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C&T

CONTROL AND THERAPY SERIES

September 2012 ISSUE 268
Australia's Leading Veterinary Forum

Feature Article

A cat with jaundice

Australia's Poisonous Plants, Fungi and Cyanobacteria

Tick Paralysis - Round Table Discussion

CALL FOR ASSISTANCE - Q fever (*Coxiella burnetii*) – has anyone been sick in your practice?

Centre for Veterinary Education
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CONTROL AND THERAPY SERIES

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B22, Regimental Crescent, The University of Sydney, NSW 2006.
Print Post Approved No. 224792/0012

SEPTEMBER 2012 ISSUE 268 Australia's Leading Veterinary Forum

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BrandQuest
PRINTERS
University Publishing Service
Environmental policy
The C&T is produced using soya-based inks and paper from sustainable forests.
COVER IMAGE
Courtesy of Anne Fawcett

The last three months has seen the passage of the carbon tax bill in Australia as well as the recent changes to asylum seeker legislation. The Olympics in London have come and gone without a hitch, with our individuals and teams doing their best while receiving faint praise in the press at home. Worldwide political and economic uncertainty has continued with hung parliaments, fragile coalitions and ongoing concerns about the Eurozone and unemployment levels in the USA.

In the veterinary industry there appears to be similar turbulence. Many of our universities justifiably claim chronic underfunding and this is impacting on their veterinary schools, where the cost of educating undergraduate vets is under ever increasing scrutiny. Veterinary teaching hospitals must strive for balanced budgets, while teaching and administrative positions are inexorably reduced.

Record numbers of veterinary graduates will flood the market in the coming years, this at a time when we are seeing a steady decline in pet ownership across the nation. Despite the efforts to attract and train students who may be prepared to work outside the large metropolitan areas, the overwhelming majority of graduates will be female and seeking employment in or around the major cities, often on a fractional basis and with time off for maternity leave.

This combination of factors must be unsettling for those who are in the early stages of their careers, as the veterinary industry is subject to the law of supply and demand like any other industry or profession.

At the CVE we continue to strive to present programs and continuing education to take you to a higher level across a broad range of topics. For years observers of the profession have advocated the need for graduates to look at innovative ways to use their degree. While not every vet will establish a business like Flight Centre, win a Nobel prize or become a member of parliament, there is a myriad of pathways that the veterinary degree prepares one for, yet the majority of new graduates still want to go into small animal practice in cities.

In this issue there are numerous articles written by vets doing our Feline Distance Education course. All of the CVE DE courses enable vets of all ages to expand their knowledge of their chosen subject and often to become the go-to person in their practice about a given topic. Many DE participants go on to sit their Membership exams and even progress to Specialist status through the Fellowship or Board system.

Why don't you think about committing to some regular study? Who knows what doors may open for you?

Hugh White BVSc MVSc MACVSc
DIRECTOR

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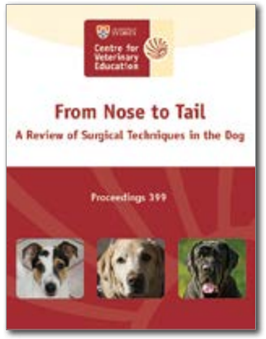

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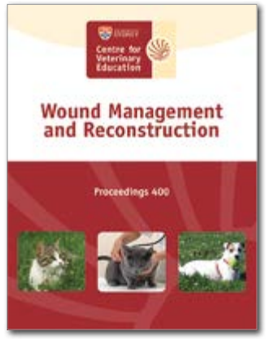
Product Code: CVEP399
Normal Price: $55.00
Member Price: $45.00
Authors/Lecturers:
Pete H. Laverty
Publisher: CVE
Month/Year Published:
April 2012
ISBN: 9781922054012
Format: Paperback
No. Pages: 74
www.cve.edu.au/vbscvep400



Download Table of Contents from the proceedings.

Product Code: CVEP400
Normal Price: $55.00
Member Price: $45.00
Authors/Lecturers:
Arthur House
Publisher: CVE
Month/Year Published:
April 2012
ISBN: 9781922054029
Format: Paperback
No. Pages: 132
www.cve.edu.au/vbscvep400

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Congratulations to Rebecca Ruby who is the Massey University 2011 recipient. Rebecca is entitled to $1,000 worth of CVE products/events due to being chosen by Massey Faculty as being the most competent in clinical skills over the clinical portion of her undergraduate years.

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21 Oct	**Small Animal Neuromuscular Diseases** Diane Shelton	Sydney
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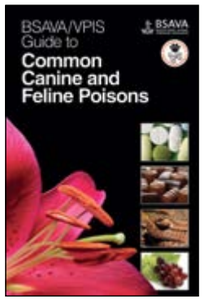
BOOK REVIEW

Reviewer

Anne Fawcett *BA(Hons) BSc(Vet)(Hons) BVSc(Hons) CMAVA*

Anne Fawcett is a small animal general practitioner based at Sydney Animal Hospitals Inner West. She is an associate lecturer in the University of Sydney Faculty of Veterinary Science and contributes to a variety of publications including *The Veterinarian Magazine* and *The Sydney Morning Herald.*

BSAVA/VPIS Guide to Common Canine and Feline Poisons



Authors: BSAVA/VPIS
Publisher: BSAVA
Pages: 174 (softcover/spiral bound)
Year: 2012
ISBN: 97819053195-9

The BSAVA/VPIS Guide to Common Canine and Feline Poisons is a handy, almost (but not quite) lab-coat pocket-sized tool for triaging small animal poisoning cases.

The book, a collaboration between the British Small Animal Veterinary Association and the UK's Veterinary Poisons Information Service, provides key information on a range of toxins known to affect dogs and cats.

It is organised alphabetically, by toxin, and divided into 2 colour coded sections, blue for dogs and mauve for cats, which are cross-referenced (for example, under the heading 'Benzodiazepine exposure in dogs' the reader is directed to the relevant page on benzodiazepine exposure in cats). The feline section is not surprisingly shorter, reflecting the cat's more discretionary eating habits.

Information is provided in point form under subheadings for each toxin. These include: alternative names, description/source, toxicology, risk factors, clinical effects (onset, common signs and other signs), treatment and prognosis.

Each potential toxin is designated with a traffic-light symbol, with those marked red likely to cause severe and life threatening signs and those marked green considered of low or negligible toxicity. Many agents in the latter category are included as the VPIS – and no doubt Australian private practitioners – receive many queries about these. It is a crude tool and I found myself on occasion disagreeing with the rating based on clinical experience, but more often than not I felt it was very accurate and spoke to my experience. Perhaps because cats are fussier, there were fewer toxins designated with a green light for cats (10) as opposed to dogs (21).

The book is not designed to be used as a sole reference on managing poisoning. The sections on treatment and prognosis are by necessity very brief and would certainly not be enough to enable a practitioner to manage a complex, critical case. You will still need a comprehensive toxicology textbook or (even better) access to peer-reviewed journal articles on management of particular toxicities. However, The BSAVA/VPIS Guide is ▶

Authors' views are not necessarily those of the CVE
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an excellent starting point and a useful general guide.

Members of the BSAVA are encouraged to use the book in conjunction with the BSAVA online Poisons Triage Tool, which can be found at ***www.bsava.com/Membership/MembershipPublications/PoisonsTriageTool/tabid/1326/Default.aspx*** For those who are not members, sample print outs on oral contraceptive pill exposure in dogs and ethylene glycol exposure in cats are available to view at no cost. The information contained is identical to that in the book, but the site is intended to be a living document, providing up to date information as it becomes available.

A case history checklist is provided to help veterinarians and nurses gather the necessary information to diagnose and manage patients, as well as submit a report to the VPIS. A VPIS case submission form is included. This is useful for Australian practitioners as the information on this form is very similar to that required if one were to submit an adverse experience report to the Australian Veterinary Pesticides and Medicines Authority.

Succinct, practical information is provided about dermal, gastrointestinal and ocular decontamination. Poisons are listed by their generic name (drugs) or Latin name (plants and animals). The index includes common names.

The Guide provides a summary of information based on available literature in addition to the VPIS's in house database of over 160,000 cases of poisoning.

As a practitioner I feel that the best application of this book is triaging cases over the phone. For example, if an owner phones the practice after applying a permethrin spot-on on their cat, I can collect as much information as possible, refresh myself on the key elements of treatment and – if necessary – seek additional information while the animal is in transit to the vet.

It's also fantastic for addressing those not-so-nasty poisonings over the phone. For example, an owner whose dog has just eaten their fish-oil capsule supply or a cat which has been nibbling on a Yucca tree.

Whether or not one subscribes to the VPIS, which is an exceptional service operating on a pay-per-case system, the case submission form is a very useful clinical record for poisoning cases. The information provided is accessible to veterinarians and experienced veterinary nurses.

For more information on VPIS please go to *www.vpisuk.co.uk/portal/Home/tabid/85/Default.aspx*

BOOK REVIEW

Reviewer

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Advanced Veterinary Therapeutics provides investigative and analytical skills in the areas of clinical pharmacology, toxicology, risk assessment and public health. Stephen has had a long standing interest in the appropriate use of veterinary medicines and vaccines and has participated in conferences and training programmes on this subject in worldwide.

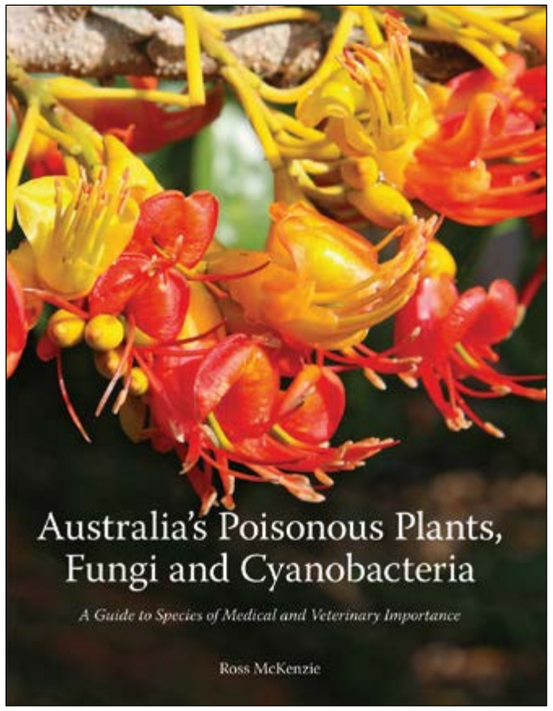
Australia's poisonous plants, fungi and cyanobacteria

A guide to species of medical and veterinary importance

Ross McKenzie

CSIRO PUBLISHING
(www.publish.csiro.au/pid/6507.htm)

ISBN: 9780643092679



Australia's Poisonous Plants, Fungi and Cyanobacteria (APPFC) is a rare example of a magnificently presented book replete with valuable information, authoritative, comprehensive, and, amazingly, just so readable. It is the literary equivalent of the signature dish from a 4 hat chef. The reader will be struck by the number and clarity of the photographs of plants and fungi associated with significant possibility of poisoning.

The author, Dr Ross McKenzie, now retired to tend his garden of toxic plants, is one of the very few veterinary toxicologists in Australia and well known for his passionate and infectious interest in the subject. This passion and dedication to teaching and learning are fundamental characteristics of this monograph, imbuing it with extraordinarily high value.

At the very start the aim is captured by the Chinese proverb that it is 'better to light a candle than to curse the darkness'. **Indeed this is the first reference book shining a light on plant, fungal and cyanobacterial poisonings of humans and animals (wildlife and domestic) throughout Australia.** Information is provided to allow the reader to recognise the main poisonous plants, fungi and cyanobacteria and the circumstances leading to poisoning. The author states that he has written the book to help *'householders, gardeners, parents of young children, child-care workers, school teachers, bushwalkers, pet and livestock owners, landholders, land custodians, medical and paramedical professionals, veterinarians, veterinary nurses, agricultural advisors, horticulturalists, park rangers and students throughout Australia.'* This is a very broad audience and an ambitious goal – a goal that I believe has been easily achieved, especially from the perspective of veterinarians, pet and livestock owners.

The opening sections of APPFC provide an excellent description of what the book includes and does not include (for example, mycotoxins of stored food, produce and plants causing contact dermatitis are not included), an outline of how to use the book followed by invaluable chapters on understanding plants and plant poisonings (including plant, environment and animal factors that increase risk of poisoning) and how to confirm tentative identifications.

Now better armed with an enhanced appreciation of toxic plants, Chapter 3 (54 pages in length) is devoted to common poisoning profiles. A total of 47 syndromes are described and importantly only 10 (those with asterisks in the following list) have an effective treatment. The syndromes described include Acute liver necrosis (15 sources of toxins identified); Cardiac glycoside poisoning (21)*; Corynetoxin poisoning (3); Cucurbit triterpene poisoning (4); Cyanide poisoning (27)*; Dihydroxycoumarin poisoning (3)*; Fern norsesquiterpene glycoside (4); Ergot alkaloid (5); Fluoroacetate (20); Galegine (4); Glucosinolate (4); Grayanotoxin (4); Iforrestine (2); Irritant diterpenoid (22); Lily poisoning of cats (>2); Lupin (quinolizidone alkaloid) (lupin spp); Macrofungal gastrointestinal syndrome (3); Methylazoxymethanol (MAM) (>4); Nicotine and other pyridine alkaloids (5); Nitrate-nitrite (35)*; Oesophageal ulceration of horses (Chillagoe horse disease) (2); Oxalates (soluble) (27)*; Oxalate crystalline intoxications (big head of horses) (16)*; Oxalate raphide crystals (>6); Phomopsins (lupinosis) (1); Photosensitization (19)*; Pregnane glycosides (3); Protoanemonine (4); Phyto-oestrogens (2); Psilocybin (5); Pyrrolizidine alkaloids (>9); Selenium (2); Senna (2); Simplexin (6); Steroidal glycoalkaloids (>3); Stypandrol (2); Sulphur (8)*; Sulphur containing organic compounds (esp S-methyl-L-cysteine sulfoxide SMCO & N-propyl disulphide) (13); Swainsonine and calystegines (9); Tannins (3); Thiaminases (4)*; Toxalbumin (4); Tremorgens (2); Tropane alkaloids (8)*; Urushiols (3); Wamps (6); Zamia staggers (>3).

The next 570 pages provide profiles of the individual poisonous species, divided into 3 parts: Part 1 Poisonous cyanobacteria (blue-green algae), Part 2 Poisonous fungi, and Part 3 Poisonous vascular plants (with chapters on ferns; cycads; grasses, sedges and mat-rushes; grass-trees; grass-like herbs (iris and lily families); forbs (non-grass-like herbs); vines (climbing plants and creepers); shrubs; and trees).

The author attests that all profiles are evidence based, meaning that all information is obtained from actual documented cases of poisoning. For each genus or species the profile contains a colour photo and information on the following subjects: Names (scientific, common, etymology, family); Description (plain language); Flowering and fruiting seasons; Main distinguishing features; Confusing species; Distribution and habitat (including map); Weight of evidence for toxic effects; Degree of danger (scored on palatability, dose and concentration, rapidity of action, severity, effectiveness of treatment – very dangerous = 9-10, low danger 5-6); Toxin name; Toxic parts of plant; Animal species affected; Conditions of poisoning; Toxic dose; Clinical signs; Post mortem changes; Management of intoxication.

The final chapter presents what is entitled a 'Digest of poisonous cyanobacteria, algae, slime moulds, macrofungi and plants in Australia'. This 200 page section summarises information on toxins, animals at risk, toxic syndrome, weight of evidence of toxicity and classification of degree of danger for all known or suspected poisonous species in Australia – a total of approximately 2,000 entries.

As if this is not enough, the book is completed with 6 appendices, a comprehensive glossary, references for further reading and a detailed index.

The appendices provide information on aids to identifying flowering plants: **The top killers** (the most dangerous plants, fungi and cyanobacteria in Australia); **Poisoning hot-spots** (in the home, garden and on the land); the major species poisoning animals; Body systems affected by the major poisonous species; and Australian states with major poisonous species.

The author includes many key messages. For example, the importance of accurate identification of suspected toxic plants, fungi and cyanobacteria is appropriately emphasised on many occasions. Such identification will frequently require consultation with experts in other fields (including botanists and mycologists). Clearly no diagnosis can be certain unless an accurate identification of the source of exposure is known. The author also notes that 'unpublished data are data lost' and entreats those encountering poisoning episodes to ensure that the identity of the implicated toxic plants, fungi, cyanobacteria etc is established in perpetuity by placing a voucher specimen in an appropriate institution. Finally, McKenzie's Maxim - 'the animal species, the dose and the circumstances make the poison' is certainly worth recalling.

Inevitably when the objective is to provide information to a large and varied audience the book cannot be expected to be definitive on every topic. For example, detailed information on the management of intoxications is not included and other sources of information will need to be accessed. Nonetheless, guidance from APPFC can be expected to help establish an accurate diagnosis. It would be invaluable to have access to the vast literature underpinning the evidence base summarised in the book. However, with around 2,000 species of poisonous organisms included there could well be 4-5,000 sources which might occupy another 200 pages.

***Australia's Poisonous Plants, Fungi and Cyanobacteria* is an amazing achievement. It now effectively and brilliantly bridges a gap of 30 years since the last edition of Selwyn Everist's remarkable 'Poisonous Plants or Australia' (1981) which provided descriptions of around 950 species (but with few photos and no distribution maps) and Alan Seawright's 'Chemical and Plant Poisons' (Animal Health in Australia, volume 2, 1982) which focused on 204 species.**

I believe that *Australia's Poisonous Plants, Fungi and Cyanobacteria* should occupy an important and prominent place on a bookshelf readily accessible to every veterinarian in clinical practice as well as those supporting clinicians or with other interests in animal health.

Editor's Note: We give the last word to Ross:-
*I have not written this as an academic text. There are no literature citations, just a short reading list, and I have tried to write in a plain language style so that non-professionals will have a chance of understanding. I want it to be accessible to animal owners and managers so that they can use it to **prevent** poisonings.*

Readers should realise that it has 2 functions: (1) information source and (2) exercise machine (it weighs 3 kg and repeated use will strengthen your arm muscles).

Authors' views are not necessarily those of the CVE

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… and more C&T articles and Perspectives needed
Thanks to every author who contributed articles or comments to the *Control & Therapy Series* (C&T). Without your generosity the Series would cease to exist. If you have treated a Large Animal, Reptile or any Wildlife lately, please write up the case and send it in. We aim to keep the Series broad and interesting.

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Entitling the recipient to one year's free membership of the CVE
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Film clips – Bird restraint
The September 2012 e-book features 5 film clips embedded in Mimi Dona's Birds article, part of the Wildlife Flash Cards Series.

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For further information on the *C&T Series* contact Lis Churchward, CVE Editor **cve.publications@sydney.edu.au** or (02) 9351-7979.

The C&T and Perspective Series is the brainchild of Dr Tom Hungerford, first Director of the PGF (1968-1987), who wanted a forum for uncensored and unedited material. Tom wanted to get the clinicians writing.

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

Examining the afterbirth of the mare

C&T No. 5243

Jim Rodger – CVE DE Tutor for our 2013 program - Equine Reproduction 1: The Mare
Centre for Equine Reproduction Medicine
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We can tell many things by examining the afterbirth (the placenta) of the mare after it has been dropped following birth of the foal.
- Is it complete or has some been retained?
- Is the mare healthy and has she had a healthy pregnancy?
- Is the placenta normal as expressed in appearance and weight?
- Is there evidence of placentitis?
- Is the placenta small and inadequate for the foal?

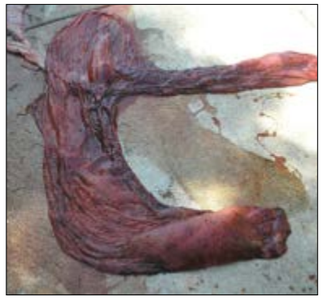

Figure 1. Afterbirth (placenta) of the mare

Figure 2. Checking the placenta

How to check the placenta

Firstly check that the tips of both horns are present. Retained pieces of placenta are usually at the tips of the horns and it is almost always the tip of the non pregnant horn that fails to separate. If you look carefully at the end of the horn it is possible to see the small 'star' where there are no villi and this is the point at which the placenta is adjacent to the papilla of the oviduct. If the star is not visible and the placenta is torn at that point you must re-examine the uterus as the tip of the horn is probably still in there.

How do you know which is the horn of the uterus? If you hold up the uterus by the tips of the horns the pregnant horn will hang down the way it was in the uterus as it is bigger and more pendulous. The placenta will hang down with the shorter side being the upper side. You will then be able to tell if the missing piece is in the right or left horn.

The tear where the foal has come through the placenta is usually at the cervical pole – rarely if ever at the tip of the horn.

The placenta can be checked for signs of infection. Ascending placentitis through an incompetent cervix (worldwide still considered to be the most common source of infection) can be identified by necrotic areas or pus at the area of the cervical star, the area where the cervix and the placenta are in apposition. Placentas that have had an infection are often oedematous and so will be thickened and heavier. On the other hand a small underweight placenta may result in inadequate foetal nutrition and intrauterine growth retardation (IUGR). A normal placenta with a healthy mare is a good indication for the next pregnancy as well as the health of the current offspring.

The uterine surface of the allantochorion should have a rich red velvet appearance with healthy villi. Uterine abnormalities such as cysts which have no villous attatchments, will be mirrored on surface of the allantochorion, as will be the site of the endometrial cups.

Figure 3. Close-up of the placenta

What can we learn from this?

The immediate application is the health of the mare following foaling. The presence of pus, excessive blood, or a retained piece of placenta are issues requiring immediate attention. Postpartum infections, haemorrhage or laminitis can very quickly become life threatening.

Information derived from the placenta can be essential for periparturient care of the neonate. A foal with an infected placenta may rapidly become septicaemic. Once they are clinically ill, reversal of the situation can be very difficult. Early intervention can be critical. On the other hand a foal that has been undernourished in the uterus will need extra care and the sooner it gets it the better. Neonates exhibiting signs of IUGR are not only smaller but are more likely to succumb to infection. Also, premature maturation of the stressed membranes will include the lining of the gut reducing absorption of immunoglobulins. Parenteral supplementation of immunoglobulins may be necessary.

Presentation of a healthy placenta without any retention is a good start when looking to get the mare back in foal, especially if looking to breed on the foal heat, but any deviation from this has to be regarded most seriously. Moreover this information is not only important in achieving pregnancy but maintaining it. Mares that had an abnormal placenta will need attending during pregnancy in case the situation recurs.

WINNER OF BEST FILM CLIP

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

Part 2: Wildlife Flashcard Series - Birds

C&T No. 5244

*This series is the result of collaboration between Mimi Dona & Dr Michael Pyne of Currumbin Wildlife Sanctuary Veterinary Hospital. Film clips courtesy of Lincoln Williams, Fotomedia (**www.fotomedia.com.au**). Non CVE members can access these flashcards and videos at **www.cve.edu.au/candt2012***

Mimi Dona

Senior Veterinary Nurse – Currumbin Wildlife Sanctuary Veterinary Hospital (CWS) & Lecturer on Animal Studies and Sustainability at the Metropolitan South Institute of TAFE.

‹e-book› Demonstrations of Bird Restraint

- medium_tawny_frogmouth
- pacific_bazza
- small_bush_turkey
- lorikeet
- waterbird_swan

BIRDS

Be Aware of:-

- Many birds are capable of defending themselves using either: their beak, claws, wings or a combination of these.
- Never put in wire cages as they can damage their feathers; line with cardboard or shade cloth if no other options. A cardboard box or pet carrier is more suitable.
- When handling care must be taken to avoid restricting sternal movement as birds do not have a diaphragm and rely solely on the movement of their ribcage for inspiration.
- Some species, particularly birds of prey, will require a high level of fitness prior to release and may need specialised training or a large flight aviary.
- If in care for some time, sea and water birds will need to be waterproofed prior to being released.
- Some species of birds are very territorial and will need to go back to the area they are from.
- Migrating birds have a regular seasonal migration and must be released prior to migration or held until they would normally return.

Handling

- Some sea and water birds can have pointy or knife like beaks and care should be taken when handling. Wear welding gloves and safety goggles if you are unsure.
- Always open the box/cage in a small room, and have a net in case it gets out.
- Small birds can be handled by gently cradling them in your hand, using a 'V' grip.
- Medium sized birds can be handled by holding your hand around the wings with the feet secured between your fingers.
- Parrots by using a pistol grip to hold their head.
- Larger birds can be handled by gently securing the head with one hand and holding the body in the other, be careful that the wings are contained.
- Birds of prey have talons that deserve respect; handle around the hock and avoid it taloning itself.
- Owls and some diurnal birds of prey can bite. Although this hurts, always keep control of the talons.

Figure 1 - The 'V' holding technique for small birds

Figure 2 - Medium bird holding technique

Figure 3 - Parrots require a confident restraint to protect yourself from their beak

Figure 4 - Bird of prey handling technique

Figure 5 - Larger sea and water birds without sharp beaks can be handled this way

Housing the sick or injured bird

- Preferred enclosure temperature is between 28º - 35º degrees celsius.
- Some birds have heavy feathering e.g. nocturnal raptors and sea birds so take care not to over heat. Keep below 28ºC unless young or unfeathered.
- The best temporary set up is a box (not ideal long term for parrots who can chew their way out) with air holes away from their eye line. Waxed cardboard pet packs are ideal and easy to clean. A rolled up towel will provide a perch and keep the bird elevated protecting its tail feathers.
- Young birds not perching need a nest – use a bowl with a small towel. A feather duster can be used to mimic mum. If the bird has no feathers it will require warmth.
- The debilitated bird should be placed on a doughnut shaped towel until it is able to perch.

Figure 6 (left)- Temporary housing using a rolled up towel as a perch
Figure 6 B (right)- Temporary housing using a box with a stick as a perch. This is not sufficent for parrots who can chew their way out.

Figure 7 (top left) - A brooder box assists in providing humidity and warmth to very young or featherless chicks
Figure 8 (right) - Enclosure design for small birds
Figure 9 (bottom left) - Enclosure design for small and medium sized birds

Emergency diet

Bird have specific dietary requirements that may not be able to be met in a Veterinary Clinic. The following emergency diets are not long term.

- Small or baby birds have a high calorie requirement and need to be fed frequently. This varies with species and age.
- If unsure of the species, the safest food to feed a baby bird is insects.
- Offer adults a variety of insects, seed, chopped fruit, greens and soaked dog kibble.
- Native vegetation and flowers are essential to many birds and are safe to feed so long as you are familiar with local native species.
- Many birds will not eat in hospital and will need to be assist fed or crop fed.

Take care when assist feeding as aspiration occurs easily in birds.

Assessment under gaseous anaesthetic

- Water bird and pigeon like species (*columbiformes*) are slow to induce.
- Fasting is required for birds prior to an anaesthetic. Take care with very small birds under 20 grams not to fast for more than 2 hours.

The fasting time varies between species and the crop can be felt under manual restraint prior to the anaesthetic to ensure it is empty.

- Must provide heat under anaesthetic.
- Give IPPV routinely throughout the procedure to reduce the risk of mucous plugs in the endotracheal tube.

Use an anaesthetic mask at 5 % induction, can take 2 - 3 minutes. Maintain using a mask on Isoflurane at 1.5 – 2% with an oxygen flow rate of 1 L/min.

Figure 10 - A Peregrine Falcon in a humidicrib demonstrating the 'donut' technique used for birds that are unable to right themself or need additional support post operatively

Figure 11 - Brown Goshawk under anaesthetic. Warmth is being provided via a Bair Hugger® blanket.

Anaesthetic agents

Propofol/Aquafol (10mg/kg) I/V – Often used in large waterbirds (Pelican, Swans).

Intubation

Use an uncuffed endotracheal tube (local anaesthetic not required) and tie in with micropore tape.

Recovery

Maintain heat throughout the procedure and post operatively. Vetarios or humidicribs are ideal.

Fluid Therapy

Give up to 10% of body weight S/C or slow I/V (warm 0.45% sodium chloride and 2.5% glucose).

Preferred routes

- Subcutaneous – administered in loose skin on flank cranial to thigh. Avoid giving sc fluids on dorsal neck – it is possible to inject into the air sacs.
- Oral – given via a syringe, tubing or crop needle (larger doses must be via crop needle or tubing).
- Intramuscular - pectoral muscle either side of the keel bone or thigh.
- Intravenous – jugular, wing vein (medial ulnar vein over elbow) or medial metatarsal vein.

Euthanasia methods

Injection of sodium pentobarbitone can be administered either by intravenous, intracardiac or intracoelomic routes.

- If administering by intracardiac, the bird should be anaesthetised first.
- Always dilute the solution with lethabarb:water 50:50.

Authors' views are not necessarily those of the CVE

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Feline Herpesvirus-1 as a differential diagnosis for feline facial pruritus

C&T No. 5245

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Figure 1. Feline herpetic facial dermatitis

Figure 2. The patient post treatment

Feline patients with allergic disease can present clinically with a reaction pattern known as head and neck pruritus. This severe and most frustrating of feline reaction patterns can result in dramatic mutilation and is often refractory to numerous treatments. A typical list of differential diagnoses includes: parasites (i.e Notoedres, *Demodex gatoi*, Otodectes) and allergic disease (Flea allergy, food allergy and atopy). Often overlooked is the possibility of feline herpesvirus ulcerative dermatitis. Feline Herpesvirus-1 (FHV-1) is most commonly associated with upper-respiratory tract disease, including rhinotracheitis and keratoconjunctivitis. Following primary infection, it is harbored in the trigeminal ganglion in latency. Following immunosuppression or stress, viral replication can reactivate and result in recurrence. This recurrence may present as a striking eosinophilic dermatitis of the face and/or nasal planum (occasionally on the extremities) that is easily mistaken by both clinicians and histopathologists for allergic eosinophilic dermatitis or eosinophilic granuloma complex. While the literature indicates pruritus may be moderate to absent (Gross *et al.*, 2005), some cases have marked pruritus and resulting self-mutilation which compounds the clinical confusion with allergic pruritus. This commonly results in a fruitless diagnostic work-up for allergic disease as well as ineffectual and possibly aggravating immunosuppressive therapeutics.

Histopathology of biopsy specimens reveals a necrotizing eosinophilic dermatitis. Intranuclear viral inclusions may not be present or may be easily missed by the histopathologist. Both PCR and immunohistochemical techniques have been investigated and validated as screening tools for herpetic dermatitis (Hargis et al., 1999, Persico *et al.*, 2011). However, the discovery of the safe and effective use of the human antiviral, Famciclovir, has revolutionized our ability to both diagnose and treat this disease (Thomasy *et al.*, 2006, Malik et al., 2009, Thomasy *et al.*, 2011). The author recommends a 2 to 3 week empirical course of Famciclovir as a diagnostic rule out in cases of feline facial dermatitis and pruritus where herpes may be suspected (125 mg PO BID). In the author's experience, response is noted in many cases of nebulous feline facial excoriation and dermatitis that were previously misdiagnosed as allergic in etiology and treated unsuccessfully with steroids and/or cyclosporine. The veterinary practitioner should be mindful of this insidious differential diagnosis when confronted with refractory feline facial dermatitis cases.

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Adenocarcinoma Feline

C&T No. 5246

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A 9-year-old male, neutered, domestic short hair presented because the owner identified a small, discharging wound on the ventral abdomen. The patient weighed 7 kilograms and had a body condition score of 6 out of 9. There was no history of a catfight or trauma. The cat was an outdoor cat and fed a diet of Hills Feline Adult. Four days prior to presentation (05/04/12) serum biochemistry and a complete blood count were performed (see Tables 1 and 2) because muscle wastage was identified when the patient presented for annual vaccination. Results of the biochemistry and complete blood count were unremarkable.

On physical examination a serosanguinous discharge was identified from a superficial wound on the caudoventral abdomen. Examination identified 2 puncture marks 15mm apart. Erythema was present surrounding the puncture marks. Differentials at the time were catfight or an insect bite. The patient was treated with cefovecin 8mg/kg subcutaneously.

The patient returned 14 days later for a recheck. Over that period appetite had reduced, the cat had lost weight (1.4kg) and the patient was lethargic.

Physical examination showed pale mucous membranes. Marked pain was elicited on palpation of the ventral abdomen.

Severe bruising and erythema were present on the ventral abdomen (see Figure 1). The lesion felt soft and fluid filled. The cat had generalised oedema.

A complete blood count was performed (Table 1). A mild regenerative anaemia was identified possibly secondary to blood loss (from the wound) or haemolysis. Blood loss was considered more likely due to a concurrent hypoalbuminemia. A moderate neutrophilia was present with toxic change suggestive of an inflammatory response possibly related to inflammation.

Serum biochemistry identified hypoalbuminemia, which was consistent with protein loss through the skin lesion. Electrolytes abnormalities were present due to inappetence or third space fluid loss. Addison's disease was considered unlikely.

Differential diagnosis at the time was a pansteatitis secondary to atypical infection (actinomycosis, actinobacillosis, mycobacteriosis, nocardiosis), deep mycotic infections, neoplasia, foreign body and steatitis secondary to pancreatitis or immune mediated disease. Nutritional deficiencies secondary to excess consumption of fatty acids or a vitamin E deficiency were also considered.

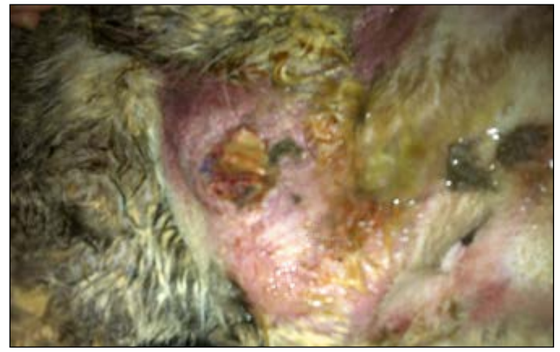

Figure 1. Images of the cat – 2 weeks post initial presentation

The plan was to stabilise the patient and subsequently explore the abdominal wound with the view of collecting deep biopsies for histopathology.

Subsequently, intravenous fluids (0.9% sodium chloride at 4mL/kg/hr) were started to help rehydrate the patient. Gelofusin was administered (0.8ml/kg/hr) for colloid support. Four quadrant antibiotics, metronidazole 15mg/kg IV BID, cephalothin 22mg/kg IV TID and marbofloxacin 4mg/kg PO SID were initiated to treat for possible bacterial infection. Methadone 0.3mg/kg SC q4h was given for pain relief.

Thoracic radiographs (left and right Lateral plus ventrodorsal views) and an abdominal ultrasound were performed to check for evidence of neoplasia prior to performing any surgery.

Thoracic radiographs were unremarkable. Abdominal ultrasound showed a thickened hyperechoic irregular pancreas.

A fine needle aspirate of the integument lesion was performed. A large amount of serosanguinous fluid was aspirated. The sample was sent for cytology and culture and sensitivity.

Whilst waiting for cytology, differentials were considered. In cats, fat necrosis following pancreatitis has been rarely identified.[2] This was considered unlikely due to a normal spec feline pancreatic lipase immunoreactivity test result.

In cats, pansteatitis is often caused by a nutritional deficiency of vitamin E or excess consumption of unsaturated fatty acids. This was considered unlikely as the cat was not fed a fish based diet.

A similar case to the one discussed has been reported where the patient had a cutaneous necrotising panniculitis and steatitis associated with pancreatic adenocarcinoma in an 11-year-old male Siamese cat.[3]

Pancreatic tumours rarely occur in cats. They are generally invasive and liver metastasis is common. No evidence of liver metastasis was present on ultrasound; however, this cannot be ruled out. In humans, pancreatic carcinoma are often associated with subcutaneous fat necrosis.[4,5] It has been hypothesised that the aetiology is fat necrosis secondary to systemic release of lipolytic enzymes.

Cytology of the fine need aspirate identified an exudate, likely carcinoma (see Table 3).

It was hypothesised that the patient had adenocarcinoma of the pancreas with secondary pansteatitis. The animal was euthanised on request of the owner. A post mortem was declined. Culture and sensitivity of the fluid was cancelled. ▶

Table 1: Complete Blood Count (Idexx External Lab)

Test	05/04/12	23/04/12	Units	Reference Range
RBC	5.4	3.7	x10^12/L	4.9 - 10
Haemoglobin	92	67	g/L	77-156
Haematocrit	0.29	0.23	L/L	0.25-0.48
Reticulocyte%		6.4	%	0.0-0.4
Reticulocyte ABS		237	x10^9/L	0-60
MCV	54	62	fL	43-55
MCH	17	18	Pg	13-17
MCHC	317	291	g/L	282-333
Platelets	Clumped and adequate	Clumped and adequate		
Platelet Count	294	267	x10^9/L	300-800
WBC	16.4	31.9	x10^9/L	5.5-19.0
Neutrophils%	83	86	%	
Neutrophils	13.6	27.4	x10^9/L	2.0-13.0
Band Neutrophils%		7	%	
Band Neutrophils		2.2	x10^9/L	0.0-0.2
Lymphocytes%	11	3	%	
Lymphocytes	1.8	1.0	x10^9/L	0.9-7.0
Monocytes%	3	3	%	
Monocytes	0.5	1.0	x10^9/L	0.0-0.6
Eosinophils%	3	1	%	
Eosinophils	0.5	0.3	x10^9/L	0.0-1.0
Basophils%	0	0	%	
Basophils	0	0	x10^9/L	
Blood smear examination	Normal red and white cell morphology	Moderate toxic change. Moderate anisocytosis. Moderate polychromasia.		

Table 2: Serum Biochemistry (Idexx External Lab)

Test	05/04/12	23/04/12	Units	Reference Range
Sodium	157	139	mmol/L	144-158
Potassium	5.1	5.9	mmol/L	3.7-5.4
Chloride	123	106	mmol/L	106-123
Bicarbonate	12	12	mmol/L	12-24
NA:K ratio	30.8	23.6		>29
Anion Gap	27.1	26.9	mmol/L	15.0-31.1
Glucose, Serum	4.9	5.6	mmol/L	3.2-7.5
Urea	5.5	14	mmol/L	5.0-15.0
Creatinine	0.15	0.11	mmol/L	0.08-0.20
Calcium	2.2	2.1	mmol/L	2.1-2.8
Phosphate	1.3	2.4	mmol/L	1.0-2.3
CA:P ration	1.7	0.9		1.1-2.3
Protein Total	55	47	g/L	60-84
Albumin	27	22	g/L	25-38
Globulin	28	25	g/L	31-52
Bilirubin	6	8	umol/L	<7
ALP	19	8	IU/L	5-50
AST	26	43	IU/L	2-62
ALT	6	5	IU/L	19-100
CK	269	710	IU/L	64-400
Cholesterol	3.1	3.8	mmol/L	2.2-5.5
Gamma GT	<3	<3	IU/L	0-5
Sample Appearance	Normal	Normal		
T4	11	<10	nmol/L	10-60
Spec FPLi		0.5	ug/L	0.1-3.5

Table 3. Idexx cytology report

Cytopathology
***Cytology interpretation specimen:**
Integument lesion. 1 fluid submitted.

Cytology:

The sample was red and opaque and largely cleared with centrifugation.

Red cell count 0.4 (0 x 10^{12}/L)
Nucleated cell count 39 (0-1.5 x 10^{9}/L)
Protein 33 (0-30 g/L)
Albumin 16 g/L

The nucleated cell population was composed of a mixed population of neutrophils and activated and phagocytic macrophages. Within this was a population of large angular cells in rafts, sometimes in acinar like structures. These exhibited round to oval nuclei with lacey chromatin and commonly single or multiple, sometimes large, nucleoli. The N:C was moderate and the cytoplasm moderately basophilic and commonly finely vacuolated. No other significant cellular population or microorganisms were identified.

Interpretation: Exudate, likely carcinoma.

Comments:
Exudates may reflect septic or non-septic inflammation or neoplasia. In this case, there is a mixed, pyogranulomatous inflammatory response. Within this is a cellular population with significant atypia which appears most consistent with epithelial origin. As such, underlying carcinoma is suspected in this case.

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Practice Tips

C&T No. 5247

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Muzzles: For clients that get a bit 'cranky' when you announce you are going to muzzle their dog, politely advise them it is a *WorkCover* (OH&S in Victoria) issue, WHICH IT IS. The difference in their attitude is astonishing! We take no risks with dogs that even MIGHT bite!

Off label ocular Cyclosporine: Take one 100mg Atopica® (cyclosporin A; Novartis) capsule, pierce with a needle and squirt the entire contents into a small dispensing bottle and add 9mL liquid paraffin B.P. We use Faulding Remedies Paraffin Liquid BP® which comes in a 100 mL bottle. Remember to store it in the dark. (This formula is courtesy of Animal Eye Care here in Melbourne.)

CALL FOR ASSISTANCE

Q fever (*Coxiella burnetii*) – has anyone been sick in your practice?

University of Sydney researchers, Amanda Shapiro (PhD candidate) and Drs Jacqueline Norris and Karina Bosward, are investigating the potential role companion animals may play in the transmission of *Coxiella burnetii* to humans. This research has been sparked by an outbreak of Q fever in veterinary personnel in a Sydney companion animal veterinary hospital in 2010, following a cat caesarean section. In the preliminary stages this is being investigated by determining the seroprevalence of previous exposure to *Coxiella burnetii* in dogs and cats in eastern Australia as well as investigating the potential role of this organism as a cause of disease in our companion animals. **We would therefore love to hear from any veterinarians and veterinary nurses who have been confirmed or are suspected of having Q fever resulting from contact with infected dogs and cats. All that we require to further develop our diagnostic assay is a serum sample from the suspected or confirmed animal.** All cases will be dealt with strict confidentiality.

Please contact and supply further information to:-
Jacqui Norris, ***Jacqui.norris@sydney.edu.au***
Karina Bosward, ***Karina.bosward@sydney.edu.au*** or
Amanda Shapiro, ***Amanda.shapiro@sydney.edu.au***

Figure 1. Neonate (courtesy of Anne Fawcett)

Figure 2. Neonate (courtesy of Anne Fawcett)

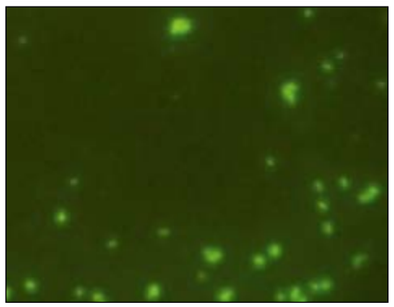

Figure 3. Indirect IFA (courtesy of Jacqui Norris)

Re-published here courtesy of Vetnostics, North Ryde

ADRENALS: What you won't find in a textbook

C&T No. 5248

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Part 1: Signalment – Hyperadrenocorticism (HyperA)

Part 2: Clinical Signs – Hyperadrenocorticism (HyperA)

PART 3. ROUTINE CLINICAL PATHOLOGY

Hyperadrenocorticism (HyperA) – (Cushing's Disease) Haematology

1. Lymphopenia and absolute eosinopenia are the most frequently cited haematologic abnormalities (approximately 80% of dogs according to Feldman and Nelson 2004). However, they are not always present. Lymphocyte counts in hyperA dogs at Vetnostics quite often seem to be normal which would be consistent with the finding in one study that only 14% dogs with hyperA had lymphopenia (Peterson, 1984). **Eosinopenia seems far more common than lymphopenia** (consistent with the figure of 84% by Peterson, 1984). However, eosinophils can be normal to increased if there is a concurrent eosinophilic process (uncommon but occasionally seen).
2. Nucleated red cells are seen quite commonly (usually in low numbers, 1-3/100 WBC). Whilst this number would be classed as normal, it is more common in dogs with hyperA than in other dogs of similar age with no reported history or signs typical of hyperA.
3. **High normal or increased platelet counts are often apparent.** The cause and significance of this is unknown.

Biochemistry

1. Increased alkaline phosphatase (ALP) is widely cited as the most common routine laboratory abnormality but it is not always increased. A normal ALP does not rule out hyperA.
2. Increased ALP is largely due to induction of a specific ALP isoenzyme by glucocorticoids. Although this isoenzyme can be evaluated, it has been shown that an increase can be caused by a variety of disorders and is not specific for hyperadrenocorticism (Solter et al 1993). The steroid-induced iosenzyme cannot be used to distinguish spontaneous or iatrogenic hyperA from liver disease or diabetes mellitus for example. It is often stated that 70-100% of the increase in hyperA dogs will be due to this isoenzyme but this is certainly not always the case with either iatrogenic or spontaneous hyperA (Feldman and Nelson 2004).
3. **Lipaemia is very common in hyperA dogs.** Most of the old studies that reported biochemistry findings did not report the frequency of hypertriglyceridaemia in hyperA dogs as veterinary laboratories have not typically run triglyceride ▶

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concentrations. The triglyceride increases in hyperA dogs are often quite marked and the serum/plasma consequently often has a strawberry milkshake appearance, even on a fasted sample. HyperA should always be on the DDx list for fasting hypertriglyceridaemia in an otherwise well dog (or cat!). This also holds for breeds such as Miniature Schnauzers, not of all which will have familial hypertriglyceridaemia; Miniature Schnauzers do get hyperA (See Part 1: Signalment – Hyperadrenocorticism (HyperA)).

4. Mild hyperglycaemia is reported as occurring in 45-60% of hyperA dogs (Peterson 1984, Feldman and Nelson 2004) but this would not be the case in my own patients or Vetnostics' cases. Whilst hyperglycaemia can occur, it would seem to occur at a much lower rate in our patients.
5. Alanine aminotransferase (ALT) is commonly increased but again, not necessarily. It is not usually increased to the same extent as ALP.
6. Urea concentration may be decreased due to polydipsia.
7. Hypokalaemia and hypernatraemia may occasionally be seen and are probably more common in dogs with adrenal tumours as the cause of their hyperA (presumably excessive mineralocorticoid secretion).
8. Bile acids test results may be increased in dogs with hyperA (Center et al 1985).
9. Serum lipase may be increased by exogenous corticosteroids (dexamethasone) thus possibly by endogenous glucocorticoids also. Although it would not be routinely measured in hyperA cases, this must be borne in mind when analysing lipase in potential pancreatitis cases. The effect of glucocorticoid excess on cPLI is currently unknown.

Urine

1. Urine in dogs with hyperA is usually isosthenuric or hyposthenuric and in one old study (Meijer 1980), 80-85% of dogs had a USG <1.013. However, as we often pick up hyperA much earlier (i.e. before they become textbook classics) that figure is probably an overestimate in modern medicine. Not all hyperA dogs have polydipsia/polyuria as presenting signs; only 82% of 300 hyperA dogs had PU/PD in one report (Peterson 1984). In addition, many dogs can concentrate their urine reasonably after being in a hospital (see Part 2: Clinical Signs – Hyperadrenocorticism (HyperA)) so the urine concentration measured at any one moment, could be hyposthenuric, isosthenuric or concentrated.
2. Urinary tract infection (UTI) reportedly occurs in 40-50% of hyperA dogs (Feldman and Nelson 2004). Again, I think it would be interesting to review that figure. Frequency of UTI has probably decreased with earlier detection of the disease but as urine culture is not routine, this is impossible to assess.
3. It is important to remember that (a) hyperA dogs with UTIs may not have any pyuria or haematuria (presumably because of the anti-inflammatory effect of excess glucocorticoids) and (b) routine sediment examination on a wet preparation may fail to detect white cells and bacteria in dilute or weakly concentrated urine. A stained, air-dried smear will increase detection of both white cells and bacteria but culture is usually required to detect UTIs in dogs with hyperA. UTIs may well be undiagnosed in hyperA dogs.

Hypoadrenocorticism (HypoA)

Haematology

1. Lack of a stress leucogram in a sick dog can be an indication of hypoA and may be the only clinicopathologic abnormality in dogs with glucocorticoid deficient (atypical) hypoA. When I ask vets about the leucogram in suspected hypoA cases, the common response is 'Everything is normal'. **Remember, a normal leucogram can be quite abnormal for a collapsed dog** and each count should be assessed with respect to the dog.
2. Lymphocytosis is not always present.
3. Lymphopenia is a good 'rule-out' for hypoA. I have never seen a hypoA case with lymphopaenia: However, a recent study on lymphocyte counts in dogs with hypoA (Seth et al 2011) did identify a few low lymphocyte counts. In this study, 100% of hypoA dogs had a lymphocyte count >0.75x10^9/L and 92% had lymphocyte counts >1.00x10^9/L.
4. Eosinophilia is also not always present.
5. I have never yet seen a hypoA dog with an eosinophil count of 0, thus an eosinophil count of 0 would be a good 'rule-out' for hypoA. Let me know if you have a hypoA dog with an eosinophil count of 0!

Biochemistry

A Na:K ratio of <27 is NOT diagnostic for hypoA. In one study only 24% of dogs with Na:K ratio <24 had hypoA and 41% of dogs had renal disease (Roth and Tyler 1999). It is worth noting that all dogs in that study with Na:K ratios <15 had hypoA. Other diseases causing low Na:K ratios include whipworm (and other gastrointestinal diseases), renal disease, pancreatitis, diabetes mellitus, pyometra and body cavity effusions. Another larger, more recent study showed that whilst hypoA was the most common cause of a Na:K ratio <27, only 16.7% of dogs with Na:K ratio <27 had hypoA (and that was after the cases with suspected EDTA contamination had been removed from the sample population).

General

1. Combining the Na:K ratio with the lymphocyte count is diagnostically superior as a screening test than either alone (Seth et al 2011).
2. A faecal flotation test should be performed in all dogs with a low Na:K ratio. Occasionally dogs have both whipworm and hypoA!
3. A manual differential white cell count (of at least 100 cells) is mandatory in order to detect hypoA leucocyte 'patterns' with any accuracy. Most of the in-house analysers would not perform with high enough accuracy (Papasouliotis et al 1999, Bienzle et al 2000, Papasouliotis et al 2006) to detect the subtle 'patterns' of hypoA.

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WINNER

Trying it a different way...

C&T No. 5249

Anonymous

Editor's Note: *A CVE Member sent in this article about her own cat which we believe is both very good and very honest. In order to encourage more vets to share valuable experiences/lessons learnt with our members/readers, the CVE/PGF has always published articles anonymously where appropriate.*

Background

'Maxine', a 4-year-old spayed female DSH, was presented for lethargy and a fluctuating appetite of 1-3 weeks' duration, depending on which owner was questioned.

She had a history of weight gain 2 years previously after the loss of the family dog she was bonded to. Her owners admitted to coping with her cries for attention by giving her extra food. Later, the introduction of a kitten to the household helped alleviate the situation and return her to more normal eating habits and her previous weight. Her temper then deteriorated over some months, supposedly because of jealousy and 'Feliway' was used to keep the peace between the 2, to some effect.

Her recently erratic appetite was initially put down to her 'prima donna' temperament. There was the possibility that her drinking was increased, however she led a normal indoor-outdoor life, going to stables and paddocks, seemingly healthy. She alternated between hissing at the younger cat and playing with her. She had occasional dust-ups with neighbouring cats.

Examination

Her condition was light (BCS 3/9; BW 3.7kg, previously recorded at 4.7kg), she had an unkempt coat and a moderate pyrexia (39.6ºC). The rest of the examination was unremarkable, including auscultation of the chest and palpation of the abdomen. Her behaviour was smoochy and alert, and she purred readily.

Laboratory results

The haematology abnormalities were MCH 21.4 (12.0-20.0) and platelets 902 (175-600). The white blood cells were all WNL, the neutrophils low normal, if anything, 3.57 (2.5-12.5) and lymphocytes 1.54 (0.40-6.80). The plasma was moderately icteric however and her haematocrit 33%.

A pre-anaesthetic profile was done and glucose, urea, crea, TP, ALT and ALP were all within normal values. A urinalysis was not performed. She was FeLV/FIV negative on the Idexx in-house SNAP test.

Next

Maxine was started on clindamycin 25mg bid for a suspect bacterial infection. After 4 days her pyrexia had not abated. Her HCT was down to 31%, the icterus of the plasma had increased but she was not herself icteric. The WBC count by this time was low, with a neutropaenia (2.08) and all the others low normal. This was presumed to be secondary to high demand and consumption.

Ear blood samples were collected for mycoplasma examination but no blood was sent for a PCR, unfortunately. She was started on 5mg doxycycline bid. The blood smear result was negative but she seemed to respond; the pyrexia disappeared and her appetite improved for a few days. On the resumption of her inappetance she was started on 1mg/kg prednisolone bid orally to decrease erythrophagocytosis.

Pilling her soon became a major problem especially as the more adept owner works away from home part of the week. Most of the efforts for the next 10 days were placed in ensuring that treatment was given properly.

At this point her abdomen was re-palpated as the owners were concerned about constipation. She was found to be mildly constipated and treated with Duphalac syrup.

Her condition deteriorated rapidly over the next 2 days. Blood was sent away to Idexx laboratories after discussion with their pathologist. She was put on an intravenous crystalloid infusion (Hartmann's) and given prednisolone sodium succinate (Solu-delta-cortef®) IV instead of oral prednisolone. She was started on a carafate slurry tid in case of GIT ulceration, and given maropitant (Cerenia®) to help with any nausea.

Her appetite deteriorated further and didn't respond to cyproheptidine (Periactin®) before a meal. Diazepam IV was equally unsuccessful.

The haematology showed a HCT of 26%, low WBC (5.2), this time due to a lymphopaenia 0.16 (0.9-7); the neutrophils were WNL at 4.94. Reticulocyte count normal range: 5 (0-60).

Electrolytes showed low sodium (143), and chloride (103), normal potassium, marginally low calcium 2.0 (2.1-2.8), a normal Ca:P ratio, a low protein 50, from low albumen 23 as well as globulins 27. The low calcium was deemed due to hypoalbuminaemia.

The liver enzymes were now increased ALP: 129 (5-50), AST 81 (2-62), ALT 126 (19-100). The GGT was 0 (0-5). The Total bilirubin was high 34 (<7)

The urinalysis showed a USG 1.036, 2+ bilirubinaemia, 2+ blood, rest normal.

Her T4 level and Feline pancreas-specific lipase were normal.

Comments from the pathologist:

On the biochemistry: panhypoproteinaemia: GI loss, loss into body cavity, DDx hypoalbuminaemia: GI or renal loss, decreased hepatic production. Elevated ALP, ALT, AST, TBil: hepatopathy, neoplasia. Consider imaging the liver.

Haematology: low normal HCT, at present not anaemic. lymphopaenia non-specific: stress-related or secondary to corticosteroids, pain, pyrexia.

Next

The next day she looked pale and weak and her HCT had gone down to 8%.

No attempt to diagnose the reason for this sudden drop was made as she needed an emergency transfusion. This being a country practice on a week-end there was no means to type ▶

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her and she was given 50mL freshly collected dog blood instead. There was no reaction during the transfusion and she was much improved. Her HCT was 22% post-transfusion.

She remained stable overnight other than developing a mild pyrexia, thought to be due to the transfusion. An abdominal ultrasound was performed the next morning to locate a potential source of bleeding. There was a small amount of free fluid in the caudal abdomen near the bladder and in the caudodorsal abdomen. The only other outstanding finding was a hugely enlarged and abnormal left kidney: 53.4 mm length (Normal: 36.6 +/- 4.6mm) and 40mm height (N: 22.1 +/- 2.8mm) and mixed echogenicity in the cortex (Figure 1). For a moderately experienced rural ultrasonographer the rest of the abdomen, including the liver and right kidney, appeared normal.

A decision was made to use the time-honoured ultimate diagnostic tool of a laparotomy, while she was stable enough. She had been on fluids since the transfusion, and was urinating though not otherwise dramatically improved.

A routine midline laparotomy incision was performed. There was no trace of current or past haemorrhage and the fluid collected was straw-coloured and formed a stable foam when shaken. As per the ultrasound the major finding was of a grossly abnormal and enlarged left kidney with extensive adhesions (Figure 2). A nephrectomy was performed and the abdomen was routinely closed.

The following day the samples were sent to the laboratory. The fluid came back negative for FIP. The kidney histopathology showed a poorly differentiated lymphoma.

Before the results were back Maxine crashed again (HCT 10%) and her owners elected euthanasia. She was buried next to her mate the dog, 38 days after first examination.

Discussion

Names clearly have been changed or suppressed to protect the innocent!

There were a number of mistakes made that stemmed from the sheer disbelief of a problem of this magnitude in a young active cat. Common things occurring commonly, the pyrexia was thought to be due to an occult bacterial infection or an early viral infection. The plan was to repeat the retroviral test in 3-4 weeks.

The relatively swift change from clindamycin to doxycycline was merely the result of the low normal haematocrit and the appearance of the plasma. A mycoplasma titre would have been helpful from the outset, negating the assumption of a false negative smear examination and hopefully inducing a more thorough search of the pathogenesis of her illness.

A false sense of security was derived from the normal liver enzymes in the original test. The low normal WBC initially, followed by seemingly erratic changes in laboratory results also contributed to confusing the clinical picture. The urinalysis should have been done on day one since it is part of a minimum database; bilirubinuria is always abnormal in the cat which would have rung alarm bells.

Time constraints on the part of owners and clinicians (though not money), and her resistance to medication also delayed the onset of a more logical diagnostic course.

From the first early discussions the advice was to image the liver and pancreas but this was not done until late in the course, though they were both assessed via her blood. Abdominal palpation was also done only a few times and did not yield any hint of kidney enlargement until the last few days. Her urine specific gravity and kidney enzymes remained normal throughout and since the right kidney was grossly normal it is assumed that it compensated fully for the other one.

The alarming drops in HCT from the mid-20's to around 10% seemed inexplicable without a source of haemorrhage. Indeed it was felt that the check for constipation had triggered the drop, indicating a friable blood-filled lesion in the abdomen.

While she is described as a DSH, she had the appearance of a British Shorthair cross and this made it statistically more likely that FIP might be a problem. However, it was ruled out at the end.

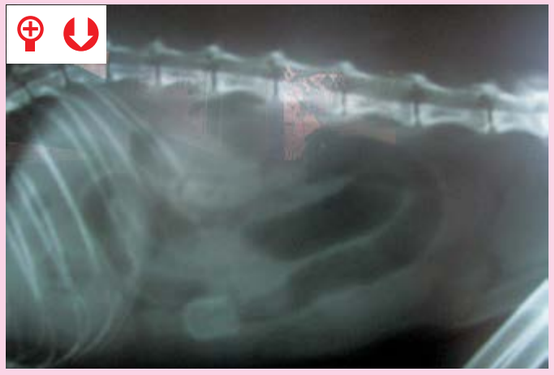
Conclusion

Other than the obvious of always providing yourself with a through minimum database, there is much to be learnt from this unusual case.

Upon consultation with specialists Dr Vanessa Barrs from Sydney University and Dr Richard Malik from the CVE, it seems likely that the right kidney and the liver were indeed affected, whatever their gross appearances. I am also told that a renal lymphoma can blow out in size very rapidly, explaining why this obviously huge kidney was not felt until very late. Dr Malik also reported that cases of feline renal lymphomas diagnosed in cats older than 18 months of age have a survival time of about 3-4 months. The younger cats respond better to multi-agent chemotherapy and may survive for many years. So the outcome was never likely to be a good one. However, there is a lingering sense of not having risen to the occasion when presented with the unexpected.

What's Your Diagnosis?

Answer to C&T No. 5236



Thanks to Michiru Oshima, Stephen Gibson and Kay Gerry for sending in answers. The winner is Kay Gerry whose answer appears below. Wayne enjoyed Stephen's answer, which was pretty close: *Could it be a rubbery finger protector people wear when sewing? (I think Wayne Mizon has one himself.)*

C&T No. 5250

Kay Gerry
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My diagnosis is that the cat has a small intestinal obstruction secondary to swallowing a rubber feeding teat e.g. for lamb.

Note: Kay wins a CVE proceedings of her choice – see Vetbookshop.com

Replies to Tick Paralysis in the cat

C&T No. 5193, Dec 2011, Issue 264

C&T No. 5251

We would like to thank Dr Graham for taking the time to articulate her response to Dr Gaschk's article and – more importantly – allowing us to publish it in the C&T, thus setting in motion this lively 'Round Table Discussion' allowing Australian vets to debate this highly topical subject. Due to time and space constraints, this Round Table Discussion will be published in 2 parts, with more contributions from vets all around Australia appearing in our upcoming December 2012 issue.

Reply No. 1

Karina Graham
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Dr Gaschk has nicely summarised an approach to tick paralysis in cats which is very similar to the way I personally treat these patients. The one exception was his comment regarding pulmonary oedema, '*consider* chest radiographs (ddx: pulmonary oedema), tx frusemide 1-2mg IV q6hrs'. It is my view, and that of many of my friends and colleagues in Sydney, that we don't observe cardiogenic pulmonary oedema in cats OR dogs caused by paralysis tick envenomation. Therefore, the use of frusemide is not only unwarranted, it potentially compromises renal perfusion in dehydrated, sedated cats.

Dr Campbell's PhD studies in 2000 looked at cardiovascular function in canine tick paralysis patients and concluded that cardiogenic pulmonary oedema does occur in dogs and postulated that it is the likely contributor to death in severe cases. She claimed the heart failure was due to diastolic dysfunction; however, this was based on M-mode measurements (LVIDd) and not more accurate measures of diastology like mitral inflow studies or tissue Doppler imaging (TDI). I suspect mitral inflow studies were not routine then, and certainly TDI was in its infancy. However, in 5 patients where BALs were performed, the fluid collected was low in protein and thought to be consistent with cardiogenic pulmonary oedema. Her study was well designed and certainly suggests cardiogenic pulmonary oedema occurs; however, we just don't seem to see it clinically.

Figure 1. **Ixodes holocyclus *paralysis tick in water (Courtesy of Anne Fawcett)***

My mentor, Dr Richard Churcher, begun investigating this hypothesis because of the discrepancy between what was reported and what we were seeing in practice and performed echocardiology on several dogs with acute tick paralysis. No abnormalities were found using mitral inflow studies. We contemplated conducting a prospective controlled study, but we considered it too distressing for many of these patients to undergo full echocardiography while experiencing severe respiratory compromise and aborted the idea.

In our referral hospital, we may treat up to 5-10 tick paralysis patients on any given day during the tick season, usually with 1-2 receiving mechanical ventilation, and never have I used frusemide. In fact, I don't know of any specialist practitioner in Sydney (especially those treating large numbers of tick paralysis patients) that use frusemide routinely, if at all. I believe the respiratory compromise is due to a combination of respiratory muscle paresis/paralysis (including upper respiratory tract structures causing upper airway obstruction), aspiration pneumonia and occasionally non-cardiogenic pulmonary oedema (perhaps secondary to upper respiratory tract obstruction – especially in cats!). In severe cases, we provide mechanical ventilation, continuous positive airway pressure (CPAP), extensive respiratory physiotherapy, nebulisation, antimicrobials if indicated, frequent arterial blood gas analyses and careful nursing care; very rarely do we lose patients (if costs are not an issue...). I believe the cause of death in most cases is severe respiratory acidosis (due to ventilatory failure) and occasionally severe aspiration pneumonia or ventilator associated injury. Patients can rapidly become acidaemic from hypoventilation, often with pH values below 7.0. We presume such patients, if untreated, develop cerebral oedema, followed by death. With increased monitoring of arterial blood gases, we are much more aggressive with ventilation and as a result very few patients die (I would estimate less than 1 patient each season). Furthermore, all cases where food and water are withheld (all but the very mild cases) receive maintenance intravenous fluid support from admission.

I am not suggesting there are no cardiac effects from the toxin; however, I do think practitioners need to be well aware of the pros and cons when considering frusemide or other heart failure medication. Dr Gaschk's recommended dose of frusemide is very low. Indeed, for cardiogenic pulmonary oedema it may be sub-therapeutic. The concern is that vets will use increasingly higher doses in patients that are seemingly not responding when perhaps they require intubation to relieve the obstruction (especially cats) or artificial ventilation for worsening hypercapnia (especially dogs). In short, I don't believe cardiogenic pulmonary oedema is a feature of tick paralysis in either cats or dogs.

Q & A (Qs by Richard Malik/As by Karina Graham)

1. Have you read the Ilkiw papers?

I skimmed them; they are quite old so I didn't read them in detail. To be honest, if they said 'without a doubt' that congestive heart failure is caused by tick paralysis I still wouldn't take too much notice – because we don't see it clinically. It MAY have existed, I don't know. Now you have me more interested, I will review them further… Perhaps the best thing is to ask Fiona Campbell her thoughts? She is an outstanding cardiologist who used to see (and maybe still does evaluate) many tick paralysis patients. Do they treat any tick paralysis patients for heart failure? If so, how come we get away without treating them?

Editor's Note: Dr Campbell's Comment will be published in our December 2012 issue.

2. Can you extrapolate from dogs to cats?

There are differences i.e. cats appear to suffer from upper respiratory tract obstruction more than dogs, and often you can just sedate them and intubate them. No frusemide though! ▶

Dogs more often require mechanical ventilation. However, either species can have it the other way around too.

3. What about measuring pulmonary wedge pressure and cardiac output?

Maybe. We would need to organise a prospective study to sort this out.

4. How many dogs did you or Dr Churcher echo?

I'll ask as I'm not entirely sure. I'm guessing a handful of cases, but they were before my time; that's why I confidently don't use any heart failure medication. However, it is very difficult to restrain these patients for thorough echocardiography.

5. Do you use CPAP or PEEP?

Both. PEEP when using the ventilator (for many patients) and CPAP mainly when we're weaning them off ventilation. CPAP is a great tool when we want them conscious but they're not strong enough yet to adequately ventilate, or have ventilation related respiratory fatigue. It has many other applications too, some use if for recovery of brachycephalic patients.

6. Why do you do so many arterial blood gases - why not capnography?

We have capnography constantly monitored; however, it is not nearly as accurate as arterial blood, and it doesn't tell us what the pH is. Together they are great.

7. Do you have many cats that just need an endotracheal tube and not ventilation, like with a laryngeal problem?

Yes.

8. Why do cats and dogs have an end-expiratory grunt?

Good question – not entirely sure. Several thoughts, not sure which is correct.

9. Have you not treated any cases where you actually see edema fluid come back out of the tracheal or the endotracheal tube?

No. I have seen that many times with fulminant congestive heart failure, however not with any tick paralysis patients. Any fluid I have seen has been more of an exudate, related to aspiration pneumonia OR saliva (usually aspirated) because they can salivate excessively and have a weak gag reflex and megaoesophagus.

10. Do you think the tick anti-toxin works (TAS) - is there any evidence?

Yes, I do. I can't imagine a double blinded, controlled study being ethical. I'm not going to NOT treat them. My own dog had a tick a month ago above his eye and he had palpebral paralysis for 3½ weeks! That's without TAS because I was lazy AND because I watched him closely (he didn't develop any other signs). My point is, I haven't seen paralysis last that long (especially without other signs). I assume if I gave him TAS, it would not have lasted that long – but who knows?

Figure 2. **Ixodes holocyclus** *paralysis tick showing scale (Courtesy of Anne Fawcett)*

Comment courtesy of:

Frank Gaschk BVMS
Vetwell Veterinary Group, Brisbane QLD
Grrinninbear Designs (Studio)
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First up, I'm a bog standard general practitioner with an interest in cats, who qualified in WA, so it's hilarious to me to find myself talking about an east coast parasite and disease. But I do practise and practice in Brisbane, so it's not so hilarious when I think about it. I've got no extra post-noms, I just read things that interest me or that I can use in general practice. I do love a cat conference and enjoy trying to communicate information through cartoons or a practical conversation. So, most definitely my knowledge always needs updating and improving, so I thank you for your excellent response. I do know how long things take to type, re-edit and type, so thank you also for taking the time.

I do hope we stir up some discussion with the readers of the CVE *Control and Therapy Series*.

I'm glad we follow a similar protocol; I caught that part. I love knowing what other vets do. Let's then focus on the pulmonary oedema and frusemide.

The dose of frusemide is correct. I'm sure our colleagues will double check the most recent literature. I have used it in only a few cases where I thought I saw pulmonary oedema radiographically and interestingly enough those cats were also on carefully monitored IV fluids based on the earlier steps in the protocol. Quite a few of the cats are dehydrated purely because they haven't been able to get up to have a drink. I don't know if the frusemide in those X-rayed cases made any difference or if it made things worse; there is so much going on in the multi-modal approach to treating a tick paralysis case, it's hard to tell. The poor critters are there, unable to resist our ministrations, and what vet is unable to resist a fully compliant feline patient? So we try to do everything and that may be a mistake in itself.

No cats died in the formulation of the protocol – at least I can say that. I know one cat I recently put to sleep with end stage renal disease after treating him for tick paralysis in 2002. I don't think I used frusemide on him but I remember he almost died of breathing related problems during the tick paralysis treatment. I was only new to Brisbane at the time, so it stays in my memory. I do know I probably used too much ACP because that was the way the Queensland vets treated cats at the time. Thankfully we have moved on from that, even though we cycle through it every few years. That cat only comes to mind because of his recent departure and I do love an anecdote as an antidote to EBM (evidence based medicine) and other dehydrated acronyms… (just teasing Richard Malik, in case he eventually finds himself reading this).

The more severe tick paralysis cases I see these days are sent off for 24 hour care at various emergency centres, so I haven't used frusemide in the last few seasons. Not in this particularly active tick season either.

Like you, I really didn't use it that often at all. Its inclusion in the protocol mainly stems from continuing education talks. The continuing education talks were heavily canine case based and biased toward canine therapeutics. There seemed to be no protocol for treating cats. So the questions and answer sessions winkled out the cat information into my notes, passaged through a few cat practitioners' brains in Brisbane, and then into the protocol that I set out for myself, that found its way into a talk at a local cat practice (2004) and morphed into a talk at a conference based on some retrospective studies... and eventually was published in the *C&T Series*.

Authors' views are not necessarily those of the CVE

You are correct in detecting where the foundation information comes from. It is a handover from question and answer sessions with Dr Fiona Campbell and Prof Rick Atwell. It would be interesting to hear what Rick Atwell and Fiona Campbell would have to say about the pulmonary oedema these days, its genesis and treatment.

Your preventative care instincts are spot on. Protect those kidneys! In the wrong situation, with the wrong practitioner, harm can be done, so if the frusemide has no therapeutic benefit, then it should be removed from the protocol. You've got me thinking of the stressed vet who just looks at a protocol and gives everything, maybe not even taking an X-ray.

I'd just make the observation that general vet practitioners, on X-raying a cat's chest and seeing a pattern that suggests oedema, will have their hand half way to the frusemide before the X-ray is dry. So there may be a better way I could word it? As not mentioning frusemide to a GP well schooled in its use in cardiac disease (unrelated to tick paralysis), combined with its potential risks in tick paralysis patients may prove equally as deleterious to the cat.

It will be exciting to see this dialogue stimulate something beneficial for our patients.

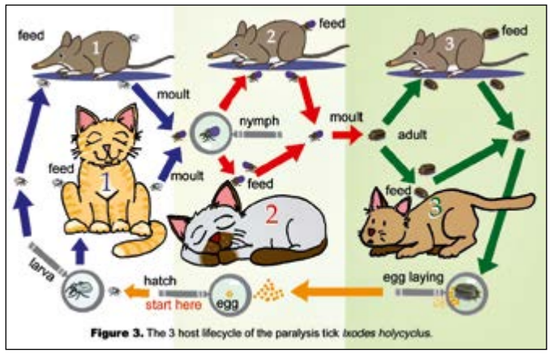

Figure 3. The 3 host lifecycle of the paralysis tick *Ixodes holycyclus*.

Figure 3. Courtesy of Frank Gaschk

Reply No. 2

Dr Kim's cat amongst the pigeons…
Questions asked of Vera Pickering

Kim Kendall
East Chatswood Cat Clinic
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Could I ask your practical opinion on a couple of things?

1. *Do you think the Tick Serum makes a difference to cats? Whether IV or IP? Did you see any difference in survival when there was none available, or did you have reserves?*
2. *What do you do for people with minimal financial resources whose cats (dogs?) need treating? Do you ever NOT give TAS?*

Reply from Vera Pickering

Mona Vale Veterinary Hospital
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My standard tick treatment for cats is as follows **and yes, TAS makes a big difference**

1. Remove tick if seen.
2. Sedate cat with butorphanol/acepromazine as for premed per body weight.
3. At the same time give Histamil Antihistamine Injection® (Ilium; Chlorpheniramine maleate 10mg/mL) (not all the vets do this) and LA Betamox® s/c (repeated every 2 days).
4. Wait 20 minutes and give IP bolus of TAS @ 1mL per Kg or less.
5. Leave cat alone as much as possible in a quiet environment with classic FM playing quietly!
6. Search for ticks/clip as soon as practical but if cat at all anxious give pentobarb IV to do so.
7. Put into vetario – intensive care unit with oxygen if any respiratory distress.
8. Give pentobarb for a good sleep rest if respiratory problem or cat very anxious.
9. Express bladders twice daily.
10. Give s/c Hartmann's Solution after Day 1 as required; usually 100 mL does it – quick and easy and not stressful.

Comments

a. Generally have a quick and uneventful recovery – yes, some die but usually 5% and pretty much early in the season.
b. Northside Emergency Veterinary Service charges about $2,000 a day for a ventilator and their success rate this season has been phenomenal – new ventilator that warms and humidifies the air and pushes it in with the animal's attempt at breathing.
c. Many animals come in yearly for tick paralysis – I do use prednisone sodium succinate but only in those that turn up 1 month later, not next season?!
d. I have seen very few anaphylaxis over the years – maybe 6 and we average 250 cases a year – one of which was my cat after IV TAS in the 1980s and I haven't given cats IV TAS since but do routine IV diluted and slow in dogs.
e. Re cost: TAS 3 mLs for a cat IP does not cost much so not a problem with finances for people. I'm happy to do that as it is ½ mL/kg and probably enough.
f. Toxin in blood only is neutralised not that bound to neuromuscular junction so best to treat early. Once very paralysed with respiratory issues it is going to be hard to deal with and serum is only useful if there is still a tick on the animal pumping in toxin.
g. I can't remember the last time I gave a second dose of serum after a couple of days – don't think it is useful.

Note: I have been treating ticks since 1974, and as a student working with Paul Pemberton in Avalon in 1971 to 1974 we used to bleed some local hyperimmune dogs and use their serum to give to some patients, otherwise we just nursed them through it.

The current word from Rick Atwell is that antihistamines do nothing but somehow I can't bring myself to stop using them considering the majority of our patients return year after year for treatment.

Comment from Kim Kendall

I've been using the Frontline Plus®, but am going back to the Original.

Frontline: (original same as Frontline Plus®)

I had not tuned in to the fact that it can take **72 hours aka 3 days** for fipronil to kill ticks. Obviously VERY important in terms of tick prevention (Vera has also commented that cats or dogs on Program® (lufenuron) don't seem to get tick paralysis as badly (?) and that she recommends applying Frontline a few days early each cycle). BUT ALSO means there is NO POINT ▶

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IN APPLYING IT as a treatment when the animal comes in paralysed or if you can't find a tick – the alcohol base is what kills the tick, but also drops the body temperature and probably scares animals (smell, noise, sticky, cold etc) who are already fearful.

The other 'trick' is to give the cats cypoheptadine (Periactin®) as a premed – 4 orally but may come as injection – as a big part of their anaphylaxis is serotonin mediated rather than histamine mediated, according to Annette Litster who studied cat lungs specifically. It also calms them.

And their shock organ is the lung, not the liver like dogs.

It's all about cats...

Editor's Comment

1 2 Rollover to read Kim Kendall's C&T No. 5147, Dec 2010, Issue 261 'Paralysis tick tricks'

Editor's Note: *We will be canvassing the views of a number of vets in Australia and publishing a 'Round Table Discussion' on the Treatment of Tick Paralysis in our upcoming December 2012 issue. Rob continues the discussion here.*

Comment courtesy of:

Rob Webster BVSc MACVSc (emergency and critical care)
Gold Coast Animal Emergency Service
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I have been a full time emergency vet in the Brisbane and Gold Coast region for the last 12 years. I have just completed an alternative training program in emergency and critical care and I hope to sit the fellowship examination of the Australian College of Veterinary Scientists next year. Our practices focus on referral management of the worst cases of tick paralysis and we use 5 intensive care ventilators between the 2 practices. My clinical and research interest is management of severe tick paralysis and the causes of respiratory failure.

Critical care of patients with tick paralysis

Until one year ago I would have completely agreed with Dr Karina Graham's comment published above. We just did not recognise pulmonary oedema clinically in our patients (dogs or cats) with severe tick paralysis (TP). Last year however, we started performing histopathology on lungs of patients which died from TP, and I was shocked by both the number of patients which had lung disease, and the number where lung disease consisted of pulmonary congestion and oedema. The results of that study are submitted awaiting approval for publication.

Administration of tick anti serum (TAS), and removal of embedded ticks are the 2 most important aspects of treating **tick paralysis**, but severely affected patients ultimately die from **respiratory failure**, and this needs to be managed objectively to improve the chances of successful treatment.

The separation of TP and respiratory failure is a key concept in critical care of affected patients. The neuromuscular syndrome can result in multiple causes of respiratory failure, such that it becomes a guessing game if you just chuck different treatments at the patient hoping that something will work. We need to be looking for the reason behind the respiratory difficulty rather than trying to treat it as part of one disease syndrome.

Dr Rick Atwell's research on large numbers of 'regular' tick paralysis[1] patients did not show any really significant differences in survival of patients regardless of the way we perform the basic treatment strategy. He has looked extensively at how much tick anti serum we (veterinarians) administer, which route of administration we use, whether we pre-medicate the patient, and if we administer anxiolytic medication. He also looked at clipping, bathing, or spraying with frontline. None of these things really made much difference to actual survival, with the only really important thing being administration of 'some' TAS!

I advocate minimal intervention (apart from TAS and tick searches in mild cases of TP), but early, goal oriented measures to support respiration and sustain life when respiratory difficulty is noted.

In general, patients with worsening TP either fail to breathe sufficiently to support life because of advanced neuromuscular paralysis, or they become hypoxaemic because of pulmonary parenchymal disease. If you keep an animal breathing long enough, they almost all eventually recover from tick paralysis. The use of clinical examination, arterial blood gas analysis, and thoracic radiographs will permit logical decision making regarding oxygen therapy and ventilation.

Respiratory Assessment

To recognise deterioration in a TP patient, frequent respiratory assessment is required. This involves physical examination and whichever diagnostic tools are available. The deteriorating patient will exhibit one of 3 types of breathing:-

- **Hypoventilating** patients have reduced respiratory rate and effort. Patients with respiratory rates below 20 and advanced tick paralysis are likely to be exhibiting severe enough hypoventilation to warrant treatment. Quantifying hypoventilation involves either capnography to measure end tidal CO_2 ($ETCO_2$), or blood gas analysis (preferably arterial, but venous is acceptable; it should be about only 5mmHg higher than $PaCO_2$). The critical limit we use for PCO_2 (arterial or venous) is 60mmHg. Levels higher than this indicate severe hypoventilation and mechanical ventilation is required. Some animals will survive much higher PCO_2 levels than 60mmHg, but this is unpredictable. Respiratory acidosis is the pathological problem associated with hypoventilation, and this can be assessed using the blood gas analysis along with the CO_2 level.
- **Restrictive breathing patterns** which involve increased rate and work of breathing are consistent with pulmonary parenchymal disease. TP patients are known to develop pulmonary oedema, and aspiration pneumonia. Hypoxaemia often occurs, but the patient will work very hard to prevent this, and **may respiratory arrest due to fatigue while the SPO_2 level is adequate**. The level of hypoxaemia can be quantified using arterial blood gas analysis or pulse oximetry. The critical limits for these measurements are: SPO_2 >90% and PaO_2>60mmHg. Management of hypoxaemia and increased work of breathing involves oxygen supplementation initially. Mechanical ventilation is required should this not be effective in maintaining adequate oxygen **and** reducing the work of breathing to a sustainable level.
- **Upper airway obstruction** occurs frequently in TP patients especially brachycephalic animals and those intubated for a prolonged period. The classic clinical signs of stridorous breathing and inspiratory dyspnoea may not be apparent if the patient is showing marked paralysis. Additional clinical signs include gagging, retching, regurgitation, and anxiety. The suspected upper airway obstruction can be confirmed if the signs resolve after placement of an endotracheal tube. Management of upper airway obstruction involves maintaining an endotracheal tube under anaesthesia until the signs resolve, or placement of a tracheostomy tube. ▶

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Complexity

Management of one component of respiratory failure can be straightforward, but it is typical for a patient with severe TP to have contributing problems from the upper airway, the pulmonary parenchyma, and have marked hypoventilation as well. Despite its paramount importance, physical examination of the respiratory system in patients with TP may not clearly identify the underlying abnormalities. This is often attributed to the fact that respiratory failure may be caused by multiple concurrent mechanisms, and because the clinical signs of respiratory disease may be masked by the generalised weakness and central respiratory depression which occur with TP. Hypoxaemia may be present due to lung failure, neuromuscular paralysis, or a combination of both. Stridor may not be heard if a patient with upper airway obstruction cannot inspire vigorously because of muscle weakness. Similarly, inspiratory crackles may not be auscultated when a patient cannot inspire deeply. In summary, patients with respiratory failure because of TP may adopt a rapid shallow (restrictive) breathing pattern which diminishes the classical physical signs of respiratory disease. Each patient requires a different life support strategy which should be developed and monitored by repeating the physical assessment and arterial blood gas analysis.

Conclusion

In the next issue of the *C&T* I will be publishing a standardised treatment guide for management of respiratory failure from tick paralysis. It will include how to differentiate the causes of respiratory failure and then monitor lung function during treatment.

PS: Frank I am sorry about the tape, but emergency centres will continue to use the most adhesive type we can buy. The consequences of a catheter which pulls out at an inopportune time can be far more devastating than the difficulties encountered removing the catheter after recovery.

1. Atwell RB, Campbell FE, Evans EA. Prospective survey of tick paralysis in dogs. *Aust Vet J* 2001;79:412-418.

Reply to C&T No. 5240: Applying leaking car technology to senior cats' urination problems

June 2012 Issue 267

C&T No. 5252

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What a great idea! Another suggestion (by a client of mine) is a potting tray – excellent for geriatric cats.

The best and cheapest litter tray money can buy! It's a potting tray – big, and low at the front for those old age pensioner kitties. See:

www.google.co.uk/products/catalog?hl=en&cp=10&gs_id=t&xhr=t&q=potting+tray&biw=1280&bih=556&bav=on.2%2Cor.r_gc.r_pw.&wrapid=tljp1343771966796014&um=1&ie=UTF-8&tbm=shop&cid=13487633063683262648&sa=X&ei=Q1UYUOC1B4Ts0gWPnoHIAw&sqi=2&ved=0CGgQ8wIwAA#

What's YOUR Diagnosis?

C&T No. 5253

Ursina Schiltknecht – DE Feline 2011 participant
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Figures 1&2. Flaccid paralysis in the chin and salivation problems.

A 2½-year-old male neutered cat presented at Dr Katharina Josi's practice in Spiez, Switzerland. The owner told us that the cat all of a sudden had 'tears in its eyes'. After the tear episode stopped, they noticed that the cat's jaw was 'hanging down ever since', causing the cat problems when eating and with salivation. This problem occurred in the evening and the cat was presented to us the next morning after this acute onset of symptoms.

Later, the owner told me that the cat might have been chewing some plants (orchids) before this event.

On clinical examination the cat had a normal body temperature, the neurological exam was normal apart from the chin hanging down in a flaccid paralysis and with no skin sensitivity in this area. Otherwise, the face and eyes were normal.

We put the cat on Convenia® and long-acting steroids and kept the cat at the practice. After 2 days we did some blood tests. Everything showed normal, except there were signs of dehydration so we gave some subcutaneous infusions. FeLV and FIV test were negative.

The tonus of the tongue may have been mildly reduced. We had to 'help' the cat to eat and drink.

There was spontaneous complete remission after some days.

What could be the reason for an acute onset of these symptoms?

Replies or Comments

Please send your Replies or Comments to Lis Churchward, Editor at **elisabeth.churchward@sydney.edu.au** for publication in the following issue. The Winner of the best answer is entitled to a free CVE proceedings – please go to **www.vetbookshop.com** for a list of CVE titles.

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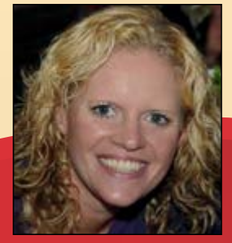
Feline Endoscopy

Part 2 – Gastrointestinal Endoscopy



Andrea Harvey
Co-Tutor of CVE's Feline DE programme

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Andrea Harvey graduated from University of Bristol Veterinary School, UK in 2000 and, after a couple of years in small animal practice, returned to Bristol Vet School to undertake a residency in feline medicine, funded by the Feline Advisory Bureau (FAB). She then remained at Bristol Vet School as FAB Clinician in Feline Medicine until 2010 and has since been doing a variety of work in private referral practices in the UK and more recently at Small Animal Specialist Hospital in Sydney, together with lecturing widely throughout Europe, contributing to numerous textbooks, and working closely with the International Society of Feline Medicine (ISFM) on a variety of initiatives, including development of the 'Cat Friendly Clinic' scheme. Andrea obtained the RCVS Diploma in Feline Medicine and European Diploma in Internal Medicine in 2005 and is a RCVS Recognised Specialist in Feline Medicine and European Veterinary Specialist in Internal Medicine. Andrea has wide interests in all aspects of feline medicine, and is passionate about both providing the best care for her patients, and helping to support other veterinarians to do the same.

Part 1 – Introduction & Respiratory Endoscopy (June 2012, Issue 267)

Part 2. Gastrointestinal endoscopy

Equipment

For gastrointestinal endoscopy, a flexible endoscope is required with 4 way tip deflection (and ability to retroflex > 180˚ in one plane), and must have the mechanical functions of insufflation, irrigation and suction. These functions should always be checked prior to anaesthetising the cat. For cats, a single gastroscope can generally be used in all sized cats for oesophagoscopy, gastroduodenoscopy and colonoscopy.

The larger the biopsy channel, inevitably the larger the biopsies that can be collected, and therefore the more likely they are to be of diagnostic quality. The compromise is, that the larger the distal tip size, the more difficult pyloric intubation will be. The instrument channel should be at least 2mm in order to be able to obtain diagnostic biopsy samples. This is achievable with a 5-6mm distal tip (e.g. Olympus Lucera video-gastroscope GIF-XP260 6.5mm insertion tube with 5mm distal tip and 2mm biopsy channel, Olympus SlimSIGHT GIF-XP180N 5.5mm outer diameter, 2mm biopsy channel, 1.1m working length, and Storz 13820PKS video-gastroscope 5.9mm insertion tube, 2mm biopsy channel and 1.1m working length), and this can be intubated into the pylorus relatively easily even in small cats. A 1.4m working length, 7.8mm outer diameter gastroscope is available with a 2.8mm biopsy channel (Storz, 60714PKS) which will allow procurement of significantly larger biopsy samples. An experienced endoscopist will be able to intubate the pylorus of most larger cats with a 7.8mm distal tip, but an inexperienced endoscopist may struggle, and it may not be possible in smaller cats. The 1.4m insertion tube is quite long for cats, which can make manoeuvring more challenging as the insertion tube outside the patient tends to loop.

The main forceps required for feline gastrointestinal endoscopy are biopsy forceps. Grasping forceps and basket forceps are also useful, for removing gastric foreign bodies, although these are less commonly encountered in cats compared to dogs. Basket forceps are also required for placing percutaneous endoscopically placed gastrostomy (PEG) tubes. There are many different types of biopsy forceps, varying in shape (oval, round), edge (smooth, serrated), fenestrated or unfenestrated, those with a central spike, and swing jaw forceps. The author prefers oval fenestrated forceps without a central spike. The fenestrated cups reduce crush artefact, and the oval cups tend to collect more tissue. Central spikes can damage the biopsy tissue. The type of edge used depends on the toughness of the tissue being sampled. The author tends to mainly use smooth edged cups, but if the tissue is tough, these can slip off, in which case switching to serrated edged cups is advisable. When biopsing the intestine, swing jaw forceps help turn the cups into the intestinal wall, but are much more expensive forceps.

Prior to anaesthetising the patient, ancillary equipment should also be ready (protective clothing, mouth gag, biopsy forceps, formalin pots, sterile saline), and the equipment should be checked that it is fully functioning (including air pump, suction unit and valve, air/water valve, tip deflection, light source image, biopsy forceps, recording equipment).

Uses and indications

The most common reasons for performing upper GI endoscopy in cats is to assess for oesophageal disease in cases of regurgitation or dysphagia (where megaoesophagus has first been excluded on radiographs), painful swallowing, excessive salivation, and for balloon dilation of oesophageal strictures, and to assess for gastric and small intestinal disease in cases of chronic vomiting, unexplained weight loss, unexplained anorexia, haematemesis or melaena, chronic diarrhoea, ultrasonographically detected thickening or loss of layering of gastric and/or intestinal wall, and for PEG tube placement/removal. Unless there is a specific oesophageal disorder (e.g. stricture) which may make advancing beyond this unnecessary, full oesophago-gastro-duodenoscopy should always be performed.

Colonoscopy is indicated for investigation of large intestinal diarrhoea, haematochezia, tenesmus, dyschezia, constipation and investigation of palpable rectal mass or stricture. Upper GI endoscopy should always be performed whenever colonoscopy is being performed, since lesions are rarely confined to the colon even when clinical signs are predominantly large intestinal.

GI endoscopy has the advantages of allowing examination of the mucosal surface of the GIT, being a much less invasive ▶

way of obtaining gut biopsies compared to surgery; the patient can be discharged the same day as the procedure, there is no convalescence or wound healing and immediate corticosteroid treatment can be started if indicated.

Limitations and contraindications

There are relatively few contraindications for gastro-intestinal endoscopy. The main one, that is essential to consider, is where systemic causes of clinical signs have not been excluded and there have been inadequate investigations performed prior to considering endoscopy. Other contraindications include patients that are a poor anaesthetic risk, presence of a bleeding disorder, any patient that has had recent GI surgery, and where food has not been withheld, or the stomach has been found to be full on imaging.

Whilst being a very valuable way of assessing the GI tract, there are limitations of endoscopy. Firstly, it does require the appropriate and fully functioning equipment together with a competent endoscopist, with good biopsy technique, to enable adequate evaluation of the GIT and collection of diagnostic quality biopsies. It is also not able to assess functional and motility disorders, hypersecretory disorders, the whole GI tract, submucosal lesions or intraperitoneal lesions. When endoscopic biopsies have failed to produce a diagnosis, where non-mucosal disease is suspected, or disease is known to be beyond the reach of an endoscope, or where there is evidence of intraperitoneal disease (e.g. mesenteric lymphadenopathy, ascites, hepatic/pancreatic abnormalities), then surgical biopsies are indicated.

Patient preparation

For upper GI endoscopy, patient preparation simply requires withdrawal of food for at least 12 hours prior to the procedure, to ensure that the stomach is empty. If any barium contrast studies have been performed, then GI endoscopy should not be performed for at least 24 hours. For colonoscopy, withdrawal of food for 24 hours is required, in addition to 'cleansing' the large intestine. There are various different laxative protocols, but the author's preference is to administer 10-15mL/kg of polyethylene glycol laxative (e.g. Kleanprep), via a naso-oesophageal tube (just placed for each administration and then removed in between), on 2-3 occasions within the period approximately 4-20hours prior to colonoscopy, and then to administer a sodium citrate rectal enema (Microlax) 2-4 hours prior to the procedure.

Pre-medication prior to induction of anaesthesia is routine (e.g. ACP, buprenorphine combination). Atropine is not used routinely, although some advocate that it can make pyloric intubation easier, and it may reduce the risk of vagally induced bradycardia during the procedure. An IV catheter should be in place, and IV fluids administered throughout the procedure. Induction of anaesthesia with propofol or alphaxan is usually appropriate, maintaining on halothane, isoflurane or sevoflurance with oxygen. Nitrous oxide should not be used since insufflation of the stomach permits diffusion of nitrous oxide and causes gastric overdistension. Intravenous midazolam or diazepam can be useful if there is difficulty intubating the pylorus, or if the cat becomes uncomfortable (e.g. tachypnoeic) during this procedure, or if the GI tract is particularly inflamed.

An ET tube should be placed. Cuffed ET tubes are usually advised to prevent the risk of reflux, however caution needs to be used with cuffed ET tubes in cats as they can easily cause trauma to the trachea, and presence of the cuff usually reduces the size of ET tube that can be passed. The author prefers to use the largest possible uncuffed tube – in most average sized cats, a size 5 uncuffed ET tube can be passed. The tube should be secured in place, and a mouth gag must always be inserted to prevent damage to the endoscope. Pulse oximetry monitoring should be used, and appropriate warming equipment to ensure the cat does not become excessively hypothermic during the procedure.

The cat should be positioned in left lateral recumbency for routine GI endoscopy (upper and lower) (Figure 1), so that the gastric antrum is uppermost allowing air to fill it and make the pylorus more visible, and that the descending colon lies ventrally, which aids intubation of the transverse and ascending colon. For oesophagoscopy where conditions such as megaoesophagus, oesophageal strictures or oesophageal foreign bodies are suspected, the cat should be kept in sternal recumbency with the head elevated in order to reduce the risk of aspiration. For PEG tube placement, the cat is positioned in right lateral recumbency. The endoscopist should be positioned in such a way that they have a good view of the video monitor, but also in a way that the insertion tube outside of the patient is kept in as straight a line as possible to prevent difficulty steering and advancing which occurs if there is looping of the insertion tube outside the patient.

Technique

General technique

As the endoscope is inserted into a viscus, air needs to be instilled to be able to obtain a clear view. The rate of air insufflation needs to be adjusted to ensure adequate inflation without over inflation. The small size of cats makes it easy to overinflate the GIT and this needs to be monitored carefully, by using a low setting on the air pump, ensuring the airhole is not inadvertently covered continuously leading to continuous insufflation, and that the anaesthetist observes and palpates the abdomen regularly to ensure overinflation is not occurring. This is extremely important as overinflation not only makes the endoscopy more difficult, particularly pyloric intubation and biopsy collection, but more importantly will lead to impairment of venous return, and can quickly result in severe cardiovascular and respiratory compromise, in addition to risking perforation of a viscus. Inability to sufficiently insufflate can be the result of a faulty air pump, leaking seal, air escaping from the viscus, or may indicate significant GI pathology.

Steering is achieved through a combination of insertion and retraction of the insertion tube, longitudinal rotation of the insertion tube (torquing), up/down and left/right tip deflection and passive movement as the endoscope follows the wall of a viscus. Attention should be paid to keeping the insertion tube as straight as possible to assist in more accurate steering.

The lens will often become obscured by blood, mucus and GI contents, and this requires flushing by depressing the air/water button. Any fluid pooling in a viscus requires suctioning to examine the underlying mucosa. Air should always be suctioned from the viscus before withdrawing the endoscope.

Technique for upper GI endoscopy

Delay in intubating the pylorus, and insufflation of air, makes pyloric intubation more difficult, therefore it is usual to only quickly visually inspect the oesophagus and stomach on the way down, leaving more complete evaluation to the end of the procedure (except where oesophagoscopy is the main purpose of the procedure). The endoscope is inserted through the upper oesophageal sphincter by applying only gentle pressure and insufflating small amounts of air. Once through the sphincter the tip should be adjusted so that the oesophageal lumen is in the centre of the view, and the endoscope gently advanced to the lower oesophageal sphincter. A quick inspection of the mucosa on the way down ensures that any pathological lesions can be distinguished from iatrogenic damage when closer inspection is performed on withdrawal. In cats, the distal oesophagus has distinct circular folds and the lower oesophageal sphincter may be seen as a star or slit like opening (Figure 2).

The most common pathology observed in the feline oesophagus is oesophagitis, oesophageal ulceration and oesophageal strictures (Figure 3). Biopsies are not routinely taken from the oesophagus as the mucosa is very tough. Masses should be biopsied, but there are rare. With oesophageal strictures, endoscopy can be used to guide balloon dilation, by passing a balloon catheter alongside the endoscope (Figure 4). When the stricture is very narrow it can be difficult to insert the balloon into the stricture and great care needs to be taken when directing the balloon to ensure that the tip does not cause further oesophageal trauma or oesophageal perforation. Further texts should be consulted for more details on this technique.

Once at the lower oesophageal sphincter, the endoscope tip should be angled towards it with continued insufflation and gently advanced through into the stomach. As the endoscope enters the stomach, the junction of the fundus and body of the stomach can be seen with parallel rugal folds on the greater curvature running towards the pyloric antrum (Figure 5). This provides an important landmark in locating the pylorus. The other most important landmark is the angularis incisura (angle of the lesser curvature) with the cardia above, and the pyloric antrum below (Figure 6), and can be located by retroflexing the endoscope tip fully to first locate the cardia (Figure 7) and then slightly reducing the retroflexion to bring the lesser curvature into view. Some insufflation is required to visualise these landmarks, but over inflation will make pyloric intubation difficult, and this is the most common mistake made. In cats, the angle of the lesser curvature is quite acute, and a slide-by technique can be useful for passing the endoscope into the antrum. This involves gently advancing the endoscope along the mucosal surface of the greater curvature; the endoscope tip will be impinging on the gastric mucosa and so red-out will occur, but provided this is moving and the endoscope is not advanced against any resistance, it can be continued to be advanced along the greater curvature until the pyloric antrum comes into view. Ensuring that the pylorus is in the centre of the screen, and suctioning as the insertion tube is advanced towards it, assists with pyloric intubation.

Once through the pylorus the insertion tube is advanced past the cranial duodenal flexure, where red out is likely to occur, and then with intermittent insufflation of air, and deflection of the tip, the lumen of the duodenum should come into view (Figure 8). The endoscope can then be gently pushed along the duodenal mucosa as far as required. With a 1m length endoscope, the proximal jejunum may even be reached in cats. It is common for the mucosa to appear macroscopically normal even when significant microscopic disease is present. Multiple biopsies (approximately 6-10) should therefore always be taken from different regions of the small intestine (see below for biopsy technique). The most common small intestinal disorders in cats are inflammatory bowel disease (Figure 9) and intestinal lymphoma.

Once the duodenum has been assessed and biopsies taken, all the air should be suctioned out as the endoscope is withdrawn. The proximal duodenal flexure should be evaluated on the way out for lesions (e.g. ulcers) before withdrawing the endoscope back into the stomach for more complete evaluation. Retroflexion and rotation of the endoscope along its long axis allows full inspection of the cardia and the fundus. Multiple biopsies (total approximately 6-10) should be taken from all areas of the stomach, in addition to any macroscopic lesions. In cats, the most frequent macroscopic lesions identified are neoplastic disorders, most commonly gastric lymphoma. The mucosa is often pale, lumpy and friable, however this is very variable and there is no pathognomonic appearance (Figure 10).

Technique for colonoscopy

Colonoscopy is more straightforward to perform than upper GI endoscopy, provided that the patient has been adequately prepared. The preparation is essential, otherwise colonoscopy will be a waste of time if significant amounts of faecal material remain within the colon. The insertion tube should be lubricated with KY Jelly (taking care not to get lubricant over the lens), and an assistant is required to gently pinch the anus around the insertion tube to prevent air from escaping. Once inserted into the rectum, air should be insufflated until the mucosa of the descending colon is in view, before advancing further. The junctions between the descending and transverse colon, and transverse and ascending colon can be readily detected as obvious bends, prior to reaching the ileocecocolic junction, identified by the opening into the caecum and the prominent raised appearance of the ileocolic sphincter. It is possible to pass biopsy forceps through the sphincter to biopsy the ileal mucosa. Approximately 6-10 biopsies should be taken from all parts of the colon, prior to suctioning the air out and withdrawing the endoscope and examining the rectum on the way out. ▶

Figure 1 – Patient positioning for upper GI endoscopy. Note that the cat is in left lateral recumbency, the ET tube is secured in place, and a gag and pulse oximeter are being used (courtesy of Great Western Referrals)

Biopsy technique

An assistant is required to operate the biopsy forceps and it is important that they are familiar with how to operate them before starting. Instruments must always be passed through the biopsy channel in a closed position and never forced against resistance. Squeezing too hard to close the cups can break the wire of the forceps so must be avoided. Care must be taken when passing instruments through the deflected tip of the endoscope, as forceful passage can easily damage the inner lining of the instrument channel. The instrument channel also serves as a suction channel, so suction will be much reduced when an instrument is within the channel, and if the instrument channel cap is open. When the scope has passed several intestinal flexures, the forceps can be difficult to open as the wire may be bent, and straightening the endoscope will help with this.

The quality of the biopsies obtained are determined mainly by the size of the forceps (dependent on the size of the scope), and the pressure exerted on the tissue by the operator. This is a big limitation in feline endoscopy as the patient size limits the size of endoscope, and therefore size of biopsy forceps that can be used. Therefore in order to obtain diagnostic quality biopsies, there is no room for poor operator technique. Exerting maximal pressure can be achieved by positioning the biopsy cups perpendicular to the tissue being sampled (Figure 9). Being able to do this effectively, and knowing how much pressure can safely be applied, comes with experience. Deflating the viscus before biopsy also helps increase the size of the sample by reducing stretching of the mucosa.

Once the biopsy has been collected the forceps are removed from the biopsy channel, the cups opened and immersed in 10% formalin releasing the tissue. The forceps must be rinsed in water before reintroducing into the endoscope. Alternatively, tissue samples can be laid on card or tissue cassettes prior to being placed in formalin.

Complications and aftercare

Serious complications associated with GI endoscopy are rare. The most common problem that care should be taken to avoid is gastric overinflation, which as well making the endoscopy more difficult, particularly pyloric intubation and biopsy collection, also leads to impairment of venous return, and can quickly result in severe cardiovascular and respiratory compromise. It is important to avoid overdistension, and to remember to remove air with suction prior to withdrawing the endoscope at the end of the procedure. Other potential complications include bradycardia – which may occur as a vagovagal reflex, usually in cats with severe GI disease, and is usually resolved with atropine – and gastrointestinal perforation which, when it occurs, is usually the result of severe GI disease, accompanied by forceful use of the endoscope without adequate visualisation, or poor biopsy technique. Occasionally with severe GI disease, perforation can occur just with overinflation. Emergency laparotomy is required if perforation is evident or pneumoperitoneum develops. Significant mucosal haemorrhage associated with biopsying is rare, and it is not necessary to routinely prescribe gut protectants following endoscopy. Most cases of routine endoscopy require no specific aftercare.

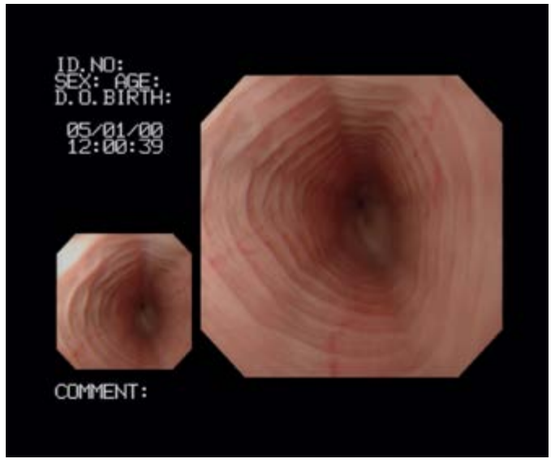

Figure 2 – The endoscopic appearance of the normal feline oesophagus (courtesy of University of Bristol)

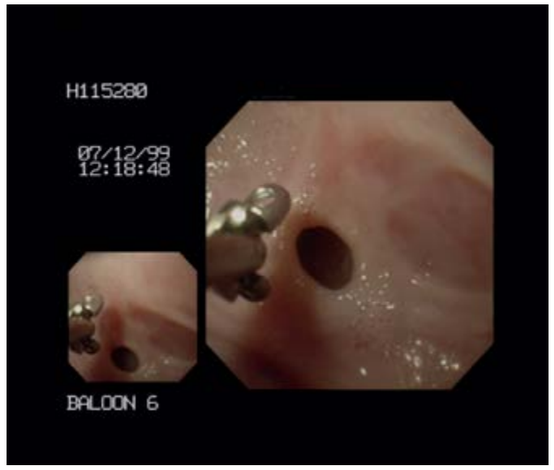

Figure 3 – Endoscopic view of an oesophageal stricture (courtesy of University of Bristol)

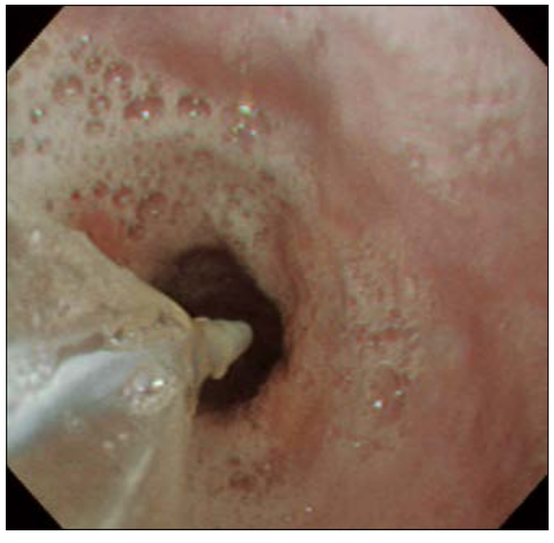

Figure 4 – A balloon catheter is being advanced through the lumen of the oesophageal stricture to allow balloon dilation (courtesy of University of Bristol)

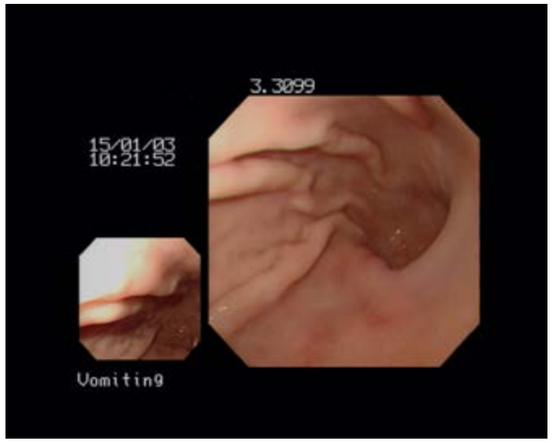

Figure 5 – The normal endoscopic appearance of the area at the junction of the body/fundus of the stomach on the greater curvature. Note that the parallel rugal folds are running towards the pyloric antrum beneath the angularis incisura (courtesy of University of Bristol)

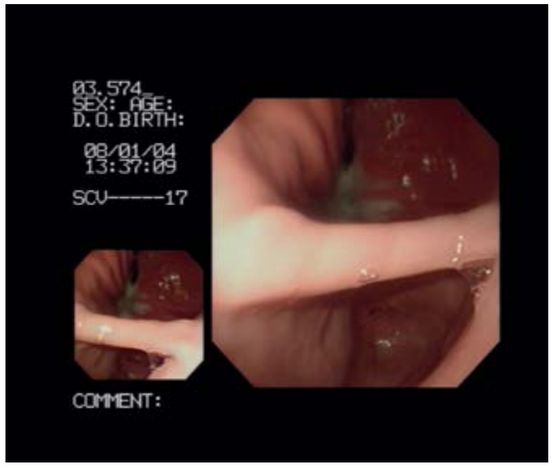

Figure 6 – Endoscopic view of the angularis incisura with the fundus/cardia dorsally and the antrum ventrally (courtesy of University of Bristol)

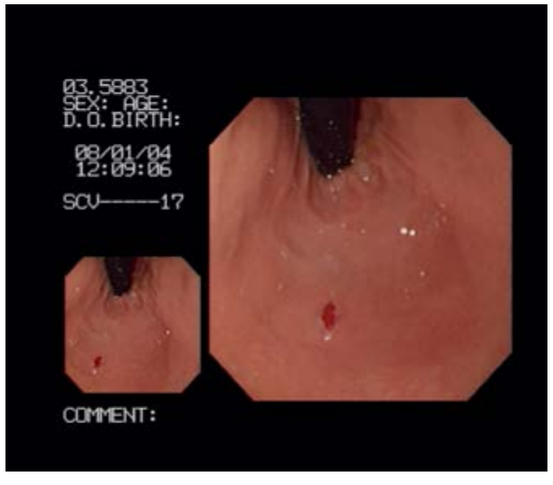

Figure 7 – Here the gastroscope is being retroflexed to examine the cardia (courtesy of University of Bristol)

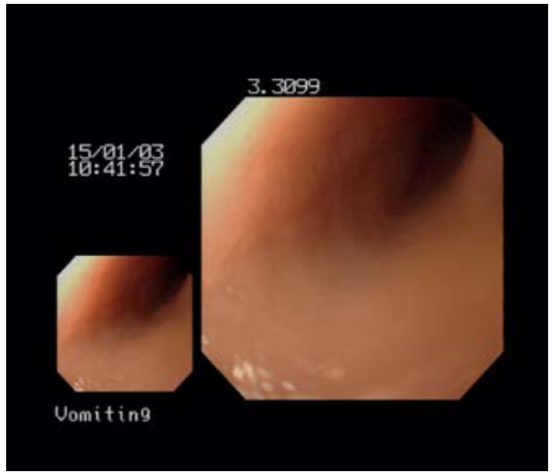

Figure 8 – Endoscopic appearance of the normal feline duodenum (courtesy of University of Bristol)

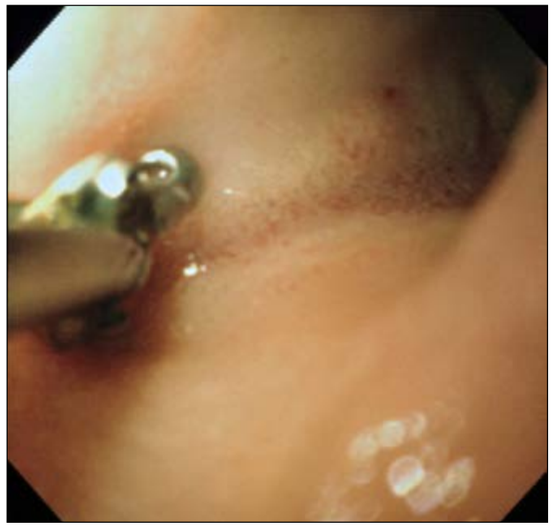

Figure 9 – Duodenum from a cat with inflammatory bowel disease, illustrating biopsy technique, positioning the biopsy cups perpendicular to the mucosa (courtesy of University of Bristol)

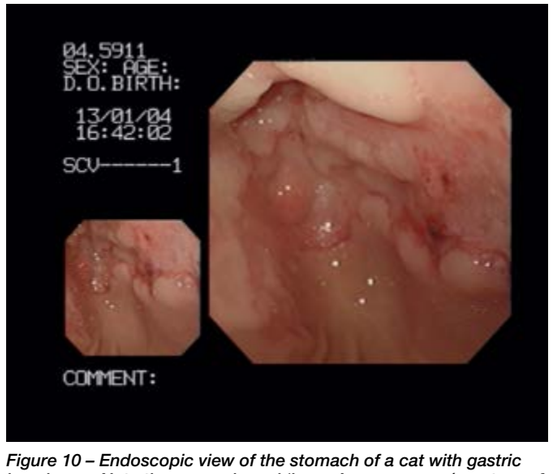

Figure 10 – Endoscopic view of the stomach of a cat with gastric lymphoma. Note the very pale and 'lumpy' appearance (courtesy of University of Bristol)

What's YOUR differential diagnosis and diagnostic plan?

C&T No. 5254

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Donna is a CVE Feline DE alumnus who generously contributed this case to the DE Feline forum.

We invite C&T Members/Readers to formulate a systematic reply to this case presentation. There is an 'illness script' in this case i.e. a pattern strongly suggestive of a particular diagnosis. We would like you, the reader, to submit a differential diagnosis and a diagnostic plan based on the information below provided by Donna. The winner of the best answer will win a CVE publication. (Go to www.vetbookshop.com to view our titles.)

Figure 1. Toe before cleaning

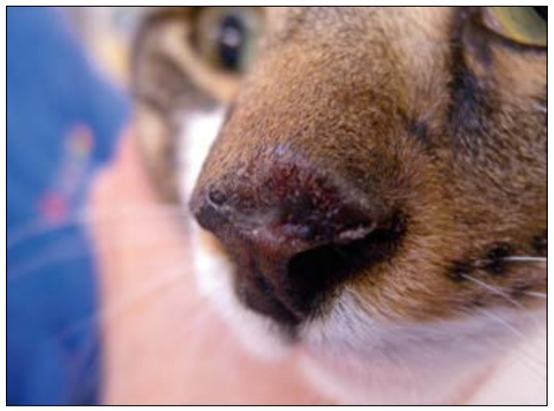

Figure 2. Nose

Figure 3. Toe 2

Figure 4. Toe 3

Figure 5. After cleaning toe

Figure 6. Toe showing ulceration

'Marley' is a 1-year-old desexed and fully vaccinated DHS who had access to outdoors. His owner noticed some swelling and crusting of 3 toes (all on different feet) and reported that Marley was licking his toes but was not lame and was otherwise well i.e. eating well etc.

A physical exam was unremarkable and aside from the toes I noted a demarcation of the skin on his nose (see Figure 2) which I felt was soon going to ulcerate. On clipping and cleaning the affected toes there was severe swelling and ulceration and we removed lots of thick inspisated pus from the crevices of the nail fold. A swab was sent for culture and a smear for cytology at the cat was commenced on doxycycline.

My concern was that it may have been an immune mediated condition with involvement of the nose and likely the next step would be biopsy and retroviral testing.

Replies or Comments

Please send your Replies or Comments to Lis Churchward, Editor at **elisabeth.churchward@sydney.edu.au** for publication in the following issue.

MAJOR WINNER

A cat with jaundice

C&T No. 5255

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Signalment: A 2 ½-year-old male neutered Turkish Angora.

History: 'Mac' presented with a 3-4 week history of lethargy and dullness at home. He had a poor appetite but was still drinking, and the owners felt that he had lost weight. He was an indoor cat so had no access to toxins, was up to date with his vaccinations (FHV, FCV, FPLV), and had recently been wormed.

Physical Exam: Mac was quiet, alert and responsive on exam. The weight loss was confirmed, with a decrease from 4.74kg to 4.17kg, and he currently had a BCS score of 2/5. Profound jaundice was evident around Mac's ears, sclera and mucus membranes. No other abnormalities were discovered in the rest of the physical exam.

Problem List and Differentials: Jaundice, weight loss, poor appetite, lethargy. The primary problem was considered to be the cause of the jaundice with the other problems secondary to this.

Differentials for jaundice included: pre-hepatic causes – haemolytic anaemia, hyperthyroidism; hepatic causes – inflammatory hepatopathies, hepatic lipidosis, FIP, sepsis, neoplasia, hepatotoxicity, toxoplasma, amyloidosis, telangiectasis; post-hepatic causes – bile duct obstruction (pancreatitis, cholecystitis, cholelithiasis, hepatobiliary mass, duodenal mass) or bile duct/gall bladder trauma.

The history and age of the cat made hyperthyroidism and neoplasia unlikely. The owners were sure there had been no access to toxins although hepatotoxicity could not be ruled out at that stage. There was no evidence of intra-abdominal haemorrhage, thus making amyloidosis and telangiectasis less likely. There was no history of abdominal trauma as he hadn't escaped from the owners' house.

The most likely differentials were likely to be haemolytic anaemia, inflammatory hepatopathy (acute or chronic neutrophilic, or lymphocytic cholangitis), hepatic lipidosis, pancreatitis, cholecystitis or cholelithiasis. The noneffusive form of FIP was also possible, although he didn't appear to have any ocular or CNS signs.

Investigation: Bloodwork was initiated, including a full biochemistry profile to evaluate the severity of bilirubinaemia and his liver enzymes, and also to get a general picture of his health status. Haematology was carried out to assess any anaemia present, and electrolytes were checked in view of his poor appetite.

A FeLV/FIV test may also have been a potential test to run at this point although his history as an indoor cat would make these infections less likely.

Assessment: The blood results for Mac are as follows:-

TEST	RESULT	RANGE
UREA	5.9	5.7-12.9 mmol/L
TP	67	57-89 g/L
TBIL	**174**	0-15 umol/L
PHOS	1.57	1-2.42 mmol/L
GLU	5.04	4.11-8.83 mmol/L
GLOB	38	28-51 mmol/L
CREA	127	71-212 umol/L
CHOL	4.3	1.68- mmol/L
Ca	2.52	1.95-2.83 mmol/L
AMYL	1048	500-1500 U/L
ALT	**374**	12-130 U/L
ALKP	**127**	14-111 U/L
ALB	29	22-40 g/L
Na	161	150-165 mmol/L
K	4.9	3.5-5.8 mmol/L
RBC	**4.9**	5-10 x10^9/L
HGB	11.7	9-15.1 x10^{12}/L
HCT	**25.9**	41-58 %
WBC	**4.13**	5.5-19.5 x10^9/L
NEU	**0.37**	2.5-12.5 x10^9/L
MONO	0.36	0.15-1.7 x10^9/L
EOS	0.08	0.1-0.79 x10^9/L
BASO	0.0	0.0-0.1 x10^9/L
LYM	2.94	0.4-6.8 x10^9/L
PLT	445	175-600 K/uL

Mac's electrolytes were within normal limits and his biochemistry revealed markedly raised bilirubin and elevated liver enzymes (ALT much more than ALKP). His urea was at the very low end of the spectrum, but still within normal limits. The haematology showed mild anaemia with a lowered haematocrit and red blood cell count slightly below the normal range. Neutropenia was also present.

The mild anaemia would not be enough to explain the markedly raised bilirubin so is more likely to be anaemia of a chronic disease and so secondary to the main problem. The high liver enzymes suggested a hepatic or post-hepatic cause to Mac's problems. The neutropenia could have been due to viral infection, autoimmune destruction, or excessive tissue demand, and suggested possible FIP or retrovirus infection, although his globulin level is still normal.

Management

At this point, I would proceed to diagnostic imaging – specifically ultrasound – to check for biliary obstruction and also to evaluate the liver and pancreas. I would also like to send off a specFPL test to check for pancreatitis in case the ultrasound results were inconclusive. I would admit Mac for supportive treatment in the form of fluids and analgesia.

However, in Mac's case it was decided to investigate the anaemia in more detail so bloods were taken and sent to the lab for a Coombs test and also a PCR for *Mycoplasma haemofelis*. Mac was booked in for a scan and sent home. ▶

When Mac returned to the surgery, the owners reported that he was still the same. A scan of his liver revealed a slightly thickened bile duct. The pancreas was not imaged at this time, although there is no note of whether it couldn't be seen or just wasn't looked for. The blood results returned from the lab with a normal red blood cell count and haematocrit, a mild neutropenia, and negative results for the Coombs test and *M. haemofelis*.

Assessment

At this stage, biliary obstruction was ruled out as was haemolytic anaemia, so the most likely causes were cholangiohepatitis, hepatic lipidosis and pancreatitis. The retroviruses and FIP could not be excluded at this stage either.

Management

After discussion with the owners, it was decided to proceed with an exploratory laparotomy and liver biopsies. A FeLV/FIV snap test was carried out prior to the surgery, and the results were negative. Mac was commenced and maintained on fluids through the surgery and also given Amoxycillin/Clavulanate (Augmentin, 37mg IV).

The liver was reported at the time to look quite mottled in appearance with greyish-green and darkened areas throughout. No evidence of the granulomas typical of noneffusive FIP was found. Biopsies of the liver and pancreas were taken and it was mentioned that the mesenteric lymph nodes were enlarged. The intestines were examined grossly but no biopsies were taken although the reason for this is unclear. The risks of gut wound dehiscence appears low in the literature, and the variability of the gross tissue appearance with infiltrative gut disease is marked, leading to the opinion that gut biopsies should be taken almost every time endoscopy or exploratory laparotomy is performed. With the potential risk of Mac having triaditis, it makes more sense to take all the potential samples required while the surgery is being undertaken, rather than having further surgery later to collect anything more.

A nasooesophageal (NO) tube was placed to aid Mac's nutrition over the next few days once his abdominal wound was closed. Mac developed respiratory arrest while recovering from the anaesthetic and became cyanotic. He was reintubated and IPPV was carried out, whereupon his colour improved and spontaneous breathing began again.

Mac was given marbofloxin (Marbocyl®, 8.2mg SC), buprenorphine (Vetergesic®, 0.09mg SC), cefalexin (Convenia®, 32mg SC) and also clindamycin (Antirobe® 25mg bid by mouth) in case of any infection and in view of his neutropenia, and also to aid in pain relief. Feeding through his NO tube was carried out also using Royal Canin Convalescence Support, chosen for its ability to be used for tube feeding. The antibiotics Mac was given all have similar spectrums of activity, and it would have been more logical to have metronidazole as an extra antibiotic to cover all possible bacterial quadrants instead of all 3 that have been mentioned.

The following day, Mac seemed brighter in himself and was starting to eat with a bit more enthusiasm. He became very aggressive when his IV catheter was to be taken out, however, necessitating sedation although no problems with respiratory arrest were encountered this time.

The results came back from the lab as mild to moderate lymphocytic cholangitis, an infiltrative disease characterised by excessive numbers of lymphocytes invading the portal tracts. There was no evidence of pancreatitis histologically and his specFPL test came back as 1.3 (normal range less than 3.5). As Mac seemed very bright and was now eating well, he was discharged.

At this point, it is recorded on the clinical notes that discussions were had with IDEXX who recommended finishing the antibiotic course, maintaining the neutraceuticals, and retesting the bloods in one week. If the hepatic enzymes and bilirubin were still raised, then they would consider prednisolone therapy. This does seem unusual given that a definitive diagnosis of lymphocytic cholangitis had been reached and the treatment for this is largely agreed to be immunosuppressive doses of corticosteroids. It also seems strange that there seems to be a suggestion that the condition should have resolved within a week when most cases of liver disease appear to take weeks to fully resolve.

However, Mac was sent home with instructions to return in one week. His medications consisted of marbofloxacin (10mg sid by mouth) to continue his antibiotic therapy, ursodeoxycholic acid (Destolit®, 37.5mg sid by mouth) to promote bile flow and also for its anti-inflammatory and immunomodulatory effects, and Samylin®, a commercial hepatic support preparation containing SAMe (to restore glutathione levels and decrease oxidative damage), Silybin® (for antioxidant effects), and vitamin E (for antioxidant effects). He was also sent home on Royal Canin gastrointestinal diet. This was chosen instead of the hepatic support diet as it came in a wet form, and was indicated for hepatic conditions, containing a moderate level of protein (although higher than the hepatic diet), increased levels of electrolytes and also essential fatty acids.

Management (1 week on)

When Mac returned, he again required sedation and his blood results were as follows:

TEST	RESULT	RANGE
UREA	6.6	5.7-12.9 mmol/L
TP	63	57-89 g/L
TBIL	**101**	0-15 umol/L
PHOS	1.41	1-2.42 mmol/L
GLU	4.71	4.11-8.83 mmol/L
GLOB	33	28-51 mmol/L
CREA	136	71-212 umol/L
CHOL	4.09	1.68- mmol/L
Ca	2.52	1.95-2.83 mmol/L
AMYL	999	500-1500 U/L
ALT	**286**	12-130 U/L
ALKP	**173**	14-111 U/L
ALB	29	22-40 g/L
Na	163	150-165 mmol/L
K	4.2	3.5-5.8 mmol/L
RBC	**4.6**	5-10 x10^9/L
HGB	10.1	9-15.1 x10^{12}/L
HCT	**26.2**	41-58 %
WBC	**4.71**	5.5-19.5 x10^9/L
NEU	**1.16**	2.5-12.5 x10^9/L
MONO	0.33	0.15-1.7 x10^9/L
EOS	0.26	0.1-0.79 x10^9/L
BASO	0.01	0.0-0.1 x10^9/L
LYM	2.94	0.4-6.8 x10^9/L
PLT	510	175-600 K/uL

The hepatic enzymes and bilirubin were still markedly raised and the anaemia and neutropenia were still present. Mac did seem brighter in himself at home and had a better appetite although was still obviously jaundiced on exam. His weight was the same as the previous week.

Assessment: Although Mac was doing well clinically, and his liver values had improved slightly, the disease process appeared unresolved. This seems likely to be due to the fact that the underlying cause had not been addressed. Prednisolone was added to Mac's medications at this time at 2.5mg sid by mouth, and he was to continue with his UDCA. The owners were having a bit of trouble getting Mac to take the Samylin® and reported that he seemed a bit more temperamental at home. They were encouraged to persevere if possible but that the prednisolone and UDCA were of higher importance.

Follow-Up: One month later, Mac appeared to be doing very well. His weight had increased to 4.3kg and he was no longer jaundiced. He was bright and eating well at home but he did seem to be more bad tempered. He was unable to be examined conscious in the surgery and so was sedated to repeat his bloods. Physical exam was normal at this time.

TEST	RESULT	RANGE
UREA	6.4	5.7-12.9 mmol/L
TP	66	57-89 g/L
TBIL	8	0-15 umol/L
PHOS	0.92	1-2.42 mmol/L
GLU	4.93	4.11-8.83 mmol/L
GLOB	32	28-51 mmol/L
CREA	127	71-212 umol/L
CHOL	3.97	1.68- mmol/L
Ca	2.53	1.95-2.83 mmol/L
AMYL	1397	500-1500 U/L
ALT	**185**	12-130 U/L
ALKP	74	14-111 U/L
ALB	35	22-40 g/L
Na	159	150-165 mmol/L
K	3.3	3.5-5.8 mmol/L
RBC	6.7	5-10 x10^9/L
HGB	33.8	9-15.1 x10^{12}/L
HCT	45	41-58 %
WBC	8.9	5.5-19.5 x10^9/L
NEU	3.39	2.5-12.5 x10^9/L
MONO	0.3	0.15-1.7 x10^9/L
EOS	0.28	0.1-0.79 x10^9/L
BASO	0.0	0.0-0.1 x10^9/L
LYM	2.9	0.4-6.8 x10^9/L
PLT	286	175-600 K/uL

Mac's blood results were much improved, with resolution of the anaemia and neutropenia and only slight elevation of the ALT in the biochemistry panel. On the basis of this, it was decided to wean the dose of prednisolone down over the next 2 weeks and then to stop all his medications.

Two months later, the owners reported that Mac had deteriorated and prednisolone therapy was recommended at 2.5mg bid by mouth. Despite this, the owners reported that he was becoming more aggressive with them at home, to the extent that they requested euthanasia 2 weeks later.

Discussion: Lymphocytic cholangitis is recognised histologically by an infiltration of lymphocytes into the portal tracts, and is thought to have an immune-mediated aetiology. It is usually a chronic condition which develops slowly and cats may have had vague clinical signs for months prior to presentation. Younger cats tend to be affected by this condition and it may develop with concurrent pancreatitis and/or inflammatory bowel disease. Pancreatitis was ruled out with Mac although IBD could not be definitely excluded due to the lack of biopsies. Potentially biopsies may not diagnose all conditions – e.g. if an unaffected portion of tissue is sampled – although Mac was not showing other symptoms and the response to his treatment suggests that these other conditions were not features in this case.

The treatment for this condition is considered to be steroids, supported by a wealth of clinical experience. Some reports claim that they have found no benefit from using steroids however and would use UDCA as the first choice of treatment.

The prognosis for cats with lymphocytic cholangitis is variable, as response to treatment can be difficult to monitor. A mean survival time of 3 years has been mentioned. The outcome of this case was very disappointing, and it would have been worthwhile to try and investigate Mac's deterioration to prove it was a relapse of the condition rather than just initiating steroids. There was no note of aggression in the literature as an effect of the condition and some cats can become less easy to manage after being hospitalised and undergoing various procedures in the surgery. The problem for the owners was his increasing aggression towards them at home, and to not investigate this or suggest behavioural therapy feels as though we have failed to manage this in the best way for the cat and the owners.

Feline Q&A: What dose of tramadol do you suggest in cats?

C&T No. 5256

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Q. What dose of tramadol do you suggest in cats? I recently saw an old cat with arthritis on meloxicam but it was still in pain. However, it responded nicely to oral Temgesic® for a few days.

A. Courtesy of:

Richard Gowan
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I wouldn't expect the cat's pain to come under control in the short term as arthritis is a chronic degenerative process. It can take weeks to months to control pain adequately in some patients. Some are just poor responders. Part of the lack of response may be the chronicity of the disease and the neurobiological changes that have occurred, as well as degeneration to muscles, nerves and tendons etc. Some cats feel better on low doses of Metacam® (+/- 0.02mg/kg SID PO), but they don't show it in marked changes to mobility for quite a while longer.

Try low dose PO Temgesic® SID (0.01-0.02mg/kg) as an adjunctive for a week or so to the Metacam®. This can be used in lieu of NSAIDs. We do however use a low dose compounded combination of tramadol and Gabapentin SID to BID (5mgT/10mgG small cats) (8mgT/15mgG for larger cats) with the Metacam® and have found this to 'work' well without the adverse taste and dysphoric effects.

Chronic pain control is an art rather than a science; what works for one patient may not suit another. But it takes a lot longer than I think we realise to reverse the chronic neurobiological pain process that takes place in our patients with OA.

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WINNER

Treatment of Diabetes Mellitus in 2 cats

C&T No. 5257

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View Tracey's excellent powerpoint: How to manage your diabetic cat. (Also available at **www.cve.edu.au/CandT/resources**)

Introduction: Two cats were presented to the Alice Springs Veterinary Clinic at similar times, both diagnosed with Diabetes Mellitus. The treatment of these 2 cases was approached in a similar fashion, in an attempt to establish a set protocol for treating diabetic cats with Glargine insulin within the clinic. A set protocol agreed upon by all vets at the clinic would allow consistent management of all feline diabetic cases presented to the clinic in the future.

Case Histories:

'Barkley' – a16-year-old male neutered domestic shorthair was initially presented for mild weight loss. There had been no change in appetite or water intake noticed by the owner at this initial presentation. Physically, Barkley appeared normal except for a slight systolic murmur and periodontal disease. One month later, Barkley developed vomiting and diarrhoea and was now no longer eating and appeared dull and lethargic. On physical examination he was found to be dehydrated, pyrexic and had lost a further 500g in the past 4 weeks.

'Dennis' – 11-year-old male neutered domestic longhair, was diagnosed as being diabetic 12 months prior to this episode. After his original diagnosis, the owner elected not to treat Dennis, but would not euthanase either. When he presented again 12 months later, Dennis was dull, inappetant and had been vomiting for the past 48 hours. He had also developed some diarrhoea during this time. Physically, Dennis was normothermic, very thin and dehydrated. He had a respiratory rate of 72 breaths per minute, and a gallop rhythm was detected on auscultation of the heart.

Differential Diagnosis: Both cats had very similar presentations. Although one cat had already been diagnosed as diabetic, the following differential diagnosis list could have been considered in both cases:

Metabolic:
- Diabetes Mellitus
- Hyperthyroidism
- Renal Disease

Mechanical:
- Gastrointestinal foreign body
- Intussusception

Neoplastic:
- Gastric or small intestinal neoplasia

Inflammatory/Infectious:
- Acute pancreatitis
- Gastritis/Gastric ulcer
- Septicaemia/bacteraemia
- Panleukopaenia

Toxic:
- Lily ingestion/plant toxicity

Investigations: Blood was taken for biochemistry and haematology to be done at an external laboratory in Dennis's case, and was suggested in Barkley's case, but not accepted by the owner. An in-house glucometer reading immediately confirmed that Barkley was diabetic.
As Dennis was quite unwell at time of presentation, in-house biochemistry was run immediately, as results from the external laboratory would take 24 hours to return. Urine was collected in both cases to check USG and the presence of ketonuria, and a T4 level was run to rule out the presence of hyperthyroidism.

Results for Dennis - In-house Biochemistry	
ALB	**34 g/L (23 – 39)**
ALKP	45 U/L (14 – 111)
ALT	186 U/L (12 – 130)
AMYL	1011 U/L (200 – 1500)
UREA	44.6 mmol/L (5.7 – 12.9)
Ca	2.75 mmol/L (1.95 – 2.83)
CHOL	8.48 mmol/L (1.68 – 5.81)
CREA	320 umol/L (71 – 212)
GLOB	51 g/L (28 – 51)
GLUCOSE	>38.11 mmol/L (3.94 – 8.83)
PHOS	2.87 mmol/L (1.00 – 2.42)
TBIL	9 umol/L (0 – 15)
TP	84 g/L (57 – 89)
Na+	151 mmol/L (150 – 165)
K+	3.9 mmol/L (3.5 – 5.8)
Cl-	111 mmol/L (112 – 129)

Urinalysis	
Glucose	++++
Bilirubin	Negative
Ketones	Negative
Blood	++
pH	6.0
Protein	+
Urobilinogen	Negative
USG	1.018

T4	
Total T4	**18.9 nmol/L (15 – 43)**

Biochemistry

Test	Result	Ref	Test	Result	Ref	Test	Result	Ref
CK	U/L 192	50-200	Lipase	U/L	3-105	S.Bile 1)	m.mol/L -	0-20
AST	U/L 36	26-43	Amylase	U/L	-	S.Bile 2)	m.mol/L -	0-20
ALT	U/L 72	6-83	Glucose	m.mol/L 26.7	3.8-6.1	! Total Protein	g/L 89	54-78
Alk Phos	U/L 56	25-93	BOHB	m.mol/L 0.42	0.00-0.05	Albumin	g/L 34	21-39
GGT	U/L < 1	1-5	Chol	m.mol/L 7.5	2.50-3.90	! Globulin	g/L 55	25-50
GLDH	U/L -	-	Trig.	m.mol/L -	0.6-1.2	A/G Ratio	0.62	0.50-1.2
Bilirubin T	u.mol/L < 2	2-17	Na	m.mol/L 154	145-156	Ca	m.mol/L 2.56	1.50-2.6
Urea	m.mol/L 18.1	7-10.7	K	m.mol/L 4.2	3.8-5.2	P	m.mol/L 1.71	1.40-2.5
Creat	u.mol/L 109	70-159	Na/K Ratio	36.67	>	Mg	m.mol/L -	0.60-1.0
Uric Acid	m.mol/L	-	CL	m.mol/L 115	110-123	Urine SG	-	-
			TCO2	m.mol/L	17-24	Haemolysis Index	-	-

Comments: The hyperglycemia with marginal ketonemia is consistent with the previous diagnosis of diabetes mellitus. The hypercholesterolemia may also be associated with the DM. The elevated urea with normal creatinine suggests increased protein catabolism rather than true decrease in GFR. No overt hepatic disease is present.

Haematology

Test	Result	Ref	Test	Result	Ref	Test	Result	Ref
HB	g/L 110	80-150	WBC	x 10^9L 10.0	5.5-19.5	PT	SECS	< 12
HCT	L/L 0.36	-	Neuts	% 69	6.90 2.5-12.5	PTT	SECS	< 15
PCV	L/L	0.24-0.45	(Bands)	% 0	0.00-0.30	FIA		
RBC	x10^{12}L 6.6	5.0-10.0	Lymphs	% 21	2.10 1.5-7.0	Clot		
MCHC	g/L 308	300-360	Mono	% 6	0.60 0.00-0.85	Lipaemia		
MCH	pg 17	13-17	Eosin	% 4	0.40 0.00-1.50	Haemolysis		
MCV	1L 55	39.0-55.0	Basophil	% 0	-	Jaundice		
Retics (Corr)	%		Other	% 0	-	RBC Aggl		
T.S Protein	g/L	60-80	Retics ABS	%	x 10^9L			
Nuc RBC	/100 WBC0		Fibrinogen	g/L	1-4			
ESR 20mm	mm		Platelets	x 10^9L	300-700			
ESR 30mm	mm							

Comments:
WBC: Cell types appear normal
RBC: Normal
Platelets: Normal

Results for Barkley

Blood Glucose	
Blood Glucose	21.2 mmol/L

Urinalysis	
Glucose	++++
Bilirubin	Negative
Ketones	Negative
Blood	Negative
Protein	Negative
Urobilinogen	Negative
USG	1.030

T4	
Total T4	25.7 nmol/L (15 – 43)

Barkley was diagnosed as suffering from Diabetes Mellitus based on the presence of hyperglycaemia and glucosuria. He was not ketoacidotic or ketonuric however, and was classified as being in a stable state. Renal disease and hyperthyroidism were ruled out. The source of the pyrexia was not found, however he was treated with a course of amoxicillin/clavulanic acid and the pyrexia resolved within a few days. It is possible that this may have been related to his periodontal disease, which was quite severe. ▶

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Dennis presented in a more deteriorated state than Barkley with marginal ketonaemia, although he was not ketonuric at this point either. While Barkley did not require initial stabilisation in hospital, Dennis did, and he received intravenous fluid therapy to correct his hydration status, and was started on Glargine in hospital at 0.5IU/kg. Twelve hourly insulin doses, blood glucose curves and serial electrolyte measurements were carried out in hospital and supportive treatment (including potassium supplementation and intravenous fluid therapy) was provided.

Once stabilised, Dennis was discharged and management of his diabetes by his owner began. Barkley was also started on home management around the same time.

Discussion: These cases presented the first opportunity for vets at the Alice Springs Veterinary Clinic to treat a diabetic cat with Glargine insulin. Previously, Caninsulin® was always used in feline diabetic patients in our clinic. Glargine is a long-acting insulin analogue, which can be administered once or twice daily. The advantage of such an insulin over others is that it 'provides excellent duration of action in cats with inadequate duration of action using intermediate-acting insulins'[1]. This extended duration of action means better glycaemic control and therefore higher remission rates than lente, NPH or ultralente insulins[2].

The possible disadvantage of Glargine is that it is 100 IU/mL, as opposed to Caninsulin, which is 40 IU/mL. The dose required for most cats is often only 1-4 IU, and it can be difficult to administer less than 2 IU accurately, even with a 0.3mL insulin syringe[1]. However, expected duration of action is more important than concentration when it comes to achieving good glycaemic control[2].

Barkley was started on a dose of 2.5IU of Glargine (0.5 IU/kg) BID, and changed to a low carbohydrate, high protein diet (Hills Prescription Diet m/d®). Dennis was also started on an initial dose of 0.5 IU/kg, however the owner could not be convinced to place Dennis on a prescription diet, and continued to feed him commercial cat food and prawns. Evidence suggests that a high protein, low carbohydrate diet (6 – 12% of calories from carbohydrates) is best for diabetic cats, because it lowers blood glucose and insulin requirements, and may increase the diabetic remission rate[2].

In order to establish a set protocol for treating all diabetic cats in the future with Glargine, the following treatment plan was established for both cats.

Rather than performing glucose curves in hospital, which proved to be a very stressful environment for both cats, pre-insulin blood glucose readings were to be taken by the owners at home, and the dose of Glargine was to be altered depending upon this pre-insulin blood glucose reading, according to the following schedule:-

Blood Glucose (mmol/L)	Change to Glargine Dose
> 18	Increase by 0.5 IU (If current dose is < 2IU)
	Increase by 1.0 IU (If current dose is > 2IU)
10 – 18	Increase by 0.5 IU
6 – 10	Leave insulin dose the same
3 – 6	Decrease by 0.5 IU
< 3	Give no insulin AND decrease the next dose by 1.0 IU

The owners were required to record daily pre-insulin blood glucose readings (taken only before the morning insulin dose), and the dose of insulin administered. Each week, these recordings were reviewed by the veterinarian in charge of the case, to allow us to determine how well the protocol was working. Water intake and urine glucose were also monitored daily by the owners.

We found that although the owners soon became very proficient at taking blood glucose readings and administering the insulin, the constant changes to the Glargine dose were becoming quite stressful, especially for Dennis's owner in particular. As well as this, neither cat seemed to be achieving a very steady state with regard to blood glucose levels. Changes to the insulin dose were required almost every day, with pre-insulin blood glucose levels ranging from 3 mmol/L to as much as 22 mmol/L in each cat. Both cats were improving clinically however, and they both had begun eating well and gaining weight. All vomiting, diarrhoea and pyrexia had resolved in both cats.

Dennis had 2 hypoglycaemic episodes during the 3 weeks after being discharged. After reviewing his insulin doses before and after these episodes, it was determined that the owner had administered the wrong dose of insulin, as he had become confused with how and when to change the insulin dose based upon the daily blood glucose reading.

Due to the apparent confusion by Dennis's owner, and the fact that neither cat seemed to be stabilising well, it was decided to keep both cats on a set insulin dose, but continue to record pre-insulin blood glucose levels each day. Barkley was given 2 IU BID and recorded the following results:

Day of Treatment	Pre-Insulin Blood Glucose Level (mmol/L)
1	17.8
2	13.5
3	14.2
4	14.6
5	15.2
6	15.6
7	12.8
8	18.0

As the blood glucose level was sitting slightly higher than it should (<12mmol/L pre-insulin is preferred), the insulin dose was increased to 2.5IU, and another week of readings were taken.

Day of Treatment	Pre-Insulin Blood Glucose Level (mmol/L)
9	7.8
10	3.8
11	7.1
12	13.0
13	7.7
14	2.8

The dose of insulin was kept at 2.5IU BID and Barkley's pre-insulin blood glucose readings continue to remain between 4 and 10 mmol/L. Urine glucose was checked by the owner weekly, and she kept a daily diary of water intake as well as the blood glucose readings. Currently Barkley is clinically well, with a steady insulin dose and good glycaemic control. Although he has not yet achieved remission, this is still a possibility. Remission is likely to occur if the nadir glucose is in the normal range, and the pre-insulin blood glucose is less than 12 mmol/L)[3].

Day 14 showed a blood glucose level which was quite low; however, it was noted by the owner that Barkley did not eat his food as usual on this day, which may explain this particularly low reading. Following this he returned to eating normally the next day.

Dennis's blood glucose readings also stabilised after keeping the Glargine dose steady (full data not recorded on permanent record however). It was adjusted 2-3 times, and although did not reach the same level of stability that Barkley's did, the hypoglycaemic episodes stopped, and clinically he remained well.

Conclusion: Tracking the progress of these cases allowed a good review of the protocol for treating diabetic cats with Glargine insulin in our clinic. A treatment protocol was established to allow consistent management of diabetic cases between the 9 vets employed at the clinic. The protocol now used in our clinic is outlined below:

- After initial stabilisation in hospital (i.e. cat no longer ketonuric or ketoacidotic, eating well and hydration and electrolytes normal), cat should be placed on an initial dose of Glargine of 0.5 IU/kg SC (if blood glucose is >20 mmol/L, or 0.25 IU/kg SC if blood glucose is between 12 – 19 mmol/L[2].
- Owners are taught to take pre-insulin blood glucose readings daily before the morning dose of insulin. The dose of Glargine should remain constant during this time.
- Owners also must keep a diary of water intake, and measure urine glucose at home using urine glucose dipsticks.
- Pre-insulin blood glucose readings to be reviewed by vet each week, and changes to insulin dose to be decided week to week depending on the readings.
- Changes to the dose of Glargine should be made according to the following schedule:

Blood Glucose (mmol/L)	Change to Glargine Dose
> 18	Increase by 0.5 IU (If current dose is < 2IU)
	Increase by 1.0 IU (If current dose is > 2IU)
10 – 18	Increase by 0.5 IU
6 – 10	Leave insulin dose the same
3 – 6	Decrease by 0.5 IU
< 3	Give no insulin AND decrease the next dose by 1.0 IU

- Cats should be placed on a low carbohydrate, high protein diet (preferably Hills Prescription diet m/d® or Royal Canin Feline Diabetic Formula®). Cats should be allowed free access to wet or dry food. Feeding does not need to be coordinated with insulin doses, as the post-prandial increase in blood glucose is very prolonged in cats (18 hours or more)[2].
- Once stable, monitoring of pre-insulin blood glucose, urine glucose and water intake can be reduced to once weekly.

Although initially it was thought best to alter the dose of insulin daily based on pre-insulin blood glucose reading (similar to human diabetics), it was shown that keeping a set dose initially was more beneficial. Constant changes in insulin can be confusing for some owners to make, and neither cat had stable or consistent blood glucose readings while following this protocol.

Daily recording of pre-insulin blood glucose readings by the owners at home allowed us to monitor the progress of both cats in their normal surroundings, rather than the artificially stressful environment of the clinic. Very good glycaemic control was achieved in Barkley, which provides the best possible chance of remission at some time in the future. Reasonable glycaemic control was achieved in Dennis, although it is likely that Dennis's diet is not appropriate which does not allow the same level of control compared to a cat fed a low carbohydrate, high protein diet.

The difference in outcomes for both cats seems greatly affected by the level of client compliance, which was excellent in Barkley's case, but less than ideal in Dennis's case. It is not expected that Dennis will achieve remission, as he was diabetic for some time before receiving treatment, and his glycaemic control is not good enough to achieve this. Barkley has a fair to good chance of achieving remission however, as his glycaemic control is good, he received treatment very promptly and client compliance is excellent.

Post-script: We ended up doing a dental on the cat in question.

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WINNER

Multiple Myeloma in an 18-year-old cat

C&T No. 5258

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'Sleepy' was a long term patient of our practice and at 15.5-years-of-age sustained severe facial fractures in a road traffic accident resulting in bony distortion of the nasal cavity and enucleation of her right eye. Subsequent to this Sleepy developed a chronic nasal discharge (that was usually serous). Her nasal discharge became muco/haemorrhagic 6 weeks prior to presentation and she had a reduced appetite.

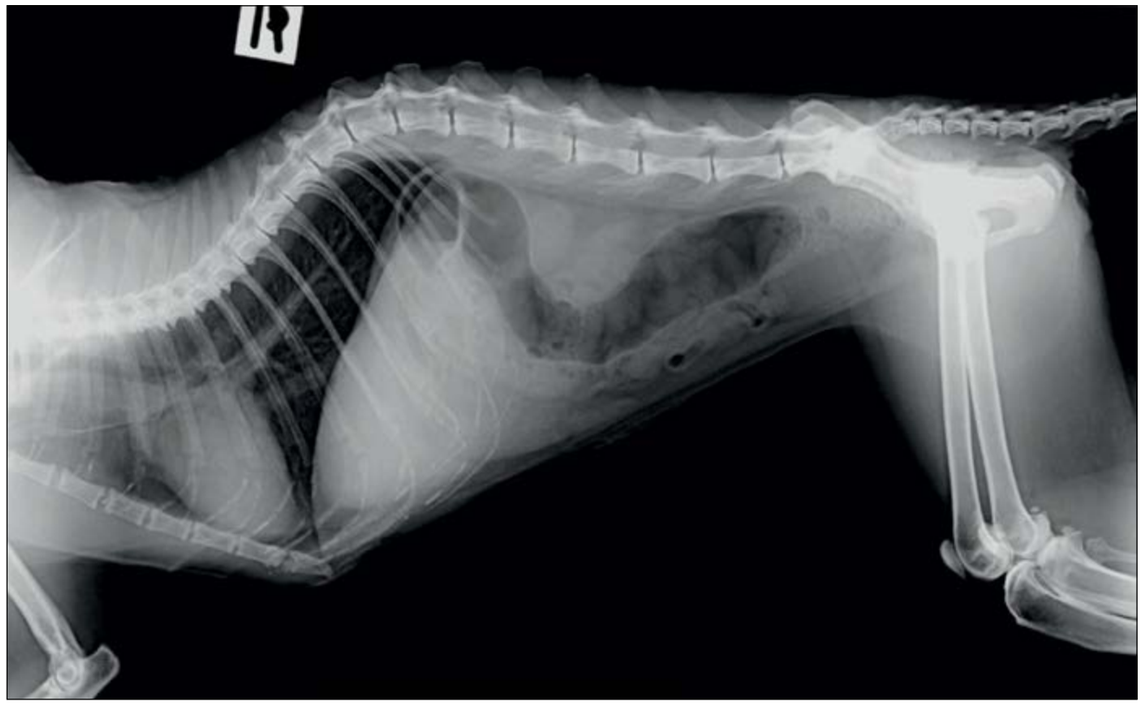
She was treated initially with doxycycline (5mg/kg BID) but there was no improvement in the discharge after 1 week. The antibiotics were changed to clindamycin and a mucolytic was added to the treatment – imaging/ rhinoscopy were also offered at this stage.

At her check up 3 weeks later her owners reported that there had been an improvement in the nasal discharge and initially her appetite had improved. However, she was once again inappetant and had become lethargic and less interactive with her family. She was also favouring her right front leg and seemed unable to place it correctly. She had lost 0.5kgs in 3 weeks and her body and coat condition were poor (2/5). She had pale mucous membranes and was mildly dehydrated and there was an increased inspiratory effort and mild stridor associated with her chronic upper respiratory tract disease. Her heart rate was 192bpm and systolic BP (Doppler) was 130mmHg.

Biochemistry/haematology and urinalysis were performed - **abnormal** results were:

- Amyl 3143 (300-1100) USG **1.032**
- BUN 19 (4-11) Dipstick 4+ protein; 2+ blood; pH 6.0
- CREA 191 (27-186) **No** hypercalcaemia (2.39 mmol/L)
- Na 141 (142-164)
- TP 146 (54-82)
- GLOB 115 (15-57)
- HCT 0.19 (0.28-0.45)
- Lymp 0.8 (1.6-7.0)
- Mono 0.9 (<0.6)
- Retic 0.5% (abs 20 x 109/L)
  - – Marked hyperglobulinaema
  - – Non-regenerative anaemia
  - – Stress leucogram
  - – Renal Insufficiency
  - – Haematuria/proteinuria

Differential diagnosis at this stage with such marked hyperglobulinaemia included:-

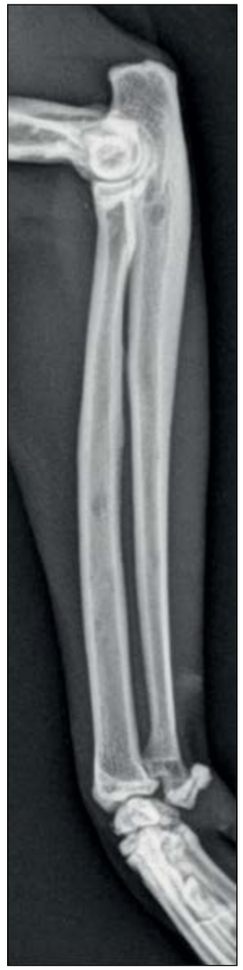


Multiple Myeloma
- Other neoplasia (eg B-cell lymphoma)
- Marked Inflammatory process

FIP
- Chronic infection (reported in dogs - leishmaniasis, ehrlichiosis, chronic pyoderma)
- Amyloidosis
- Other (reported in humans)
- Waldenstroms macroglobulinemia
- MGUS (monoclonal gammopathy of undetermined significance)
- A protein electrophoresis was requested

Protein electrophoresis
- Alpha-1-Globulins 1 g/L
- Alpha-2-Globulins 20 g/L
- Beta Globulin 5 g/L
- **Gamma Globulin 84 g/L**

  A monoclonal gammopathy suggesting multiple myeloma or lymphoid neoplasia
  - Survey thoracic, abdominal and skeletal radiographs were taken
- No mediastinal mass; note thoracic vertebral spondylosis



Left Radius – centrally there is a small area of boney lysis and 2 smaller lesions distally
Left Ulna – similar lesion proximally

Rest of long bones – no obvious lesions
- Based on the clinicopathological findings gathered so far it was considered most likely Sleepy had **multiple myeloma**
- Historically the diagnostic criteria for multiple myeloma are derived from canine/ human studies

Diagnostic criteria (2 of 4) that should be satisfied for a diagnosis

1. ≥20% plasma cells in the bone marrow (or ≥5-10% if atypical plasma cells)
2. Monoclonal or biclonal gammopathy on serum protein electrophoresis
3. Radiographically-evident osteolysis
4. Light chain (Bence-Jones) proteinuria.

Sleepy already showed a **monoclonal gammopathy and radiographically evident osteolysis** – following the above criteria this would have been sufficient to make the diagnosis. However, it had been recommended to me that to achieve a definitive diagnosis of multiple myeloma, diagnostic investigations should specifically include cytological evidence of abnormal plasma cell infiltration – ideally in the bone marrow. However, there is evidence that cats with multiple myeloma commonly have extramedullary involvement (usually in the spleen and liver).

A literature review highlighted the following: **Patel** *et al* **2** in a retrospective study of 16 cats (2005) showed: *Common findings in feline multiple myeloma include atypical plasma cell morphology, hypocholesterolemia, anaemia, bone lesions, and multi-organ involvement.*

Based on the results of this study they advocate modifying the classic diagnostic criteria in cats to include:-
- consideration of plasma cell morphology and
- visceral organ infiltration

They also stated: *Although we did not use extramedullary involvement as a diagnostic criterion, it was a common finding in the cats in this study, suggesting that assessment of extramedullary sites could assist in the diagnostic evaluation of hyperglobulinemic cats,* ***especially when marrow aspirates cannot be obtained.***

Mellor *et al* **4** (2008) in their retrospective analysis of 26 cases:-
- Reported that marked extramedullary involvement at initial clinical presentation is significantly more common in the cat than in human myeloma related disorder (MRD) patients.
- Demonstrated that cats with well differentiated tumors more commonly have extramedullary involvement than human myeloma patients with well-differentiated tumors (90% versus 20%)
- Obtained results that contrasted strongly with the human myeloma model of primary intramedullary neoplastic transformation and suggested that primary extramedullary neoplastic transformation may be more common in feline MRD.

Considering these factors the following diagnostic plan was formulated
- Bone marrow cytology/histopathology was **declined** by the owners as they did not want Sleepy to have a general anaesthetic.
- There was also the possibility of not achieving a diagnostic sample with a single aspirate - the marrow tumours can be very localised/focal and so multiple aspirates from different sites is recommended – particularly if the affected bone sites can be sampled. This would have been much more invasive and again not what the owners wanted.
- I have little experience with BMA and sample handling (in our practice we have more experience with ultrasound guided FNA's and radiography)so this likely influenced my clinical decision making. I used these less invasive diagnostic modalities to try and fulfil the criteria needed for definitive diagnosis of multiple myeloma.
- Sleepy was sedated and abdominal ultrasonography and FNAs of the liver and spleen were performed
  - – 4 x FNA – 2 x spleen and 2 x liver
  - – All samples contained **plasma cells** – more seen in the samples from the spleen than the liver
  - – The plasma cells were reported as well-differentiated

Diagnosis – Multiple myeloma
Criteria satisfied:-
- Monoclonal gammopathy
- Lytic bone lesions ▶

Authors' views are not necessarily those of the CVE
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- Plasma cells in spleen/liver (not a 'classic' criteria but evidence suggested a very common finding in feline multiple myeloma so very supportive of diagnosis)
- Other diagnostic tests available (but not performed) are:-
  - Demonstration of Bence-Jones proteinuria
  - Bone marrow cytology/histopathology would have been ideal (but the owners were unwilling)
  - At the time I did not consider whether a FNA in the region of the lytic bone lesions in the radius may have yielded a diagnostic sample – although it may have been technically challenging given the size and location of the lesion.

Treatment plan

- **Induction therapy** - 0.1mg/kg **melphalan** (alkylating agent) – compounded into 0.25mg capsules – SID for 14 days then every 2nd day until clinical improvement or leukopaenia develops
- Followed by continuous **maintenance therapy** of 0.1mg/kg once weekly
- **Prednisolone** – administered continuously at a daily dose rate of 0.5mg/kg
- Antibiotic coverage (initially doxycyline) whist in the induction phase as patients with multiple myeloma are already severely immune-compromised
- **Monitoring** – CBC and serum globulins (or total protein) weekly

Sleepy had 10 weeks of treatment

After 1 week of melphalan and prednisolone Sleepy's globulin level remained the same (119). She developed leukopaenia in the second week of treatment. The melphalan was stopped (prednisolone and antibiotics (now marbofloxacin) were continued) until her WBC returned to normal which did not occur until Week 6. Her total protein was measured with a refractometer at each weekly visit as a simple in-house test to track any reduction in protein levels (globulins could then be checked separately). At no point in this 6 week period was there any reduction. The melphalan was restarted in week 6 (now administered ONCE weekly). The following week her WBC had dropped again and there was still no reduction in the globulin level. No repeat radiographs were taken of the bony lesions or cytology of spleen and liver (which would have been another way to monitor response to therapy).

At this point it was decided to cease melphalan therapy as it appeared to be ineffective and was causing unacceptable bone marrow suppression. Prednisolone was continued as palliative treatment – treatment certainly had improved her appetite and demeanour.

In **week 9** Sleepy became lethargic and ataxic. There was weakness in her LHL and she tended to drift to the left. A neurological exam showed:-

- Normal withdrawals
- Exaggerated patella reflexes
- Normal foreleg placing
- Hind leg placing – only 2/10 times
- Wheelbarrowing fore was OK; collapsed on hind
- Couldn't hemi-walk
- Suggestive of TL lesion – we certainly questioned whether there was a myeloma lesion in the vertebrae

We offered X-rays but the owner declined. Cats with multiple myeloma have developed neurological signs (Wyatt K 2005). Causes can be multi-factorial and can relate to:-

- Hyperviscosity
- Hypercalcaemia
- Amyloid causing polyneuropathies
- Paraneoplastic immune mediated disease
- Tumour mass causing compression

Sleepy's HCT had dropped to 12 and her anaemia was worsening with blood loss versus chronic disease versus bone marrow infiltration of plasma cells. Globulins were 116. We recommended adding in tramadol and increasing the prednisolone dose. VitB12 injections were started and we considered iron supplementation. However Sleepy continued to deteriorate and she was euthanased shortly after ☹.

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Lethargy and weakness in a 9-year-old dog

C&T No. 5259

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History: A 9-year-old male neutered Staffordshire Bull Terrier was presented with a history of lethargy and weakness of 1 day's duration. The dog had a good appetite but had been noticeably losing weight over the past few weeks. No vomiting was reported. Mild diarrhoea had been observed by the owner over the past few weeks, described as voluminous and malodourous, with no mucus or blood. Oily stains had been noted on the dog's bedding after he arose from rest.

Physical examination findings: Dog was bright and alert on examination. Mucous membranes were pink and capillary refill time was <2 seconds. Chest auscultation was unremarkable, abdomen was palpated as soft and relaxed, HR 200 bpm, pulses normal, temperature 38.4˚C and loose faeces were noted on the rectal thermometer.

Initial Problem List

1. Diarrhoea
2. Weight loss
3. Oily discharge
4. Lethargy and weakness

Initial Assessment: This dog had been presented to the veterinarian primarily because the owner was concerned about the lethargy and weakness. However, these are non-specific clinical signs which may have had many causes. The clinical signs that were considered most specific in this case were diarrhoea and weight loss. The non-specific problems were then considered in relation to the specific clinical signs.

Diarrhoea: This patient exhibited diarrhoea of several weeks' duration, described as voluminous and malodourous, with no mucus or blood. The leading questions were:

- Was the diarrhoea small or large bowel in origin?
- Was the diarrhoea due to primary gastrointestinal or secondary gastrointestinal disease?

The description that the faeces were voluminous suggested that the faecal bulk had increased, which is characteristic of small bowel diarrhoea. The offensive odour of the faeces may have been due to excess gas production, which commonly occurs with small bowel diarrhoea (fermentation of malabsorbed carbohydrates by colonic bacteria). There had been no mucus or blood observed, which made large bowel diarrhoea less likely (but does not rule it out completely).

Small bowel diarrhoea can occur with either primary or secondary gastrointestinal (GIT) disease. There was no vomiting involved; however, this did not definitively rule out primary GIT disease. As mentioned above, diarrhoea was not the primary presenting complaint in this case. This raised the suspicion that a secondary GIT disease was occurring. The clinical picture of weight loss, voluminous and malodourous faeces, and an oily discharge on the dog's bedding made exocrine pancreatic insufficiency (EPI) high on the list of differentials, but is not pathognomonic for EPI. Primary small intestinal disease may also cause similar signs.

Weight Loss: Weight loss accompanied by diarrhoea is often a feature of malassimilation; for example, pancreatic insufficiency (maldigestion) or small bowel disease (malabsorption). If this was in fact the case, then the diagnostic approach was the same as for chronic diarrhoea. However, we couldn't rule out that the weight loss and diarrhoea may have been 2 separate problems.

When dealing with weight loss, it is important to first consider whether the dog had been receiving an adequate intake of food. This dog had always been fed on a balanced adult dog food, a combination of canned and dry. Additionally, the commencement of weight loss did not coincide with receiving less food, or with an onset of anorexia. In fact, the dog had a good appetite. Therefore the dietary intake was adequate for the dog's nutritional needs.

Weight loss in a dog with an adequate intake of nutrients can be due to:

- Maldigestion
- Malabsorption
- Malutilisation
  - Increased nutrient loss – diabetes mellitus, protein-losing enteropathy, protein-losing nephropathy
  - Cardiac disease
  - Elevated metabolism (e.g. hyperthyroidism, chronic fever)
  - Neoplasia
  - Chronic infection/inflammation

This dog had no evidence of cardiac disease and was not pyrexic.

Lethargy and weakness: These are non-specific clinical signs with many causes. This problem was considered after the more specific problems were assessed.

Oily discharge: An oily discharge where the dog had been lying was observed by the owner. Assuming that this discharge was from the rectum, fat malabsorption needed to be considered. Fat malabsorption can be associated with biliary obstruction, lymphangiectasia, severe villus atrophy and exocrine pancreatic insufficiency.

Overall: This patient was exhibiting signs of chronic small bowel diarrhoea, with weight loss, lethargy and weakness. The possible causes of chronic small bowel diarrhoea to be considered in this case included:

- Primary GI disease
  - Diet → lactose intolerance, dietary allergy/intolerance, gluten intolerance
  - Parasites → worms, *Giardia* sp., coccidia
  - Infection → ARE, *Campylobacter/Salmonella*
  - Infiltrative disease
  - Lymphangiectasia
- Secondary GI disease
  - Exocrine pancreatic insufficiency (EPI)
  - Hyperthyroidism – not unless the dog has a thyroid mass
  - Hypoadrenocorticism
  - Liver disease

The weight loss in this dog may have been considered as either secondary to the chronic diarrhoea or a problem on its own. The differentials to consider in this case included:

- maldigestion - e.g. EPI
- malabsorption
- malutilisation
  - increased nutrient loss - diabetes mellitus, PLE
  - Elevated metabolism (e.g. hyperthyroidism)
  - Neoplasia
  - Chronic infection/inflammation

Exocrine pancreatic insufficiency and intestinal parasites were high on the list of differentials and easy to rule out before more invasive tests were required.

Initial Plans: This dog's diarrhoea had persisted for several weeks, therefore a thorough investigation was more appropriate rather than giving symptomatic treatment and observing for a response. Additionally, the fact that the dog was losing weight was cause for concern and an indication for a detailed investigation. The most appropriate first step was to perform a faecal floatation and direct saline smear to investigate for intestinal parasites. These tests are cheap, non-invasive and can be done quickly in-house to rule out the presence of parasites. If there was any doubt, a broad-spectrum anthelmintic could be given regardless of the findings.

Exocrine pancreatic insufficiency was high on the list of differentials, therefore a canine trypsin-like immunoreactivity test (cTLI) needed to be carried out to rule in/out this disease. This required an overnight fast and a single serum sample sent to an external laboratory so the result was not available immediately. ▸

A complete blood count was important to investigate whether there were any infectious/inflammatory pattern to the white cells.

A biochemical analysis allowed measurement of the blood glucose (to rule out diabetes) as well as measure the protein levels in the serum in the case of a protein-losing enteropathy.

Electrolyte levels were important to measure any metabolic abnormalities that may have occurred secondary to the diarrhoea and also to survey for hypoadrenocorticism and liver disease.

If a diagnosis was not reached after the above diagnostic tests were performed, the next step would have been faecal culture and intestinal biopsy.

Results of diagnostic procedures

- Faecal float and smear negative
- CBC, MBA and electrolytes all within normal limits
- Canine TLI → 1.3 ug/L (normal is 5.0 - 35.0)

Revised Assessment: The cTLI result, in combination with the clinical signs of maldigestion (weight loss despite good appetite), voluminous and malodourous faeces, oily discharge presumably from the rectum) was highly diagnostic for severe EPI.

Revised Problem List: Problems identified by history and physical exam:

1. Diarrhoea
2. Weight loss
3. Oily discharge
4. Lethargy and weakness

Problems identified by diagnostic tests:

1. Exocrine pancreatic insufficiency

Therapeutic Plans: Following the diagnosis of EPI, this patient was placed on pancreatic enzyme supplements, with the owner being made aware that the dog needed to be on the supplements for life. The dog's diet was modified to a low fat, low fibre, highly digestible food (hills l/d®). The dog was fed 2-3 times a day and the enzyme supplements given at each meal.

Metronidazole was also administered at 15 mg/kg twice daily for the first 2 weeks of treatment.

At the 3 week follow-up, the owner reported that the dog had noticeably more energy and had started to put on some weight. The patient was then lost to follow-up.

Ocular examination tips

C&T No. 5260

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I find the distant indirect ophthalmoscopy technique most helpful (hand lens + light source). You don't need mydriatic eye drops (as long as the room can be made dark) and you can get a great view of the entire fundus in less than 30 seconds in most cats.

For those of you less familiar with the technique, the advantages of this over direct ophthalmoscopy are:-

- the pupil does not constrict since the light source is far away from the eye (by your eye)
- a large area of the fundus can be seen in one view (if you use a 'pan retinal' lens for example)
- it is very well tolerated by the cat – much more comfortable than direct ophthalmoscopy
- it's a great way of getting a 'global' view of the back of the eye very quickly – even non-ophthalmologists can quickly see whether the eye looks normal or not

Also, see the Free Downloads section on **www.catprofessional.com** for articles on:-

- ocular manifestations of systemic hypertension (lots of labelled pictures) including one showing how to do the technique. There is also a short video demonstrating the procedure.
- doppler guide to measurement of blood pressure in conscious cats

I've been training a UK practice in this technique recently and other top tips are:-

- you can get good hand lens for as little as £30-35 (through Veterinary Specialty Products)
- light source: ideal is a focussed light source e.g. Finhoff transilluminator head on an ophthalmoscope/otoscope body BUT you can also use direct ophthalmoscope to get almost as good a picture; hold the ophthalmoscope by your eye and set the beam on the direct ophthalmoscope to the middle sized circle
- cat: minimal restraint with head in a neutral position or chin slightly tilted up (otherwise you will be grovelling on the floor when you try and see the fundus)
- hold the light source by your eye and the lens just in front of the cat's eye (I rest my middle finger on the cat's head, holding the lens between my thumb and forefinger. The arm of the hand holding the lens should be straight – i.e. you are arm's distance away from the cat. You then tilt the lens and move your head (and hence light) until you see a good view of the fundus
- when learning this technique, start by using the distant light source to get a tapetal reflection (the bright reflection you get like with 'cat's eyes' on a road. Once you have this tapetal reflection, insert your hand lens (close to the cat's eye) and you'll get a great view of the fundus
- occasional problems: cat closes its eyes or pops up its third eyelid obscuring your view. Your helper can assist with the former by gently holding the eyelids apart.

In my experience this technique is a great way of identifying hypertension in cats coming in for vaccinations etc – you are never too short of time to do an eye examination! Also very helpful for diagnosis of infectious diseases like FIP.

WINNER

Dealing with grief: The owner's relationship occurs with the individual, regardless of the species

C&T No. 5261

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'Bun', a neutered male lop-eared rabbit who was 6-years-old when I first met him, was an extraordinary rabbit with great owners. The family consisted of 2 young adult owners, 1 geriatric cat, 2 turtles and Bun. When Bun was around 8-years-old, the family expanded to include 2 dogs, both joining the family as puppies. Bun was predominantly an indoor rabbit, which had free access throughout the house and used a litter tray for toileting. He had regular supervised outside time with his owners.

Bun was a confident and friendly rabbit who had a deep bond with his owners. The house was set up to accommodate his needs. As a young rabbit, Bun had some issues with chewing electrical cables, walls and furniture but Bun's owners researched this behaviour and methods to prevent damage both to Bun and to their house. They provided other chewing options and ensured that his environment was enriched and stimulating. He did still occasionally still chew furniture or other parts of the house; however, his owners loved him deeply and did not ever consider this behaviour to be a problem.

Bun had a complicated medical history with renal and urinary tract disease issues as well as degenerative joint disease as he grew older. Bun was seen regularly by a veterinarian with a special interest in rabbits and my role was effectively as his general practitioner vet, coordinating his care on the recommendation of his specialists. Bun's owners spent largeamount of time and money on his health care. I was also involved in the care of their geriatric cat, a desexed female ex-stray of unknown age which had severe hyperthyroidism, hypertension, renal failure and degenerative joint disease. I saw at least 1 member of this family almost every week for several years. There was no difference in their approach to their care of their rabbit, cat, dog or turtles.

Figure 1. Rabbit (courtesy of Anne Fawcett)

Sadly Bun died at the age of 9 years while in the care of an emergency veterinary centre one weekend. I have counselled many owners before, during and after the death of a pet, and the death of this rabbit was no different. His owners were deeply distressed and went through the same grieving process that all bonded pet owners experience. They experienced guilt arising from not being with him when he died and the usual anger and questions that occur in these situations. They chose to have his body cremated. Bun was such an important part of their lives that his female owner placed a small amount of his ashes in her wedding bouquet 6 months after his death. Several years on, we still often talk about Bun when I see other members of their animal family.

The relationship between Bun and his owners highlights the special bond that can occur with particular individuals. I have known these owners for 5 years and have been with them through the death of 2 rabbits, a cat and a turtle. Although these owners are deeply bonded to all their pets and provide each with outstanding care, each experience has been different due partly to different circumstances surrounding the deaths and partly to the personality of each pet. Shortly after Bun's death, the couple bought 2 young rabbits, 1 of which died unexpectedly at around 1 year of age. Although the owners were greatly upset, they did not have the same deep bond with this rabbit. However, when their geriatric cat was euthanased fairly recently, their pain and grief was palpable as they had a very close bond with their cat, as did I after caring for her for 5 years, and although we all knew that euthanasia was the best option for her, it was still extremely distressing.

Although many owners are reluctant to elect euthanasia for their sick or debilitated animals, the sense of relief afterward is immense for many people. It is incredibly hard to watch your beloved animal in pain and sitting with them during a gentle, painless death can be a strangely positive experience. In this case, the owners felt relief that their cat was no longer suffering. The experience contrasted harshly with their experience with Bun, as they were not with him at the time and his death was not completely expected.

Relationships with pets occur on many levels and sometimes the particular personality of a pet allows for an especially strong bond, regardless of the species of the pet. Although in my professional experience, many owners chose to have a different relationship with pets such as rabbits or guinea pigs than they do with their dogs or cats, there are others for whom the relationship occurs with the individual, regardless of the species.

Granulosa-theca cell tumour in a cat

C&T No. 5262

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'Whisky', a 13-year-old, entire female, domestic medium hair cat presented with a 3 month history of aggression and excessive vocalisation. The behavioural changes appeared to coincide with the owner moving house. Significant abnormalities were not identified on complete haematological (CBC), blood biochemistry (MBA) or total T4 analyses. The aggression and excessive vocalisation were diagnosed as behavioural and Feliway® diffuser was prescribed.

Whisky represented 1 month later with continuing behavioural problems. The behavioural problems were characterised by increased howling at night and aggression towards the other cat. The owner reported that there had been no response to Feliway® and Whisky was commenced on a Clomicalm® trial for 1 month.

Two months after initial presentation Whisky was re-assessed for worsening behavioural problems and a more thorough history was obtained. Whisky was bright and alert, eating and drinking normally and was an inside cat. There was 1 other cat in the household and the owners had owned both cats for over 5 years. Whisky was an entire female cat that had never had kittens and until 2004 never showed signs of being on heat. The behaviours described by her owner were classic signs of being in season. These include: presenting her rear for a scratch, a crouched gate, howling and fighting with the other cat.

Clinical findings included; temperature 38.5˚C, heart rate 120 beats per minute, respiratory rate 40 breaths per minute, mucous membranes pink and moist, capillary refill time < 2 seconds, poor hair coat, slightly thin body condition and being fractious and dangerous to handle. Further examination was limited due to temperament.

Assessment at this stage included behavioural problems with increased intensity despite medication; entire female showing signs of persistent oestrus and possible infertility; a poor hair coat and thin body condition.

The differential diagnosis of aggression include behavioural (e.g. fear, control, dominance); hormonal (e.g. functioning ovarian tumour, adrenal tumours, hyperthyroidism); metabolic (e.g. hyperthyroidism, hepatic encephalopathy); intracranial disease (e.g. neoplasia, toxoplasmosis, cryptococcus, feline ischemic encephalopathy, meningioma) and toxicity (e.g. lead).

The diagnostic plan for Whisky included repeating blood tests to further exclude extra cranial disease and to re-evaluate thyroid status, abdominal imaging to assess reproductive tract, CT scan of the brain to assess for intracranial disease and thoracic radiographs if indicated to assess for metastatic disease.

Clincopathological tests showed no abnormalities: FIV & FeLV negative; CBC – no significant findings; MBA – no significant findings; T4 – 19 nmol/l (10-48); Urinalysis – no significant findings.

Abdominal radiographs showed a soft tissue opacity caudal to the urinary bladder on the lateral view; all other structures were unremarkable. Abdominal ultrasound findings included an enlarged uterus with thickened walls and occasional cystic components. The left uterine horn had a 0.5cm hyperechoic nodule and the left ovary was slightly larger than the right (0.5x0.8cm) with a solitary hypoechoic nodule. The right ovary had 2 hypoechoic nodules. The urinary bladder, medial iliac lymph nodes, kidneys, adrenal glands, spleen, pancreas and liver were unremarkable. Due to the abnormal findings on ultrasound, thoracic radiographs were taken to look for evidence of metastatic disease. Thoracic radiographs were unremarkable.

A CT Scan of the head was also performed to look for multifocal disease, primary intra-cranial disease or malignancy. Pre- and post-contrast CT scans were normal.

Following this extensive work-up, there was no evidence of intra-cranial or metastatic disease and given the abnormal uterus and ovaries detected on abdominal ultrasound, it was considered likely that an ovarian tumour was causing the behavioural signs.

Whisky underwent a routine ventral midline ovariohysterectomy. Surgical exploration revealed a firm right uterine horn with a 13x13x8mm moderately firm, irregular, ovoid, right ovary containing a firm, cream, irregular, ovoid nodule 10x7x4mm. The left uterine horn was similar to the right and the left ovary was 12x9x8mm, moderately firm and irregular to ovoid. The entire uterus and both ovaries were submitted for histopathology.

The right ovarian nodule was diagnosed as a granulosa-theca cell tumour and both right and left uterine horns were diagnosed as cystic endometrial hyperplasia on histopathology.

Follow up over the next 12 months revealed improvement in the behavioural signs. The persistent signs of oestrus resolved and the aggression improved but did not entirely resolve. Whisky's aggressive behaviour towards the owner resolved, however she remained aggressive towards the other cat in the household, although to a lesser degree.

Whisky's body condition and hair coat returned to normal and she continued to do well over the next 12 months with no evidence of metastatic disease. At last follow up Whisky was experiencing no significant problems although may have been starting to suffer from borderline renal insufficiency.

Granulosa-theca cell tumours are a very rare tumour in cats. However, they are the most common primary ovarian neoplasm in the cat[1]. The tumours are derived from ovarian sex-cord stromal tissues. They have the potential to be hormonally active and to secrete steroid hormones[2], producing varying amounts of progesterone, estrogen, testosterone and inhibin. Animals with hormonally productive sex-cord stromal tumours often exhibit abnormal reproductive behaviour that may manifest as persistent anestrus, intermittent or continuous estrus or masculinisation[2].

Feline granulosa cell tumours commonly metastasise[2]. Metastasis can occur in the regional lymph node, via the blood to a variety of organs or rarely by implantation into the peritoneal cavity[2]. Therefore the diagnosis of a granulosa-theca cell tumour in a queen has clinical importance for 2 reasons: it has the potential to synthesise large amounts of hormones and risk of malignancy[3]. No successful chemotherapy protocol has been reported for use in granulosa-theca cell tumours in cats.

Cystic endometrial hyperplasia (CEH) is a disorder of proliferative and degenerative changes in the endometrium associated with aging and hormonal stimulation. Progesterone induces hyperplasia of the surface or glandular epithelium and cystic dilatation of the uterine glands[4]. Endometrial hyperplasia may also be influenced by chronic estrogenic stimulation from recurrent estrous cycles that do not result in pregnancy[4]. Queens with uncomplicated CEH usually have no clinical signs of illness apart from infertility[4]. CEH should be suspected in queens that repeatedly ovulate when bred but do not conceive, provided the tom is known to be fertile. There is no effective treatment for CEH[4].

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Georgia – how sweet is too sweet?

C&T No. 5263

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In many cases the diagnosis of diabetes mellitus in the cat is quite straight forward. The cat may display classic historical and clinical signs. Basic diagnostic tests may reveal persistent and marked elevations in blood glucose and urine may be persistently glucosuric. In this instance, the diagnosis of diabetes mellitus is easily achieved and treatment can be commenced. However, in some cases, the diagnosis is less than clear. The following case study illustrates the steps taken to establish a diagnosis in a patient with equivocal evidence of diabetes mellitus. Stress was a significant confounding factor. Further testing such as fructosamine levels resulted in equivocal results. Water consumption and owner assessment were valuable too in the diagnostic process. Repeated reassessment of history, clinical signs and blood glucose were required to establish that Georgia was indeed a diabetic.

Case History: 'Georgia' was a 13-year-old, female neutered, New Zealand cat of mixed breed. Her mother was a Burmese cross.

October 2009 – A routine geriatric blood screen was run. Blood glucose was run in house on a glucometer as the profile selected does not include blood glucose. Elevated blood glucose of 18.8mmol/L was detected. Results were otherwise within normal limits, see Appendix 1. This test was taken 3 days into a stay in the cattery. The elevation in blood glucose was thought to represent stress. The owner had not noted any changes at home.

December 2009 – Georgia was admitted into the cattery for Christmas boarding. No clinical signs had been noted at home. In an attempt to avoid stress hyperglycaemia, blood glucose was retested immediately on admission. Blood glucose of 12.3mmol/L was detected. Urinanalysis showed 2+ glucosuria. USG was 1.021. Stress from travel to the clinic may have caused this elevation in blood glucose. ***Editor's Note:*** *It is very, very rare for stress hyperglycaemia to 'spill over' and cause glycosuria.*

Blood glucose was repeated the following 2 days and was 13.4mmol/L and 14.3mmol/L respectively. USG was 1.032 and 1.045 respectively, 2+ glucosuria detected on both occasions. Water consumption while in the cattery was at the high end for normal, 80mL/kg/day on wet and dry food.

Georgia was not a highly cooperative patient. Stress was still likely a confounding factor. It was noted that she appeared to have a lower than normal renal threshold for glucosuria. As she appeared to be well, body weight was stable and water consumption was not excessive, the owner elected to monitor her at home.

March 2010 – Georgia was returned to the cattery. The owner has noted some water seeking behaviour: licking water from the shower recess. She may have been drinking more water from her bowl but the owner was unable to quantify this. Blood was drawn and sent for a fructosamine level in an attempt to differentiate stress from diabetes mellitus, see Appendix 2.

Fructosamine was 445umol/L and thus supportive of diabetes mellitus. Georgia was admitted to the hospital with the intention of starting insulin therapy with Glargine. The following blood glucose curve was obtained on day 1:-

8am	9.4mmol/L
12 noon	6.8mmol/L
4pm	8mmol/L

No insulin was give on day 1. The blood glucose curve was repeated on day 2 as follows:-

8am	10.4mmol/L
12 noon	8.2mmol/L
4pm	8.8mmol/L

Insulin therapy was not started. Georgia was discharged. She was to be fed Hill m/d® dry or wet foods at home. The owner agreed to quantify her water intake over the next few weeks.

April 2010 – Georgia represented for weight loss, increased water consumption and urine production and weakness at home. The owner had a detailed water consumption history. Water consumption rose steadily over the 3 week interval, peaking at 175mL/kg/day, with an average of 125mL/kg/day.

On physical examination Georgia showed marked dehydration and had lost 700g over the preceding 3 weeks. Her mucous membranes were pale pink and tacky, CRT was <2sec. Heart rate was 190 bpm, equal to her pulse rate. She was unkempt and was weak and lethargic.

Biochemistry and haematology showed dehydration and blood glucose of 30mmol/L. She was anaemic, PCV 28%. Urinanalysis showed a USG of 1.030, 4+ glucose and a trace of blood and protein. Urine culture showed a staphylococcal infection sensitive to Clavulox. See Appendix 3 for full results.

Rehydration with IV Hartman's solution supplemented with 10mmol of Potassium Chloride was commenced and completed over a 12 hour period. Once rehydrated, she ate well and fluid therapy was discontinued. Clavulox 62.5mg BID PO for 10 days was started for the urinary tract infection.

Insulin therapy was starting using Glargine at 2iu SC BID. Blood glucose curves, sampled at 4 hour intervals were run for 2 days to ensure hypoglycaemia did not result.

One week later a blood glucose curve was repeated. Pre insulin measurement was followed by insulin administration and monitoring every 4 hours. See Appendix 4 for results. The dose was increased to 3iu SC BID. She remained on this dose for ▶

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1 week. A blood glucose curve was repeated, see Appendix 5. This curve showed good control of blood glucose. Her water consumption had returned to normal, her appetite had reduced and she had gained weight. At the time of writing, Georgia remains on 3iu of Glargine SC BID and appears to be doing very well.

Discussion: This case illustrates several difficulties that can be encountered in the diagnosis of diabetes mellitus. Stress can be a confounding factor. In cats, blood glucose can raised significantly in response to stressful stimuli. Fructosamine can be used to differentiate stress from diabetes mellitus in most but not all cases. There is an equivocal range for fructosamine. In this instance, historical and clinical signs in combination with repeated assessment of blood glucose were necessary.

It is interesting to note that Georgia appeared to have a renal threshold for glucosuria that was lower than the usually accepted cut off of blood glucose of 16mmol/L[1]. Georgia displayed glucosuric urine at a blood glucose concentration as low as 12.3mmol/L. It is important to remember that cut off levels and reference ranges do not hold true for all animals. Animals with a normal outside the reference range are not common but should be expected. Trends over time may be helpful in establishing what is normal for an individual. Georgia consistently displayed glucosuria at lower than expected blood glucoses. This consistency in results supports the suspicion that her renal threshold is lower than 16mmol/L.

Stress hyperglycaemia can confound results of blood glucose analysis in cats. Elevations in blood glucose are seen in stressed or chronically ill cats. Well but anxious cats can have blood glucose concentration as high as 16mmol/L and sick, stressed cats can have elevations in excess of 20mmol/L[2]. Elevations of this magnitude would be expected to cause a concurrent glucosuria. The elevations in Georgia's blood glucose prior to March 2010 were within the range seen in healthy stressed cats. The absence of any clinical signs suggestive of diabetes mellitus at home or while in the cattery and her feisty nature suggested the hyperglycaemia was due to stress rather than illness. However, it is important to differentiate between stress and diabetes mellitus. Earlier detection and treatment will increase the changes of diabetic remission to a non insulin dependent state through reversal of glucose toxicity[3].

It is interesting that Georgia had normal or very near normal blood glucose [normal in non diabetic, unstressed cats taken to be 9.5mmol/L or less[1]] when admitted to hospital in March 2010. Her blood glucose curves in April 2010, once insulin therapy had commenced, also showed a lack of stress hyperglycaemia. A conscious effort had been made to minimise stress when handling Georgia. All venipuncture was performed by the same vet and nurse, in the same room of the hospital, following a similar routine. Georgia was housed in a quiet part of the hospital and away from the cattery, where she was used to staying. Georgia may have found it less stressful staying in the hospital or she may have become habituated to handling. This indicates that routine, technique and 'working with the patient' may all have a significant impact on the diagnosis and monitoring of diabetes mellitus in cats, particularly in stressful animals.

Fructosamine may be used to distinguish stress hyperglycaemia from diabetes mellitus. Frustosamines are stable complexes of proteins and carbohydrates. They are produced by a nonenzymatic glycosylation of serum proteins. Albumin is the most common protein. This reaction is non reversible. An elevation in blood glucose will result in an increase in glycosylation of serum proteins that will be persistent for the life of the protein – usually 1-2 weeks. Thus an elevated Fructosamine level indicates persistent hyperglycaemia over the past 1-2 weeks. Increased protein turnover, as is seen with hyperthyroidism, or a hypoprotinaemic state will falsely lower fructosamine.[4]

Georgia had a fructosamine of 445umol/L. This was above the reference interval for the laboratory and interpreted as supportive of diabetes mellitus. However, subsequent blood glucose curves demonstrated a normal or near normal blood glucose throughout the day, NOT supportive of diabetes mellitus. Studies have shown that the upper limit for fructosamine levels may be higher than previously thought. In one study, several cats were identified as non diabetic cats with a fructosamine greater than 400umol/L. This study concluded that the diagnosis of diabetes mellitus be considered equivocal when fructosamine falls between 407 and 450umol/L[5].

Fructosamine formation is dependent on the duration and degree of hyperglycaemia. Persistent hyperglycaemia for several days is required to result in an elevation in fructosamine. Sufficiently marked hyperglycaemia is also required. Mild hyperglycaemia maintained for several days will alter fructosamine but may do so only mildly[6].

Georgia's mild elevation in fructosamine may have been a normal result falling outside the reference range. It may have also been caused by persistent mild hyperglycaemia causing a mild elevation in fructosamine. In this case, fructosamine was not helpful in differentiating diabetes mellitus from potential stress hyperglycaemia seen on previous testing. Georgia may or may not have had diabetes mellitus.

The use of glycosylated haemoglobin was considered. Glycosylated haemoglobin is formed by the non reversible reaction of glucose with haemoglobin. This reaction is dependent on both the concentration of blood glucose and the life span of red blood cells. In the cat, increased glycohaemoglobin indicates elevated blood glucose over the past 6 weeks. A falsely low result can occur with anaemia[4]. This test could have been used to assess if blood glucose had been persistently elevated. Hopefully the results of this test would not have been equivocal and a diagnosis could have been made. In Georgia's case the decision was made not to test for elevated glycohaemoglobin due to financial constraints.

Water consumption is a valuable tool in the diagnosis and management of diabetes mellitus. Water intake has been found to correlate better with blood glucose than fructosamine. Water intake in excess of 100mL/kg/day indicates polydypsia, secondary to the osmotic draw of glucosuric urine and polyuria[7]. The owner documented water consumption over the next 3 weeks. Georgia averaged a water intake of 125mL/kg/day with a maximum of 175mL/kg/day. This degree of polydypsia was not equivocal. Georgia developed other signs consistent with diabetes. She lost weight, 700g over 3 weeks and became polyphargic. The development of historical and clinical signs, in conjunction with clinicopathological changes consistent with diabetes mellitus led to her diagnosis.

High protein, low carbohydrate diets are used to improve glycaemic control in diabetes mellitus in cats. These diets reduce post prandial hyperglycaemia, resulting in decreased post prandial hyperinsulinaemia. They promote weight loss which in turn decreases obesity related insulin resistance. In cats that do not go into diabetic remission, the use of high protein, low carbohydrate diets have been shown to result in decreased insulin requirements[8]. Their use has also been shown to result in an increased rate of diabetic remission to a non insulin dependent state.[9]

When discharged for quantification of water consumption, Georgia's diet was changed from an adult maintenance dry food to Hill m/d dry food. She still received red meat once a day. A high protein low carbohydrate diet was used in an attempt to resolve glucose toxicity, promote beta cell function and control hyperglycaemia if it truly did exist as a consequence of diabetes mellitus. Georgia will continue on this diet throughout her diabetic treatment.

Georgia's case illustrated the difficulty occasionally encountered in diagnosing diabetes mellitus in the cat. It is possible that her early elevations in blood glucose were primarily caused by stress. However, they may have also been caused by mild diabetes. It is unclear why Georgia had 2 normal or near normal blood glucose curves while in hospital, immediately after an equivocal fructosamine result. She may have become less stressed in hospital as the routine became more familiar to her. She may have had normal control of her blood glucose at this time. It is possible that the glucometer used was faulty but this is unlikely as it was in frequent use across a large number of diabetic patients with no other incidences of unexpected results. Thus, in this case, repeated re-evaluation and assessment and a variety of different assessment methods were required to diagnose diabetes mellitus. Early diagnosis and treatment increases the incidence of remission and thus repeated re-evaluation of equivocal patients is warranted.

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APPENDIX 1 - 06/10/09

Red Cell Count	9.11 x 10^{12}/L	(5.00 - 10.00)
Haemoglobin	137 g/L	(80 - 150)
Hct	0.43 L/L	(0.24 - 0.45)
MCV	47 fL	(39 - 55)
MCH	15.0 pg	(13 - 17)
MCHC	321 g/L	(300 - 360)
White Cell Count	6.5 x 10^9/L	(5.5 - 19.5)
Neutrophils	66 % 4.3 x 10^9/L	(2.5 - 12.5)
Band Forms	0 % .0 x 10^9/L 0	(< 0.4)
Lymphocytes	24 % 1.6 x 10^9/L	(1.5 - 7.0)
Monocytes	4 % 0.3 x 10^9/L	(< 0.9)
Eosinophils	6 % 0.4 x 10^9/L	(< 1.6)

FILM MORPHOLOGY: RBC: Normal WBC: Normal PLATELETS: Adequate, Aggregated. Platelet clumps and aggregates present, small ++ .

Sodium	153 mmol/L	(147 - 156)
Potassium	4.4 mmol/L	(3.8 - 4.6)
Chloride	115 mmol/L	(115 - 123)
Bicarbonate	20 mmol/L	(16 - 24)
Na/K	34.8	
Urea	8.6 mmol/L	(5.4 - 10.7)
Creatinine	156 umol/L	(70 - 160)
Calcium	2.63 mmol/L	(1.75 - 2.50)
Phosphate	1.74 mmol/L	(1.29 - 2.26)
Protein	80 g/L	(56 - 80)
Albumin	44 g/L	(22 - 35)
Globulin	36 g/L	(28 - 48)
T. Bilirubin	2 umol/L	(2 - 10)
Alk Phos	23 U/L	(5 - 80)
ALT	36 U/L	(5 - 80)
GGT	1 U/L	(1 - 10)
AST	20 U/L	(10 - 60)
CK	187 U/L	(50 - 300)
Cholesterol	4.6 mmol/L	(1.9 - 3.9)

Urine Specific Gravity 1.036 SERUM INDICES (Clear/+/++/+++)
T4 = 44 [9-48]

APPENDIX 2 - 09/03/10

Fructosamine	475 umol/L	(221-340)

APPRENDIX 3 -15/04/10

Biochemistry – in house IDEXX – abnormal results only recorded

Urea	18.1mmol/L	[5.7-12.9]
Creat	245umol/L	[71-212]
TP	92g/L	[57-89]
BG	30.2mmol/L	[3.94-8.83]
Manual PCV	29%	

Electrolytes all WNL
USG 1.030, 4+ glucose on dipstick, trace protein and whole blood

Hb 98 g/L	(80-140)	WBC 13.5 x10^9/L (6.0-16.0)
RCC 6.9 x1012/L	(5.5-10.0)	Neut 11.1 x10^9/L (3.8-10.1)
Hct 0.28	(0.28-0.45)	Lymp 1.6 x10^9/L (1.6-7.0)
MCV 42 fL	(40-52)	Mono 0.5 x10^9/L (< 0.6)
MCH 14 pg	(13-18)	Eos 0.3 x10^9/L (< 1.4)
MCHC 338 g/L	(310-350)	Baso 0.0 x10^9/L (< 0.2)
Plat 459 x109/L	(200-700)	

Red cells:	Normal.
White cells:	Neutrophilia +.
Platelets:	Normal.

URINE CULTURE
CULTURE Heavy growth (>10^8 org/L)
Org 1: Coagulase negative Staphylococcus

SUSCEPTIBILITY

Ampi/Amoxycillin	S Sulpha/Trimeth S
Cephalexin	S Clavulan/Amox S
Enrofloxacin	S

APPENDIX 4 - 22/04/2010

8am	pre insulin 25mmol/L
12 noon	20mmol/L
4pm	21mmol/L

APPENDIX 5 - 30/04/2010

8am	pre insulin 10.4mmol/L
12 noon	10.2mmol/L
4pm	5mmol/L

Average water consumption over the past week at home = 100mL/day

Decreased appetite at home on m/d and kitten wet food – return to normal volume consumed
Weight gain 150g to 4.25kg.

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WINNER

Feline Progressive Dendritic Cell Histiocytosis

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Abstract: A 14-year-old female spayed domestic shorthair cat presented with a papulonodular dermatosis involving her face. Biopsy led to the diagnosis of feline progressive dendritic cell histiocytosis. Over 3 years this disorder acted in an indolent manner. Lesions would wax and wane. In the latter stages of the disorder the papulonodular change worsened and lesions ulcerated. Life quality of the patient deteriorated and euthanasia was performed. This case report will increase awareness of features of this disorder.

Introduction

Feline progressive dendritic cell histiocytosis (FPDH) presents initially as a papulonodular dermatosis (Day,M. 2008). In advanced cases, spread may extend to lymph nodes and internal organs (Affolter & Moore 2006).

Monocytes, macrophages and dendritic antigen presenting cells are all of the histiocytic cell group. Dendritic cell histiocytes play an important role in processing and presenting antigen (Affolter 2004).

Histiocytic disorders are classified based on their behaviour (Wellman 2007). Broad classification divides these into reactive or neoplastic disorders (Wellman 2007). FPDH is considered a neoplastic disorder (Affolter & Moore 2006). This classification is based on observed failure of FPDH to respond to immunosuppressive treatment (Affolter & Moore 2006).

Cases of FPDH have been reported infrequently. Affolter (2006) is the only in-depth report readily available that discusses FPDH in a group of effected cats.

The aetiology of FPDH is unknown (Affolter & Moore 2006).

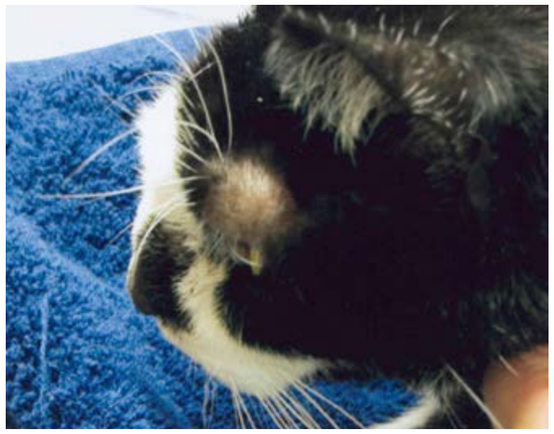
No effective treatment is known and because of the progressive course run by the disease, diagnosis carries a guarded prognosis (Affolter 2004).

This case report describes clinical findings, diagnostic investigations, treatment and final outcome of a mature cat diagnosed as being effected with FPDH.

Case Report

A 14-year-old spayed female domestic shorthair cat was presented with a facial distribution of a papulonodular dermatosis. The cat's owner had been aware of this skin disorder for the past 3 years. During this time the lesions had waxed and waned at irregular intervals. There was no correlation between seasonal change and increased size of the lesions. The owner suspected the periods when facial lesions worsened correlated with times when the cat carried large mouldy leaves in its mouth. The owner would see the cat running up the path with an enormous mouldy leaf in its mouth. Direct contact of the leaf with facial tissue was common. Over the 12 months prior to presentation, the owner noted a worsening of lesions with shorter intervals where lesions reduced in size.

Findings at the initial visit were of prominent papulonodular change on and around the nasal area and above both eyebrows. The largest nodule was 3 x 2 cm. The nodules were solid, raised, non painful, non pruritic and irregular in shape. An impression was gained of generalised lumpy change to effected sites. Refer to Photo 1 and 2 for appearance of the lesions at the time of presentation.



Figures 1 & 2. Appearance of lesions at presentation.

Despite the skin lesions, the cat appeared well with 3/5 Body score. Concurrent problems included elbow arthritis and hyperthyroidism. The latter was being managed effectively with Neomercazole® (5mg Amdipharm).

Results of complete blood count, urinalysis, biochemistry panel and TT4 were within normal limits.

The owner was not greatly concerned about the skin lesions but agreed to deep skin scrapings being taken. Cytology results were consistent with mild inflammation. An aetiology was not identified.

Elocon® lotion (0.1%. Schering – Plough) was prescribed to be applied topically to the larger nodules once daily. A follow up check a month later showed the larger nodules had reduced in size by 60%. A follow up 4 months after this showed further reduction in size of the nodules. The largest nodule at this time measured 1 x 0.4 cm.

The cat was next evaluated 6 months later. The skin condition had markedly deteriorated. Distribution of the papulonodular lesions had not changed but a far greater number of nodules, of larger size, were seen. The cat seemed untroubled by the lesions and she remained well. It was decided to trial a course of Vibravet (50mg tablets. Pfizer Animal Health). This drug was chosen because of its antimicrobial and immune modulating action and its high safety profile. 25mg of Vibravet to be crushed and mixed in food was to be given 12 hourly for an 8 week trial.

Initial apparent response seemed favourable with a report of 40% shrinkage of the nodules. However, by the 8th week there was a dramatic deterioration in appearance of the lesions. A fine needle aspirate was performed. As well as routine cytology, the lab was requested to run special stains to check for presence of mycobacteria, nocardia and actinomyces.

Cytology revealed a non suppurative macrophagic/lymphocytic inflammation and no evidence of cause was found.

A month later the largest of the nodules ulcerated and the cat started to scratch the lesion. Under general anaesthetic an incisional biopsy was taken. Refer to photo 3 for appearance of the ulcerated nodule at the time biopsy was taken.

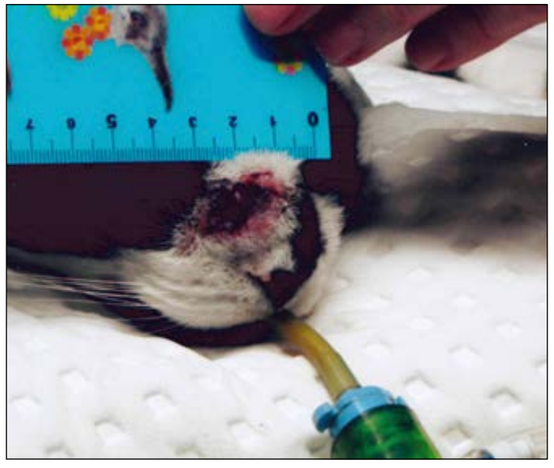


Figure 3. Appearance of the ulcerated nodule at the time biopsy was taken.

Histologic description: Sheets of histiocytic cells (macrophages) infiltrate through the superficial and deep dermis in a diffuse mass, interspersed by low numbers of neutrophils and occasional small clusters of lymphocytes. The histiocytic cells have large, round, oval or indented nuclei with fine chromatin and prominent single nucleoli. There is moderate, two fold anisokaryosis and mitotic figures are present, approximately 3 per 10 high power fields. The cells have abundant foamy eosinophilic cytoplasm. In some areas the histiocytic cells have a more vacuolated appearance to the cytoplasm.

Histologic diagnosis: Atypical histiocytic proliferation most consistent with feline progressive dendritic cell histiocytosis.

The owner was informed that no effective treatment was available.

Within 10 days of the biopsy being taken the owner reported a dramatic improvement in the appearance of the nodules. Remission did not occur but lesions regressed by 80%.

Sadly, a month later there was a rapid deterioration in the skin lesions. The cat was withdrawn and depressed and often tried to scratch the ulcerating nodules.

Euthanasia was performed. Permission to autopsy was not discussed.

Discussion

Knowledge of FPDH as a possible cause of the problem would have influenced management of this patient. It would have prompted incisional biopsy to be done sooner. An earlier diagnosis would have helped the owner to understand what to expect as the disorder progressed. Unnecessary use of antibiotics could have been avoided. Until the histology result was available the author had no knowledge of the existence of FPDH. In retrospect, the protracted period when the lesions appeared indolent and the subsequent rapid deterioration should have placed this disorder high on the differential diagnosis list. The waxing and waning of the papulonodular lesions also should have increased suspicion that FPDH may be present. It would have been ideal to perform an incisional biopsy immediately following the initial non diagnostic cytology test. It is recommended that when inflammation is observed on cytologic examination but no causative organism is found, there is a strong indication to perform a biopsy for both histopathology and microbiologic assessment (Ordeix 2009). An earlier diagnosis would have allowed the owner to be informed that although the lesions had acted benign for several years the long term outlook was poor. Because of the progressive nature of FPDH the prognosis is guarded (Affolter 2004). The owner would also have been aware at an earlier time to monitor the cat for signs of unwellness that may reflect systemic involvement of this histiocytic disorder. As no effective treatment is documented, the final outcome for the patient was not influenced by a delay in making a definitive diagnosis.

The spontaneous waxing and waning nature of FPDH made evaluation of therapeutic response problematic. On 4 occasions the nodular lesions seemed to regress after therapeutic intervention. Nodules reduced in size following use of Elocon, use of Vibravet, performing a fine needle biopsy and performing an incisional biopsy. In the latter case, lesion regression was brief but dramatic .It is hard to explain these findings but lesion regression may have been coincidental. Treatment with corticosteroids, when used early, has been reported to occasionally reduce lesion severity but not lesion progression (Affolter 2004). This may have explained the response seen with Elocon lotion. Vibravet possesses immune modulating and antimicrobial effects. These actions may have decreased the presence of secondary opportunist bacteria or may have directly influenced the immune mediated disorder. The improvement ▶

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following fine needle biopsy, and also when an incisional biopsy was taken, may have occurred due to mechanical insult inducing an alteration in the behaviour of the dendritic cells. No therapy has been shown to influence the progressive course of FPDH (Affolter & Moore 2006)

The early appearance of skin lesions in all reported cases of FPDH is similar. Waxing and waning of the lesions is also common to the reported cases. Lesions may appear as a solitary skin nodule then over time may become numerous and coalesce to form large plaques or remain as non painful large skin nodules (Affolter 2004). Lesions up to 1.5cm have been reported (Affolter 2004). In our patient, the appearance of the lesions and the waxing and waning of these lesions fitted previous descriptions of FPDH. Size of the largest nodules in our patient was twice that previously reported. In common with prior reports, our patient experienced ulceration of the nodules in the late stage of the disorder.

Distribution of lesions in published reports suggests a predilection for the head, neck and lower extremities (Affolter 2004). In Affolter & Moore's study (2006), 21 of 22 cats had leg and foot involvement and 18 of 22 had head involvement. Our patient had lesions restricted only to the facial site.

Further diagnostic investigation by immunophenotyping would have allowed a better understanding of the cell origin of the dendritic cells. A better understanding of the disorder at the time of diagnosis would have prompted this to have been investigated. Immunophenotyping in cats may be limited by the limited availability of useful antibodies or the need for snap frozen fresh tissue (Ordeix 2009).

The cause of FPDH is unknown. In dogs, some cases of histiocytosis are possibly triggered by persistent antigenic stimulation, but no antigen has been identified (Day 1999). T cell driven immune dysregulation is suspected. The canine disorders, however, appear reactive rather than neoplastic and response to immunosuppressive treatment occurs. In cats, no report of resolution on immunosuppressive drugs exist (Affolter & Moore 2006). It is likely the pathogenesis of FPDH differs from canine cutaneous and systemic histiocytosis. It is of interest that the owner of our patient was emphatic that waxing and waning of the disorder correlated with direct facial contact with mouldy leaves. FeLV and FIV status of our patient was not determined. It is therefore not possible to comment on the role these viruses may play in a dysregulated immune response.

The length of time when the lesions act in an indolent manner has previously been reported as up to 3 years (Affolter & Moore 2006). In our patient this indolent period lasted for 3 years.

It would have been useful to perform a necropsy as FPDH is poorly understood and the information gained through a necropsy would help understanding of the disorder. Late spread to involve internal organs could have been checked.

Conclusion

FPDH is a rare disorder. Being aware of its existence is important when considering the differential diagnosis list for papulonodular disease. When history includes the unusual feature of very slow progression of papulonodular lesions with waxing and waning of the lesions, one should rank this disorder high on the differential diagnosis list. Diagnosis early on in the disease allows the owner to be informed of the likely progression of the disorder and to be aware that no effective treatment is available.

Every aspect of FPDH is poorly understood and research into the disorder would be of great benefit. The cause of the disorder, its pathogenesis and possible treatment options are unknown and documenting case reports will increase awareness among clinicians.

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