seavers. I commend Dr Seavers for encouraging veterinary practitioners to constantly question the dogmas and trends that appear from time to time in veterinary practice and to do their own research and investigation of the literature. In my practice at least (when I had one), the use of tramadol was born out of the frustration of ‘what else can we do?’, for arthritic dogs in particular whose pain was either not well controlled on NSAIDs +/- other modalities, or who could not receive NSAIDs for some reason. It seemed a sensible idea to try this non-controlled human opioid which could be given orally and there seemed at the time to be no reason why it would not be both safe and effective. Decision-making regarding the use of pharmacologicals should centre on efficacy and safety in the target species. Whilst cats are not small dogs, it might equally be said that dogs are not small humans and may not even be large rats or mice.

I cannot for one minute claim to be an expert on tramadol, but from my reading on the topic I have the following concerns:

The drug is currently unregistered but available for use in dogs and cats in Australia under APVMA permit 14727. This permit mentions a number of contraindications, precautions and side-effects that practitioners should be aware of. Some basic assumptions surrounding the use of tramadol in the dog derive from rat, mouse or human studies.

The metabolism of tramadol is complex and there is significant pharmaco-volatilisation between individuals or possibly breeds which may explain the variable clinical results anecdotally observed in dogs.

Conclusions drawn from efficacy studies in dogs may be confounded by the use of concurrent analgesics.

There needs to be more work done regarding the safety of concurrent NSAIDs and tramadol in my opinion. There are papers involving rats, humans and dogs that give cause for concern.

There is a spectrum of adverse events reported to be associated with the use of tramadol in the human literature that gives cause for concern regarding the safe prescribing of the drug by veterinarians.

With these issues in mind I support Aine in encouraging practitioners to look critically at the use of tramadol in dogs and to examine the evidence themselves before deciding upon its continued use within their practices.

(The author is an employee of Zoetis Australia Pty Ltd however the opinions expressed within the above text are the private opinions of the author and not the opinions of Zoetis)

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DIAGNOSIS:

Provided by an imaging specialist: Idiopathic hepato-biliary mineralisation – almost always an incidental finding and unrelated to the reason for presentation. Wedge biopsy would be required to characterise the location of mineralisation better; however, in most cases like this, dogs are well and further investigation is unwarranted.

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Background history for the case: ‘Billy’ was a 12yo entire male Dachshund, presented for work up of an inspiratory wheeze, coughing and a dental procedure. Routine bloods were run, revealing a mild increase in ALKP = 220, with no other abnormalities on the CEO/biochem. Thoracic radiographs where also performed revealing a diffuse radio-opaque change to the liver.
On 4 December 2013, a 7-week-old female DSH rescue kitten (in care) came in with a history of furiously pawing at her face for 24 hours. She had been vaccinated with an unknown vaccine (F3) by the head carer 1 week prior. At this point, she had most erythematous skin on either side of her face and evidence of self trauma. There was ulceration of her tongue suspicious for Calicivirus infection. There was no history of access to any caustic substance. No fleas were seen. She was started on doxycycline paste approximately 8 mg once a day (1/3 notch) and an E-collar placed.

The next day she came back in as was much worse; she’d been furiously ripping at the E-collar then furiously clawing at her face overnight which was extremely distressing for all to see.

A presumptive diagnosis of orofacial pain syndrome associated with Calicivirus was made.

Adding in famciclovir 1/4 tablet once a day was easy but I was a little uncertain about dosing with opiates with such a little kitten. I discussed the case with Andrea Harvey at SASH and we decided that buprenorphine 0.05mL (20ug/kg) would be good so the one mL ampoule was drawn up in to 20 x 0.3mL 100iu/mL insulin syringes. 5 units in each. I showed the carer how to break off the needle, check that the end felt smooth and then to squirt the contents into the side of the mouth.

The final regime for medication was:-
- famciclovir 125mg 1/4 tablet once a day 8 days
- doxycycline 100mg/g paste 1/3 notch (approx 80mg) once a day 2 weeks
- buprenorphine 20ug/kg twice a day 10 days
- phenobarbitone 3mg twice a day 10 days

Thankfully this combination worked really well and one week later the kitten was MUCH improved and there was no relapse as the medications were dispensed.
This article describes 2 unrelated cases of FeLV-like disease which initially tested positive for FeLV but subsequently, on further work up, were determined to be FeLV negative. They are a powerful reminder of the need for confirmatory testing whenever FeLV infection is suspected.

CASE 1 (Marshall Thornton):

‘Skittles’ was a 3-year-old male neutered DSH. He presented with a 4-day history of weakness, lethargy and inappetence. The owner works for a local private zoo, and regularly has snakes, reptiles, birds, and mammals under care at home. Skittles had been an indoor only cat for about 6 months; however, prior to this, with another owner, he had been allowed outdoor access and had a history of fighting. He was the only cat in the house.

On physical examination, Skittles was depressed, had white mucous membranes with indiscernible capillary refill, was painful right kidney, including ultrasonographic imaging, was discharged with a grave prognosis. Further work-up of his clinical history (3 yrs). There has been a loose association of this condition with FeLV but not in every case. These cats may have enlarged spleens with normal sized lymph nodes.’

Blood film (Dr Doug Hayward):

‘Initial assessment is suggestive for the presence of erythroleukaemia given the identified rubriblasts and increased nucleated real blood cells despite marked non-regenerative anaemia. Further considerations, however, include IMHA and/or haemotropic Mycoplasma infection given the identified rubriblasts and increased nucleated real blood cells despite marked non-regenerative anaemia. Further considerations, however, include IMHA and/or haemotropic Mycoplasma infection along with close monitoring of the FBC and platelet counts are warranted.

Comments (Dr K Todhunter):

‘There is a severe non-regenerative anaemia with large numbers of erythroid precursors including blasts in the peripheral blood strongly suggestive of Acute Erythroleukaemia. Some aspects of the haematology fit including the mild thrombocytopenia, neutropaenia, and mild lymphocytosis along with the age (3 yrs). There has been a loose association of this condition with FeLV but not in every case. These cats often have enlarged spleens with normal sized lymph nodes.’

Skittles was much brighter the following day but was discharged with a grave prognosis. Further work-up of his painful right kidney, including ultrasonographic imaging, was declined by the owner. Skittles was well at home for a few days; however, he deteriorated quickly and 8 days later was euthanased.

CASE 2 (Marshall Thornton):

‘Tibbles’ was a 4½-year-old male neutered DSH. Coincidentally, Tibbles had been adopted from the same local pet store as Skittles, however several years apart. Tibbles presented with a recent history of weight loss, lethargy, reduced appetite and aggression. Tibbles was an indoor-only cat and the only cat in the house.

On physical examination, Tibbles had pale pink mucous membranes, delayed capillary refill (4 seconds), tachycardia (116/200) with an intermittent right systolic murmur and pyrexia (rectal temperature 40.5°C).

While waiting for results from Vetnostics, I started treating the life-threatening anaemia with a fresh blood transfusion. A friend of the owner supplied a healthy 4.5kg cat to be the blood donor. I used a 60mL syringe containing 6mL of citrate phosphate dextrose anticoagulant which I took from a 450mL blood collection bag. I collected 45mL of blood from the donor cat’s jugular vein under light sedation with alfaxalone (Alfaxan CD®, Jurox). The blood was transfused to a freshly emptied 100mL 0.9% NaCl bag. Skittles was transfused at 25mL/hr using a Nikko infusion pump via a catheter in his left cephalic vein. I also administered 1.25mg desamethasone SC (Dexapent®, Ilium, 0.25mL) and 25mg enrofloxacin SC (Enrotril®, Ilium, 0.5mL).

The following morning I received the following report from Vetnostics:

- FeLV antigen POSITIVE (serology, Virachek, Symbiotics Corporation).

Following discussion with the owners, euthanasia was elected based on the poor prognosis for Tibbles.

Around the time that these cases presented to me, I became aware of a research group at the University of Sydney developing a real-time polymerase chain reaction (PCR) for the diagnosis of FeLV. I sent fresh blood samples from both Skittles and Tibbles to Mark Westman and Jacqui Norris. Unfortunately, because the PCR was still being developed I did not receive results for Skittles and Tibbles until a few weeks later, after both had already been euthanised. When the results came through I was surprised to find that both cats tested negative for FeLV on PCR.

FeLV Testing – What and Why (Mark Westman)

All in-clinic FeLV test kits detect viral antigen in blood, whether whole blood, plasma or serum. The target of these test kits is the viral capsid protein p27. These tests are highly sensitive, detecting close to 100% of viraemic cats. However, there are 2 main issues associated with these tests:-

1. The low prevalence of FeLV in Australia (<2%) results in a low positive predictive value (PPV) for the test kits – thus false positive results occur as frequently as true positive results.

2. Approximately 60% of cats exposed to FeLV will become regressively infected, meaning they are not viraemic but are still infected with the virus. The only way to detect regressive infections is by using PCR to look for proviral material (DNA copies of the virus inserted into the host genome) in circulating leukocytes.

The significance of regressive FeLV infections on feline health is still largely unknown.
‘Lacy’, a nearly 13-year-old fat Cattle Dog Cross neutered male presented to the clinic for a couple of episodes of being wobbly and falling over in the last 2-4hrs. The episodes were not related to exercise or eating, but did appear to happen after a period of resting. The dog tried to rise and get moving again. Appetite normal and demeanour normal between episodes. Lacy’s on a ½ a Previcoxs tablet 22mg daily.

Physical examination showed moderate degenerative joint disease in the hips and elbows with suboptimal flexion and pain on extension of these joints. Stiffness were both thickened but had good range of motion. There was some mild spinal pain, especially around the lumbosacral region. Lacy is a very nervous dog who has been on daily Valium for some time (prescribed by a previous vet) for persistent anxiety. HR was 130 and RR panting, but it was difficult to ascertain what was ‘nerves’ and what was pathological. Chest auscultated well and clear but femoral pulses were reduced and the femoral pulse felt weaker than I would have expected in that sized dog. Mucous membranes were pale pink (maybe slightly icteric with a bit of imagination?) with a 2s capillary refill. Abdominal palpation was difficult due to the dog being overweight, and also guarding. Temperature was normal. Bloods normal, no vomiting.

I originally thought it may have been a decompenstating diated cardiomyopathy, so lateral and ventrodorsal radiographs were taken, but showed a normal cardiac silhouette and a mild bronchial pattern in the lungs attributable to age. Next step, in the absence of any other bright ideas, was bloods. EDTA and Clot tubes sent to Vetnostics for a Body Function profile.

Blood results are as follows (abnormals only):-

- Hb 92 (115 – 180)
- RCC 2.8 (5-8)
- Hot 0.27 (0.37 – 0.55)
- Urea 10.2 (2.5-9.0)
- Phos 0.4 (0.8-2)

Now the only other time I have seen a crazy low phosphate was in a severely ketoceticotic cat. Low phosphate can cause muscular weakness, so was likely the cause of the ‘wobbly’ episodes, and can also affect the heart muscle reducing cardiac output. The slightly increased urea may be attributable to this. It can also cause haemolysis, though the serum at the time of collection was clear. It was a bit alarming and the owner was called immediately and requested to come in and start oral CoPhos 2 mls EOD. Sue Foster (love that vet) from Vetnostics has a special interest in hypopH cases and kindly spoke with us at length about possibilities and further testing. A fractional electrolyte excretion of the kidneys was recommended as KNaMar treats back in 2009 were found to cause a proximal tubular nephropathy which was fatal. A thorough diet history was also recommended.

Two days later Lacy returned and bloods and urine were taken for fractional excretion of electrolytes – which came back normal. No sick kidneys then… it was recommended that we retest the P levels in the bloods in 10 days.

Ten days later, repeat bloods showed a Phos of 1.06, so normal, although the owner mentioned that Lacy is still weak at times, though not as bad, but does seem to have spells where the gums go pale. PCV had increased to 30 and the serum was clear as well. The owner was not interested in pursuing any heart workup post X-rays, so it will remain a mystery as to why Lacy went hypophosphataemic, or whether there is a heart component here as well. However, the treatment appears to be working and we will continue to follow Lacy closely.

**Follow-up:**

Routine bloods were taken on 16.12.14 to follow up how Lacy was going on the CoPhos paste every second day. The owner reported that Lacy was well, and that the dietary management to a more meaty diet was agreeing with her.

Results showed Phos 1.19 (0.8-2.0) and other parameters normal. PCV was 40.

However, in early March (9) his owner reported that Lacy had been having more episodes of ‘weakness’ and had been found unconscious the previous afternoon. Bloods were taken for a full body function this time, and abnormal results were as follows:-

- Hb 97 (115-180)
- RCC 4.0 (5-8)
- Urea 0.71 (0.8-2.0)
- Ca 2.57

The Ca was WNL at 2.42 (2.2-2.6) and the retic count was 1.1% abs 44, so not so regenerative, sadly.

So Lacy was trans fused the next week at the owner’s request, with 1 bag of greyhound blood. It was tolerated well and she was moved to daily CoPhos paste and had clinically been managing well.

Repeat bloods on the 14.4.15 showed similar findings:-

- Hb 98
- RCC 3.8
- Urea 10.2
- Ca 2.57

So Lacy was moved to daily CoPhos 2mLs and bloods taken a week later where…

- All was WNL! Phos 1.06

So, it would appear Lacy either is a poor absorber of Phosphate dietarily, or is losing it from somewhere we can’t find… either way, a high Phosphate diet and the addition of CoPhos supplement seems to be enough to hold him for now. Also, the anaemia seems to be holding for now… so not sure if the low Phosphate is causing the anaemia, or has a secondary issue? A bit of a conundrum poor Lacy, but it’s great that he has responded so well to supplementation and dietary management.
LOCALISED TICK PARALYSIS IN A GOLDEN RETRIEVER; A LESSON IN FOLLOWING ONE’S OWN ADVICE

C&T NO. 5475

Heather Shortridge
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Several years ago, I took my then 22-month-old male entire Golden Retriever ‘Pickle’ to the coast for a long weekend. Based in Armidale we didn’t normally have to worry about ticks, but the day before our trip to the coast I applied a dose of Frontline Plus® (Fipronil and S-Methoprene) to Pickle. We had a good weekend at the coast in September and returned to our normal habits in Armidale.

About 3½ weeks after our trip to the coast I noticed that Pickle’s lower jaws were sagging, revealing the inside of his gums. Pickle was subjected to a bit of poking and prodding of his gums but I couldn’t really determine what the issue was.

Several days later I happened to be patting Pickle under his chin and found a large paralysis tick (approx 1cm long) there! I removed the tick and Pickle’s jaws returned to their normal position over the next few days.

It seems that Pickle had a localised case of tick paralysis. This may be seen commonly by those who work in areas with lots of paralysis ticks, but having worked in mostly non-tick environments it came as a surprise to me.

By the time I removed the tick, it must have been there for 4 weeks, during which time Pickle had been eating normally, and being subjected to regular vigorous exercise.

The tick itself may have been slowed in its activity by the dose of Frontline® before it latched on, but it was very engorged by the time we removed it.

Fortunately, Pickle had no complications from his tick and we have now moved to Canberra so we are again living in a low risk area!

The author pictured with ‘Pickle’.

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**SOLUTION:**

Radiographic findings:
1. There is irregular bone proliferation on the ventral aspect of vertebral bodies of L6 and L7 extending across the intervertebral disc space at this level, as well as on the caudoventral margin of L5.
2. The margins of the end plates of the vertebral bodies appear within normal limits.

Conclusion
Osteoproliferative change on the caudal lumbar vertebral bodies - suspect reactive periostitis associated with metastatic spread from lower ureteral uretral or perineal/perineal neoplasm.

Comments & Follow-Up:
The reactive change is unusual in that it is primarily associated with the ventral aspect of the vertebral bodies AND the vertebral endplates are smooth and very normal. This is in contrast with vertebral disorders that it is commonly mistaken to be:

- Spondylosis deformans (see Figure 2 solution), which is typically associated with the ventral aspects of the vertebral endplates, the bone proliferation is smooth and well defined. In advanced stages it can bridge the vertebra completely.
- Discospondylitis (see Figure 3 solution)- this infection associated with the intervertebral disc extending into the adjacent vertebral endplates. The endplates are irregular with a mix of lucencies and opacities indicative of the lysis and proliferation of bone associated with the reaction to the presence of infection. In this case example the intervertebral disc space has collapsed.

The change evident on the vertebrae in this patient is more typical of a reactive change that occurs with metastatic neoplasia (typically carcinoma) along the fascial planes and adjacent periosteum of the vertebrae when tumours are present in the lower ureteral tract or in the perineal region. In some cases the reaction can include the sacrum, pelvis and even the proximal femurs.

Based upon the radiographic features and despite the dog being a castrate, I would recommend careful evaluation of the lower ureteral tract and perineal region for a mass effect. Further diagnostic tests to consider include rectal palpation to evaluate the caudal pelvic canal for evidence of urethral or prostatic mass, rectal mass or anal gland nodule/mass. Ultrasound examination of the kidneys, ureters, urinary bladder, regional lymph nodes (para-aortic/sublumbar lymphocentres) and prostate should also be performed. Ultrasound-guided aspirate of the region adjacent to the reactive change could be undertaken also for definitive determination of the underlying aetiology of the reactive change if no other (primary) lesion is identified, although in my experience the primary lesion is usually found, so direct aspirate from the soft issue adjacent to the reactive change in the vertebrae is rarely required. The prognosis is obviously dependent upon the underlying aetiology of the reactive change, hence the requirement for further ultrasound evaluation.

Outcome:
This dog was found to have prostatic carcinoma.

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**C& T NO. 5478**

**C T DIAGNOSTIC IMAGING – WHAT’S YOUR DIAGNOSIS?**

**Signalement:** 8 years old male neutered Setter

**History & Clinical Findings:** Initially presented for a one week sudden onset history of hindlimb weakness and episodic yelping. On physical examination the dog had mild lumbar pain which responded to corticosteroid treatment initially, but after two weeks, pain returned and seemed worse.

- What radiographic changes/abnormalities are evident in the two views provided?
- What is your diagnosis?
- What would you do next?

**Robert Nicoll** BSc (Vet) BVSc DACVR

Prior to specialising in diagnostic imaging, Dr Robert Nicoll worked in mixed veterinary practice in Bathurst, NSW for several years. After undertaking his residency training at the University of Wisconsin, Madison, USA, he returned to Australia. With Graeme Allan he formed Veterinary Imaging Associates and more recently, their teleradiology practice Online-Vets.com, providing an international diagnostic service. Since 1998, Robert has been an associate tutor with Graeme in the Diagnostic Imaging Distance Education course and has worked with Graeme on developing a special digital radiography stream for those who have made or are looking to make the leap into filmless radiography.

**DiAGNoSTiC IMAGiNg**

The CVE is keen to encourage our Members/Readers to embrace the ebook version of the quarterly C&T, as it’s a great complement to the print version and allows the inclusion of multi-media. To encourage reading of our C&T eBook, Robert and Graeme, tutors for the CVE’s highly regarded Diagnostic Imaging DE program, will be supplying a Question each quarter, with the Answer available only in the complementary ebook version.

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In human medicine, the reliance on abdominal radiography has waned. Abdominal radiography as a first-line test is recommended following clinical examination in only a few scenarios: suspected small intestinal obstruction, and perforation (although with the latter, the thorax is radiographed with the abdomen included on the edge of the field, so just the ‘top’ of the abdomen around the liver and diaphragm is assessed). Reasons for this are two-fold: firstly, abdominal radiography lacks sensitivity/specificity in many tests (i.e. other tests are better) and secondly there is a trend away from unnecessary radiation exposure. Alternative tests include ultrasound (but realistically, in people usefulness is limited by how ‘fat’ humans are) CT (which poses a higher radiation dose) and sophisticated blood analysis.

The human emergency department of a large human hospital is hardly comparable to modern veterinary practice, because tertiary human facilities have access (often 24 hours a day) to specialist radiologist staff and facilities incorporating XR, CT and US services. The situation remains different in veterinary medicine. Whilst the numbers of equivalent veterinary radiologists/internists has increased access to these services can be limited particularly out of hours.

There is no doubt that abdominal ultrasound is a highly useful modality in veterinary practice, particularly for non-GIT disease like pancreatitis, pyometra and determining the origin/extent of masses. It does require significant operator experience (examples include performing a thorough hepatic assessment or assessment of pancreatitis affecting the left limb or pancreatic body and high-spec equipment. Finally, it takes years to become competent at the whole-abdomen ultrasound examination given variations in patient species and size. Evaluation of the gastrointestinal tract and the right cranial quadrant of the abdomen (gall bladder fossa, the pyloroduodenal junction, the pyloric and pyloric outflow obstructions ultrasound can be superior to US and CT and more rapidly performed with results in less than 20 minutes. Finally, it can give a rapid global assessment of the state of the abdomen, particularly useful for assessing the retroperitoneal space, edge of the thorax and urinary tract rupture (radiographic contrast study).

So the question for veterinary medicine is, if we are to maximise the benefit radiography in our patients, which cases should have abdominal radiography? Re-phrased: when is it in the best interests of the patient to perform a quick, simple, relatively cheap test possibly with lower sensitivity (radiography) than a more expensive, less available test, possibly with higher sensitivity?

My firm opinion is that the following scenarios warrant immediate abdominal radiography:

1. Any patient where a gastric dilatation/volvulus (GDV) could be present.
2. Severe or protracted vomiting, where there is a suspicion from the signalment or history that a foreign body or intussusception may be causing intestinal obstruction.
3. ‘Acute abdomen’ scenario resulting in severe abdominal pain or collapse: in this case to exclude obvious pnenomoperitoneum (e.g. in hepatic abscessation or intestinal perforation). Small volumes of free gas are difficult to detect with abdominal US, and confirming the site of intestinal perforation may be very difficult. (Figure 1).

This mirrors (incidentally) the recommendations of the public health system in WA.¹

With GDV, radiography is diagnostic. With intestinal obstruction, there are well documented objective radiographic criteria that are frequently helpful in determining whether the dog or cat should proceed to exploratory laparotomy (refer to standard texts and see Figure 2). Unfortunately, radiography is not always definitive for the presence of obstruction; in these cases if vomiting persists, repeat the radiographs after 6-12 hours of symptomatic treatment to check for progression, or perform ultrasound or exploratory laparotomy if indicated.

Whilst not specifically related to vomiting, abdominal radiography remains the first test of choice to accurately localise the site of urinary tract rupture. You may have clues that there is a urinary tract rupture from peritoneal fluid analysis or decreased urine output, but a contrast study (retrograde urethrogram/cystogram for the bladder/urethra, and excretory urogram for the kidney/ureter) is required to determine the site of the rupture.

So for the vomiting dog or cat, my message is:

• Don’t be afraid to radiograph the abdomen for any case of vomiting where there is a concern that obstruction may be present.
• Radiography is always the best test to rule out a GDV. Because GDV can be catastrophic, radiography should always be considered in susceptible breeds showing vomiting or abdominal distension.
• Radiograph an ‘acute abdomen’ – it may give provide valuable information and you can do it whilst you are waiting for the results of the haematology/biochemistry panel.
Once committed to abdominal radiographs, do them properly. Sedation is usually required for a diagnostic study. Butorphanol (or another opioid) +/- low-dose acepromazine is a safe, effective combination. For a GDV, obtain a single R lateral projection – it is usually diagnostic. For other studies always take 2 orthogonal views.

- R lateral and VD
- Obtain the same lateral each time
- VD always preferable to DV for better quality image

Remember, if in doubt you can get a second opinion usually within a few hours from a tele radiography service. Embrace the digital world!

References


Personal comment from Richard Malik: My own view is that GF vets need to change the way they cost out things. Most clinics have digital radiology – so taking radiographs is quick, easy, cheap (low marginal cost) and they are easy to send off for a second opinion. Our ultrasound skills vary, and we are not nearly as good as radiologists. So why not always do both, get the most information we can, and charge it out at a fee most clients can afford. It seems silly to me not to take a radiograph (or two) that will take 5 minutes to do, to complement each and every ultrasound exam.

Feline plasma cell pododermatitis is an uncommon but well-recognised condition in cats, which is described in all dermatology and pathology textbooks. Yet the aetiology is poorly understood and a search of the veterinary literature reveals a relative lack of published information on this fascinating feline disease.

Affected cats present with purple, spongy swelling of one or multiple paw pads, typically the metacarpal and/or metatarsal pads, which are often criss-crossed by linear striations giving the pad a characteristic gross appearance. Lesions often then progress to ulceration with secondary superficial bacterial infection, and the presence of pain and associated lameness appears to vary between cases. A classic entry on the histopathology submission form – ‘the foot pad has exploded’.

The particular case described here had a brief clinical history of swollen pads affecting all 4 feet, together with involvement of the nose – a finding also occasionally described in the literature. This cat is a female neutered, 8-year-2-month-old Domestic short-hair, and the tissues biopsied were taken from a swollen right fore pad. The histological appearance of the tissue submitted is entirely typical for this disease, with an intense, predominantly mononuclear inflammatory cell infiltrate expanding the dermis as well as the underlying adipose tissue. The infiltrate is an almost entirely monomorphic population of well-differentiated plasma cells, including Mott cells (Figure 1) – Mott cells are plasma cells that have spherical inclusions within their cytoplasm (called Russell bodies) which correspond to accumulations of immunoglobulins (antibodies). Low numbers of small lymphocytes and neutrophils are observed.
This apparent gender imbalance is also seen in some of the published studies, albeit based on smaller numbers. For example, Guaguere et al. (2004) reported 19 out of 26 cases as male, while Scarampella and Ordeix (2004) had 8 male cats out of ten cases. Bettleay et al. (2003) had 11 males out of 15 cases where the gender was recorded. In our 176 cases, this apparent gender imbalance is statistically significant ($p=0.0085$).

Age was recorded for 170 of our cases, with a median age of 6 years and a range from 9 months up to 17 years (see Graph 1); there is an apparent tendency for this condition to affect young to middle-aged cats, although there is a huge variation in the ages affected. These findings agree with those in other published studies, including Bettleay et al. (2003) where the mean age was 5 years, with a range from 1 to 12.5 years, and Pereira & Faustino (2003) with a mean age 4.7 years and a range from 3 to 7 years. Scarampella & Ordeix (2004) had cases ranging from 6 months to 18 years, and Guaguere et al. (2004) from 6 months to 12 years.

For those cats in our records where the breed was noted (180 cases), all bar 11 were domestic short haired, domestic long haired, ‘domestic cat’ or ‘cross-breed’.1) The 11 pedigree cats included 5 Siamese (2.7%), and 1 each of (0.6%) of Bengal, Ragdoll, Burmese, British short hair, Persian and Abyssinian. The breed prevalence of the background feline population (based on the cat breeds recorded for over 3,000 fixed tissue specimens submitted to the laboratory over this same time span) is 60.9% non-pedigree (domestic short haired, domestic long haired, ‘domestic cat’ or ‘cross-bred’), with 1.8% Siamese, 1.1% Burmese, 2.2% Persian, 0.7% Ragdoll, 0.3% Abyssinian, 0.2% British short hair and 0.6% Bengal.

In terms of concurrent diseases (biopsies submitted at the same time as those from the feet) 5 cases had plasmacytic stomatitis (gingivitis, and 2 had eosinophilic granuloma type lesions in the oral cavity – since these are both in themselves fairly common conditions within the cat population, this is probably co- incidental. The potential link with FIV infection is very interesting; although obviously many cats with plasma cell pododermatitis do not have FIV, it is feasible that a disease resulting in altered immune function could also produce errant B-lymphocyte and plasma cell behaviour. In a paper describing 6 experimentally infected cats (Simón et al. 1993), 4 had evidence of plasma cell pododermatitis, and in 1 case they performed immunohistochemical staining on the pad tissues and found FIV-immunoreactive cells in situ. Scarampella & Ordeix (2004) reported 4 out of 9 cases were FIV-positive, while Guaguere et al. (2004) had 16 FIV-positive cats out of 26 cases, together with one FeLV-positive cat. In humans infected with human immunodeficiency virus (HIV), B-lymphocytes show signs of phenotypic and functional alterations, such as polyclonal B-cell activation, loss of B-cell memory and hypergammaglobulinemia; it is interesting to speculate whether FIV produces similar effects in feline B-lymphocytes, leading to accumulation of functionally abnormal plasma cells within tissues such as the pads, as well as the hypergammaglobulinemia described by Scarampella and Ordeix (2004).
Several diseases in humans are hypothesised to act in this way. The presence of mitotic figures and atypical bi-nucleated and multinucleated cells (suggesting nuclear division in the absence of successful cell division). These are features described by Diz-Pereira & Faustino (2000) and also noted in the present case (see Figures 2 - 4).

However, plasma cells are supposedly terminally differentiated, non-dividing cells derived from B-cells, so how is this happening? It may be that the dividing cells we see in the sections are actually plasmacytomas, a developmental ‘half-way house’ between a B-lymphocyte and a fully mature plasma cell – plasmacytomas are capable of secreting antibodies but can also still divide and act as antigen-presenting cells. Alternatively, it is possible that these are fully mature plasma cells which are capable of self-replication – but this would be a rather aberrant behaviour for a plasma cell. Additionally, mature plasma cells are not supposed to have a very long life span (with the exception of some special subtypes), in which case their presence in such large numbers is even more impressive.

In human studies looking at rheumatoid arthritis, bi- and multinucleated plasma cells (and some containing mitotic figures) have been identified within the affected synovial tissues (Perry et al 1997). The authors suggest these cells are undergoing cell division and that this is at least partly contributing to their accumulation in such large numbers within the synovium. Whatever the precise underlying mechanism, there is also something abnormal about the plasma cells we see in feline plasma cell pododermatitis.

Hypothesis number 1: this disease could be due at least in part to an infectious agent within the pads or pads. However, does the fairly common involvement of multiple feet imply that infection of 1 pad can trigger the development of disease in the others? Maybe the infection itself is fairly transient, and merely initiates a localised immune response within the pads. Some forms of trauma can have a similar affect, effectively triggering a form of auto-immune due to exposure of tissue components not normally seen by the immune system and therefore not recognised as ‘self’. If the tissue component targeted by this auto-immune attack is found only within the pads (and none, for example a particular subtype of collagen, then that would explain why the resulting disease affects the pads alone.

Hypothesis number 2: this disease could be due at least in part to an infectious agent, but the agent is not present within the pad but elsewhere – such as the gastrointestinal tract. Several diseases in humans are hypothesised to act in this way. The best understood is ankylosing spondylitis, where a link has been demonstrated between a particular MHC gene (HLA-B27), a high starch diet and the presence of Klebsiella pneumoniae within the intestinal tract (see Rashid et al. 2013 for a fascinating review) producing the disease itself at another anatomical location. Another human example is rheumatoid arthritis, in conjunction with often subclinical infections with Proteus mirabilis within the urinary tract (Ebringer and Rashid 2006, 2014). The proposed mechanism in these diseases is ‘molecular mimicry’ - bacterial antigens which are similar in structure to normal tissue components stimulate production of antibodies which not only target the bacteria but also demonstrate cross-reactivity with self-antigens. This results in auto-immune disease at distant but specific anatomical sites.

Hypothesis number 3: this could be a non-infectious, purely immune-mediated disease, possibly targeting self-antigen located specifically within the pads and nose. The plasma cells within these pads are interesting in their own right, in that they appear to be actively dividing in situ – as indicated by the presence of mitotic figures and atypical bi-nucleated and multinucleated cells (suggesting nuclear division in the absence of successful cell division). These are features described by Diz-Pereira & Faustino (2000) and also noted in the present case (see Figures 2 - 4).

Since doxycycline is both an antibiotic and an immunomodulatory drug, the response of cases to doxycycline therapy could indicate 1 of 2 things (or possibly both) – either there is some bacterial involvement (as yet to be found e.g. bacterial L-form), and/or this is an abnormal immune response...
While dairy goats generally get the bovine strain of Johne’s disease (also called paratuberculosis),2 they often do not show the typical signs that are observed in cattle i.e. watery diarrhoea, dehydration and thickened, corrugated intestines. In goats, the main clinical sign of Johne’s disease is just diarrhoea, dehydration and thickened, corrugated intestines. In goats, the main clinical sign of Johne’s disease is just diarrhoea, dehydration and thickened, corrugated intestines.

Table below:

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of goats affected</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Johne’s disease</td>
<td>19</td>
<td>28</td>
</tr>
<tr>
<td>Johne’s + another disease</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td>Digestive disorders  e.g. entero-toxaemia, acidosis, boil</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td>Parasitic gastro-enteritis</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Gut torsion</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>10</td>
<td>16</td>
</tr>
<tr>
<td>No diagnosis</td>
<td>5</td>
<td>8</td>
</tr>
</tbody>
</table>

In a study of goats herds in Norway, PCR tests were performed on bulk milk samples. It was found that 3.3% of herds which had previous Johne’s disease cases had positive PCRs, but for herds with no history of Johne’s disease there was a 0.1% positive rate for PCRs.26 This higher level in supposedly “normal” herds indicated that the diagnosis of Johne’s disease had been missed in these latter herds.

Clinical signs reported in goats with Johne’s disease include:-

- Loss of body weight
- Poor, dry coat
- Inflammation despite access to plentiful feed
- Muscle wasting, rather than fat loss

And, less often, there are the following clinical signs and gross signs on post-mortem examination.

- Apathy
- Dehydration
- Clumping of faeces
- Enlarged ileo-caecal lymph node
- Enlarged mesenteric lymph nodes
- Caseous nodules in mesenteric lymph nodes
- Thickened caudal ileum

A study in a large US goat herd with a high incidence of Johne’s disease 19 found the following gross signs during 120 post-mortems on infected goats:-

- Thickened ileo-caecal valve and ileum (49/120)
- Corrugated ileum (34/120)
- Inflamed ileum (12/120)
- Haemorrhagic ileum (4/120)
- Abscessed ileum (1/120)
- Changes in ileo-caecal or mesenteric lymph nodes (45/120)

These lymph nodes 3 times normal size or larger (33/120).

- These lymph nodes are oedematous (6/120)
- These lymph nodes are large and haemorrhagic (1/120)
- These lymph nodes are large and mineralized (1/120)
- These lymph nodes are hyperaemic (1/120)

However, a large number of goats with positive diagnostic tests for Johne’s disease had no gross signs at all on necropsy. Also, there is 1 report of a goat with nervous signs from a hepatic encephalopathy in a goat with Johne’s disease.5 This 3-year-old dairy goat had lost weight and become weak in the previous 2 weeks before being presented for veterinary examination. This doe also had depression, posterior weakness, fine head tremors, and intermittent trembling of the head and neck. The goat’s blood ammonia levels were extremely high and on post-mortem the liver showed fatty infiltration. This liver disease, due to the goat’s inappetence and wasting because of the Johne’s disease, caused the neurological signs. Most vets would not include Johne’s disease on a differential diagnoses list for neurological diseases.

Prevention of the introduction of Johne’s disease is by ensuring that all new introductions of goats are from herds that are accredited tested negative of Johne’s disease. Such herds are in a searchable database on the internet (www.edis.animalhealthaustralia.com.au/public?pg=site-mapsresearcha_ha_program–3). However, as at 26/5/15 there were only 24 Australian goat herds in the goat Johne’s disease market assurance program. In countries without such a scheme, the only option is to close their herd and only introduce new genetics by artificial breeding.32 These closed herds must also ensure no cattle, deer or alpacas or their products (manure, milk) are introduced.

Control of Johne’s disease in an already infected herd is very difficult but would involve improving general hygiene and separation of kids at birth. General hygiene includes using fenceline feeders or keyhole feeders so that there is no faecal contamination of the goats’ feed. It has also been suggested that Mycobacterium avium subspecies paratuberculosis (Map) bacteria can survive for many weeks in baits in livestock waterers8 and this has led to a US Department of Agriculture recommendation to chlorinate drinking water or add 3-10 tablespoons of chlorine bleach to 100 gallons of livestock trough water every week combined with only using galvanized metal or stainless steel water troughs. Separate boots for kid rearing areas and adult goats is also desirable, as is frequent cleaning of murren for composting for 12 months. Monthly weighing of goats to pick up cases of Johne’s disease has been used overseas, where access to other diagnostic tests has been too difficult or too expensive. As wild rabbits have been shown to have the Johne’s disease organism, rabbit control should be part of any control program.10,11a

The Goat Milk Producers Federation of the UK have a Code of Best Practice for controlling Johne’s disease and this involves annual or more frequent testing of all replacement does, never feeding bulk unpasteurised milk, rearing kids in isolation from adult goats for the first 6 months, vaccination of all young kids, necropsies of all wasting goats and routine PCR testing of bulk milk samples.2 Norway have taken a different approach and have eliminated Johne’s disease, Caprine Arthritis Encephalitis (CAE) and Caseous Lymphadenitis (CLA) from herds by snatching-birthing kids, rearing separately on cows’ colostrum and milk replacer and then replacing the original adult herd after thorough disinfection of the barns and milking areas.29

Parasitology times and temperature treatments for colistin that kill the CAE virus (i.e. 1 hour at 56 degrees F) does not necessarily kill Mycobacterium avium subspecies paratuberculosis (Map) bacteria. The temperature needed may be higher i.e. 135 degrees For 30 minutes with streptomycin.16 It has been shown that cows’ colistin pasteurized to kill the Johne’s disease bacteria will coagulate but this can be overcome by adding water and using a blender and the resultant colistin is still useful for feeding to calves.52

A key component to stopping the spread of Johne’s disease in Australian goats is the national kid rearing plan, (www.animalhealthaustralia.com.au/national/kid/rearing/plan) with its recommendation to never feed bulk milk to kids and to rear kids on milk replacer or pasteurised bulk milk. The importance of this recommendation is demonstrated by the trial results reported by Storset et al (2001).16 In this trial, 7 goats were given suspensions of Mycobacterium avium subspecies paratuberculosis in milk replacer 5 times a week between 5-8 weeks of age for a period of 9 weeks. All these goats developed a cell mediated immune response to Johne’s disease, compared to none of the 6 controls, although 2 animals did not develop significant antibody levels. These 7 kids were then observed for 2 years. Two goats started faecal shedding at 12 months and another by 2 years of age. When the goats were necropsied at the end of the trial, 5 of the 7 goats had lesions and the bacteria in the intestines and/ or the mesenteric lymph nodes. However, 2 animals had no detectable lesions in the distal ileum and colon on necropsy. Admittedly 2 years is a short period for incubating Johne’s disease, as often clinical signs do not appear for some years. However, this research shows that subclinical infections i.e. carrier, can easily be missed.

This is backed up by the findings of Thomas (1983),7 who tabulated the age of faecal shedding in a large UK goat herd in the 3 years following diagnosis and initiation of control. His results are shown in the table below:-

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Total No. of kids</th>
<th>No. of goats in this age group</th>
<th>% of kids shedding in this age group</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–12 months</td>
<td>149</td>
<td>14</td>
<td>9</td>
</tr>
<tr>
<td>13–18 months</td>
<td>154</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>19–24 months</td>
<td>55</td>
<td>17</td>
<td>12</td>
</tr>
<tr>
<td>&gt;25 months</td>
<td>58</td>
<td>16</td>
<td>53</td>
</tr>
</tbody>
</table>

Frequent testing of all adult goats is needed to remove any faecal shedding as quickly as possible. Faecal culture has a specificity of almost 100% but the test takes several days (8–10) for the bacteria to grow and this time can be even longer in goats (10–12 weeks). Sensitivity is low in the early stages of infection but approaches 100% in the advanced clinical stage. Research has shown that positive culture results were found in 69% of goats with diffuse lesions and from 44.4% of those with focal lesions found on necropsy.8 Research completed by Eamens et al (2007) showed that the faecal culture tests on pooled faecal samples can survive for many weeks in waterbefore the recommended dilution was 1 in 25 and a time frame for incubation. This dilution collected 13 out of 16 positive (3 with the sheep strain and 13 with the bovine strain) goats. This dilution was best for reducing costs without a large drop.
in sensitivity. Two low shedders were not detected except by individual culture.

Fecal smears stained with Ziehl-Neelsen can give a rapid result, but have low sensitivity and specificity. It has been suggested that fecal smears can only be used in advanced clinical cases as an interim diagnosis. False positives can occur if the environment is heavily contaminated by faeces from heavy shedders. There is also a skin test with an intradermal injection of johnin, similar to the tuberculin test, but this test has only very limited value.

Initially, Complement-Fixation or CF Tests were used in goats and this was the test used when Johne’s disease was first reported in Australian goats. Then Agar Gel Immunodiffusion or AGID tests were developed as the diagnostic test for goats e.g. when 331 goats were tested in Western Australia as part of the surveillance for proof of freedom from Johne’s disease. ELISA tests were developed and these have better sensitivity and specificity in cattle than other earlier serological tests. Research in the USA with dairy goats compared ELISA tests using sera and individual milk samples and found that the sensitivity was 64% compared with 48% respectively to fecal cultures. However, a NIROC study compared the AGID and ELISA tests and found the ELISA detected more cases of Johne’s disease in goats. ELISA tests are the serological tests mentioned in the Goat Market Assurance Program or MAP (www.animalhealthaustralia.com.au/wp-content/uploads/2011/04/GoatMAP-manual.pdf). Also mentioned in this MAP is the PCR test, which is a DNA based test and which is now widely used in cattle. A recent study looked at using ultrasonography to detect enlarged mesenteric lymph nodes (94%) and/or thickened intestinal walls (80%).

There is no test that identifies the goats that are in the early stage of incubating Johne’s disease. Goats with Johne’s disease can be classified into four stages as summarized in the table below:

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinical signs</th>
<th>Positive Blood Test Result</th>
<th>Shedding in Faeces</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early incubation/ silent infection</td>
<td>None</td>
<td>None - although may pick up a cellular immune response by inflation gamma assay</td>
<td>No</td>
</tr>
<tr>
<td>Subclinical</td>
<td>none</td>
<td>Possible</td>
<td>Yes but low numbers</td>
</tr>
<tr>
<td>Clinical</td>
<td>Weight loss</td>
<td>Yes</td>
<td>Yes - high numbers</td>
</tr>
<tr>
<td>Advanced</td>
<td>Emaciation, dehydration, clumped faeces, intermittent diarrhoea</td>
<td>Yes</td>
<td>Yes - extremely high numbers</td>
</tr>
</tbody>
</table>

The number of identified goat herds with Johne’s disease and reported by Animal Health Australia are very low and this could be due to the Caprine Arthritis Encephalitis (CAE) control program that involved snatch birthing kids and raising in isolation on milk replacers. However, goats can become infected with Johne’s disease from both cattle and, to a lesser extent, sheep with the disease. All veterinarians dealing with dairy goats in states such as Victoria and Tasmania, where Johne’s disease is prevalent, should keep Johne’s disease on their list of differential diagnoses where there is loss of body condition in adult goats. Don’t just think gastrointestinal parasites. Remember also that the sheep strain, while less likely to infect goats, is also less likely to develop severe clinical signs, sero-conversion and shedding. If doing a necropsy, vets should take the advice of Thomas (1983) i.e. that smears of ileum and mesenteric lymph nodes that are the stained with Ziehl-Neelsen stain should be examined ‘in all post mortem examinations of goats’. The Australian New Zealand Sub-Committee on Animal Health Laboratory Standards recommends that the following samples be taken for both culture and histopathology for diagnosing Johne’s disease:

- Entire ilocaecal valve
- Cecal ileal lymph nodes
- Real (caudal jejunal) lymph nodes
- Two (10 cm) pieces of ileum (one proximal and one distal (terminal))
- One (10cm) piece of proximal colon
- One (10cm) piece of caecum (for histopathology only)

If your goat clients need more information or convincing of the need to necropsy wasting goats, I would recommend they look at this PowerPoint and audio combination about the devastation that Johne’s disease caused in a Saanen goat herd in Chile over many years without any diagnosis. See: www.johnes.org/presentations/Diagnosis/Maine-DVMs-Cases.m4v

Only when an accurate diagnosis is obtained can a goat owner initiate the correct control program and start on the way to recovery. A vaccination program can then be started which will delay clinical signs and shedding, although it won’t prevent cases of Johne’s disease.

References: Due to space constraints, the comprehensive list of reference can be accessed in the eBook version of this issue.

### INTERESTING LINKS

‘The EZ Nabber is simply one of the best tools there is for getting an aggressive or scared small animal out of a cage safely’

www.youtube.com/watch?v=61LF1aJPQ2c

**www.campbellpet.com/products/Handling-and-Restraint/EZ-Nabber**

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