THE UNIVERSITY OF SYDNEY
Centre for Veterinary Education
Professional Development Leaders

C&T
CONTROL AND THERAPY SERIES

June 2013 ISSUE 271
Australia's Leading Veterinary Forum

Feature Article
Management of an extensive necrotic wound using various hydrocolloids and a mesh graft

CVE are delighted to announce that there will be no increase in Membership Fees for the next financial year.

Wildlife Flashcard Series - Bats


e-book Winner of CVE$500
Congratulations to Sarah Patullo


Surgical treatment of a mammary tumour in a Red Kangaroo


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CONTROL AND THERAPY SERIES

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COVER IMAGE
Image courtesy of Scott Reid. The baby bantam, pictured in Scott's hand, was raised in an incubator.

We'd love some C&Ts on birds…

As I write this editorial, the hot topic in e-mails and the media have been the proposed changes by the Federal Government to the tax deductibility of self education expenses, with a proposed upper limit of $2000 per head per financial year. Like the AVA, the AMA and other professional bodies, the CVE considers this move to be short-sighted and one which will not only affect continuing education providers but also the universities and other tertiary educators. While the treasurer is targeting first class travel and luxury hotel accommodation as conference rorts, why should the majority of people seeking continuing professional development be tarred with the same brush?

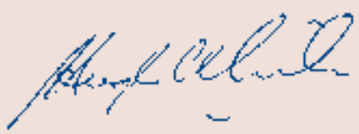
Attendance at many conferences, wet workshops and distance education courses cost well over the $2000 threshold and without doubt many CVE supporters will be hard hit. I urge you to voice your disapproval of this proposed tax change loudly and for as long as it takes to prevent its implementation. Lobby representatives for both major political parties as there is no indication that this will be revoked by the opposition if they form government after the September election.

While I am on this topic, many of you will have recently received a printed booklet from the CVE with our 2013/2014 conference and workshop calendar. We are very proud of this program and hope that having all educational opportunities in one accessible publication will make it easier to select and register for events across a broad range of topics, from short courses to our intensive DE courses. If you have not received a copy, simply download a copy from the Events main page on the CVE website **www.cve.edu.au**

This edition of C&T has another great mix of articles from wildlife to cattle, cats and dogs as well as two Perspective articles. The first is by Linda Fleeman on Monitoring Diabetes in Cats and the second is by Kim Fryer on Management of a Necrotic Wound. This year Linda has already given a one day CVE seminar in Melbourne on Managing Diabetes in Cats and Dogs, but if you missed this she will be presenting this topic again in Brisbane on 13th October. Linda is one of the foremost authorities on this topic and is always ready to share the latest information in this challenging disease.

We have had some very positive feedback on the e-book version of the C&T. if you have not yet tried it, make sure you do so with this edition, as once again there are lots of great video clips and links to previous articles.

Finally, if anyone in your practice has an interest in Cardiorespiratory diseases, register them now to attend the conference and workshops to be held in Melbourne at the end of June. This will be a fantastic program with great presenters and high quality, practical workshops. BOOK NOW!



Hugh White BVSc MVSc MACVSc
DIRECTOR

The economics of welfare in intensive farming

2013 Dr Robert Dixon Animal Welfare Memorial Symposium

Dr Robert Dixon, an inspiration to so many, passed away in February 2011.

The third annual Dr Robert Dixon Animal Welfare Memorial Symposium was held on Monday, 25 March 2013 in the Webster Lecture Theatre at the University of Sydney. This year's topic was 'The economics of welfare in intensive farming'.

Professor Paul McGreevy from the Faculty of Veterinary Science opened the symposium, extending a warm welcome to all including Robert's family—wife Roselyn, sons Justin and Jason and Robert's father Arthur. Chair Dr Chris Degeling then introduced each member of the discussion panel—Ms Kathleen Plowman, Dr Raf Freire, Mr Philip Szepe, Dr Bidda Jones, Mr John Cordina and Mr Grant Hilliard.

The Q&A then got underway to a packed lecture theatre with some thought-provoking and interesting views and responses from both the panel and audience.

View the Symposium proceedings at www.cve.edu.au/animalwelfare

Visit www.vetbookshop.com

Missed the event? You don't need to miss out entirely – buy the event proceedings.

Speaker – Vanessa Barrs BVSc(Hons) MVetClinStud FACVSc (Feline Medicine) GradCertEdStud (Higher Ed)

Head of Small Animal Medicine, Faculty of Veterinary Science, The University of Sydney, Feline Medicine Specialist

Hot topics in feline medicine and practical tips for best-practice management of feline diseases encountered in everyday practice. Vanessa Barrs presented new case-based information on hyperthyroidism in Australian cats to help vets optimise management of hyperthyroid patients with concurrent chronic kidney disease.

For further information on the proceedings visit www.cve.edu.au/node/25842

CVE 2013 SHORT COURSES

From surgery to rehabilitation, the CVE has a wide range of conferences, workshops and online courses.

Not sure about our short courses? Our conferences and **workshops** offer highly intensive learning providing you with a large hit of CPD points in a short time. Our 1-2 day seminars are a practical way to receive a thorough update or refresher. **TimeOnline courses** are delivered wholly online, giving you the flexibility to study when and where you wish and complete your course at your own pace.

All CVE courses are presented by leading experts in their field, so you can confidently choose the CVE to provide you with the quality professional development you seek to become a better practitioner and ensure the continuing success of your practice.

Visit our website (www.cve.edu.au) to find out more about our programs or you can register your interest by emailing us at **cve.events@sydney.edu.au.**

DE 2014 registrations OPEN!

Why pay more? Can't think of ways to spend $500…?

Early DE registrations allow for ease of administration and planning at the CVE's end. To facilitate that and encourage early registrations we are offering you **significant financial incentives** to register and secure your DE place NOW.

Medicine: A Problem Solving Approach 2014 & Sonology 2014 are both FULL.
Procrastinating means not only that you run the risk of missing out on a place in the course of your choice, you also miss out on saving $500.

Register and pay for your DE course **before 30 June 2014** to save $500 **AND** you will be in the draw for a chance to win 1 of our 3 iPads.

Go to the DE webpage: **www.cve.edu.au/distanceeducation**

Read the 2014 CVE brochure to find out more:

www.cve.edu.au/files/2013-4_cve_cpd_brochure_web_version.pdf

EVENTS IN 2013

2 Jun	Hot Topics in Feline Medicine	Canberra
14 Jun	ecoCPD: Practical Radiology for the General Practitioner	Sydney
24-28 Jun	Cardiorespiratory Conference	Melbourne
13-14 Jul	Approaches to Avian & Exotics	Sydney
19-21 Jul	External Fixators	Melbourne
27 Jul	Basic Echocardiography	Townsville
28 Jul	Advanced Echocardiography*	Townsville
3 or 4 Aug	Emergency Workshop	Sydney
17 - 18 Aug	Otitis	Brisbane
31 Aug - 1 Sept	Otitis	Sydney
23-26 Sept	Surgery Conference	Fremantle
5 or 6 Oct	Hip & Stifle Workshop	Brisbane
13 Oct	Diabetes	Brisbane
27 Oct	Looking Down the Microscope	Port Macq.
8 Nov	ecoCPD: Behaviour	Sydney

* Prior learning will be required to attend this workshop.

ONLINE COURSES IN 2013

3 Jun - 30 Jun	TimeOnline: Rabbits & Rodents
15 Jul - 11 Aug	TimeOnline: Wildlife (Students)
22 Jul - 18 Aug	TimeOnline: Pet Fish
5 Aug - 1 Sep	TimeOnline: Respiratory Physiology
12 Aug - 8 Sept	TimeOnline: Otitis
26 Aug - 22 Sep	TimeOnline: Small Animal Behaviour
2 Sept - 29 Sep	TimeOnline: Marine Wildlife (Students)
28 Oct - 24 Nov	TimeOnline: Anaesthetic Complications
4 Nov - 1 Dec	TimeOnline: Avian

Listed dates are subject to change. Refer to **www.cve.edu.au**, for any updates.

Long-term CVE supporter Marshall Thornton comments on the March 2013 e-book

I am writing to commend your fantastic efforts in turning the online C&T into an 'ebook', actually an interactive PDF document. I have saved a PDF copy on the desktop, and it's great to hover over an icon to see a larger photo, or click on an interactive video link to watch Mimi handling the native animals. The video idea will be well worth using. Videos of procedures, of ultrasound real time images, unusual neurological cases – endless information possible! We all have smart phones so we can take the videos.

Going back and forth over the C&T using the contents page is great. So also is the tick round table discussion. I can download the articles the discussions are referring to by clicking a link in the discussion, without having to dig out the (filed away and coffee stained!) hard copies to find the articles. But we still love the hard copies!

I can commend this interactive C&T to other colleagues; once you get the hang of using it it's very easy, and the access to videos is a great feature. Download it on Internet Explorer!

… 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.

Winners

Major Prize

Entitling the recipient to one year's free membership of the CVE
- **Kym Fryer:** Management of an extensive necrotic wound using various hydrocolloids and a mesh graft

CVE Publication Prize Winners

- **Heather Shortridge:** Surprising findings in bovine obstetrics – Schistosoma Reflexus
- **Enoch Bergman:** Systematic BVDV management for beef herds
- **Ruth Gore:** Uterine torsion in an aged, non-gravid cat
- **Aine Seavers:** External markers of internal disease; the 'not-so-humble' nail clip: A nail clip should be mandatory before an MRI or CT!
- **Natalie Burke:** Enamel hypoplasia in an 8-month-old dog: a case study.

Winner of Best Film Clip
Mimi Dona for the clip demonstrating restraint of Bats.

Winner of the CVE$500 draw!

Congratulations to Sarah Patullo, winner of the CVE$500 draw. We hope all readers/members access the complementary e-book version of our June 2013 issue – see Marshall Thornton's praise for the e-book on page 3.

All CVE Members who receive the print copy of the C&T Series are entitled to the e-book version

The e-book is the perfect complement to your print version.

With a roll of your mouse you can bring up previously published C&Ts to save you having to hunt around for back issues. For example, you may be reading the replies to previous C&Ts and wish to refer back to the original article. Simple – just 'rollover' to bring up the article on your screen or download and print it out. With a click of your mouse you can view film clips, or roll-over to enlarge and view X-rays or images.

Look for these symbols

To be emailed the link to the e-book you must provide CVE with your current email address

Contact **cve.membership@sydney.edu.au** or call Jacqui Kennedy and receive your Login and Password details. Then visit **www.cve.edu.au/candtebook** which allows you access to this current issue in e-book format and the 4 prior issues (June, Sept & Dec 2012 & Mar 2013)

Contact
For all enquiries regarding the *Control & Therapy Series*, please contact The Editor, Elisabeth Churchward at **cve.publications@sydney.edu.au** or call (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.'

WINNER OF BEST FILM CLIP

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

Part 4: Wildlife Flashcard Series

Mammals

C&T No. 5302

*This series is the result of collaboration between Mimi Dona & Dr Michael Pyne of Currumbin Wildlife Sanctuary Veterinary Hospital. Non CVE members can access these flashcards and videos at **www.cve.edu.au.***

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) Film clip courtesy of Lincoln Williams, Fotomedia **(www.fotomedia.com.au)**.

Bats

ROLLOVER OR CLICK HERE!

Part 4.1

BATS – MEGA BATS & MICRO BATS

Be aware:-

- Only persons vaccinated against Australian Bat Lyssavirus and wearing the correct personal protective equipment (PPE) should handle any bat species, or be present in the room prior to them being anaesthetised. Notify the appropriate health authority immediately if bitten or scratched.
- They can bite and also scratch using their thumb claws and feet. Their long extendable wings must be restrained as they have a thumb at the end of their Carpals for gripping.
- Micro Bats – Insectivorous bats. Can be mistaken for baby mega bats.
- Mega Bats – flying foxes (or fruit bats).
- Predominantly nocturnal although social during the day.
- Transport bats wrapped in a towel in a well-supported transport carrier – do not allow them to hang in a cage during transportation.
- Flying foxes can get tick paralysis and require treatment.

Figure 1a. Australian bats are known to carry lyssavirus so nobody should attempt to handle them unless fully vaccinated; appropriate PPE should be used at all times.

Figure 1b. Restraining technique for a microbat; place your thumb up under the chin of the bat.

Figure 1c. OR secure the head with the thumb and forefinger scruffing the back of the neck.

Handling

- Always wear PPE - vinyl gloves and then welding gloves. All bats should preferably be anaesthetised prior to handling.
- Mimic their behaviour by hanging them upside down. ▶

- Mega bats are handled by wrapping a towel around them, and gripped around the back of the head/skull to prevent being bitten.
- Provide the orphaned Mega bat with a rolled up face cloth to cling to, folding the wings around the wrapped cloth; once holding on wrap (swaddle) another small fine towel around for support.
- Micro bats can be gently handled in the palm of your hand; secure the head with the thumb and forefinger, scruffing the back of the neck or place the thumb up under the chin of the bat.

Figure 2. When handling bats always restrain their wings as they have a thumb at the end of their carpals for gripping.

Figure 3. Bats must be able to access the wire on the roof of the cage so they can hang upside down; they will get very stressed if they cannot mimic this natural behaviour.

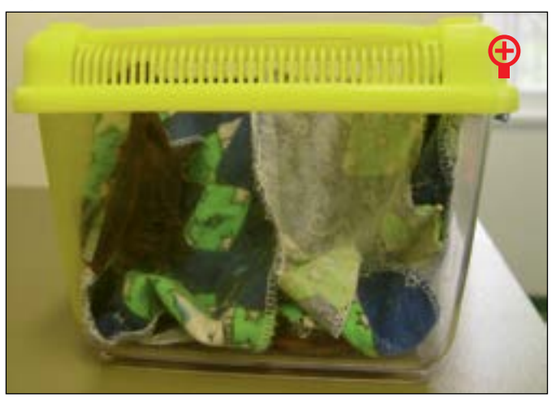


Figure 4. Microbats need to be offered somewhere to climb and hide in; hanging small towels work well.

Housing the sick or injured bat

- Mega bats - Preferred enclosure temperature = 28˚ Celsius for adults and 30˚ Celsius for orphans.
- Adult Mega bats can be placed in a large open top wire carry cage placed on its side lined with soft towels on the bottom and a towel to cover and make it dark. They must be able to access the wire on the roof of the cage so they can hang upside down; they will get very stressed if they cannot mimic this natural behaviour.
- Infants should be wrapped (which will contain them) and then placed on their back in the base of a wire cage should they get out; towels can be used to soften the base and create a gradient so they have their head down. Keep the cage covered and dark. Teats without holes can be provided as dummies. Heat must be given - full term 28˚ - 32˚ Celsius and premature 32˚ Celsius. Ideally they should be housed in a Vetario® or Humidicrib and monitored with an indoor/outdoor thermometer.
- Micro bats - Preferred enclosure temperature = 30˚ Celsius for adults and 35˚ Celsius for orphans.
- Adults can be held in a plastic aquarium or secure enclosure that they can't escape from, and must be provided with a branch to hang onto and small towel to climb and hide in.
- Place orphans in a cotton pouch with tie and provide heat - 35˚ Celsius. Ideally they should be housed in a Vetario® or Humidicrib and monitored with an indoor/outdoor thermometer or alternatively place in a plastic aquarium with a sealed/ventilated lid.

Emergency diet

- Mega bats can be offered a good quality fruit juice (e.g. apple, banana, grapes, rockmelon) or soft fruit.
- Micro bats can be given insects and encouraged with hand feeding if not self-feeding.
- Orphans can be given water and Glucodin initially for the first 2 feeds, then a suitable milk replacer (Divetelact®). This can be given either via a 1mL syringe with catheter tip or for larger orphans use a bottle and appropriate sized teat.

Assessment under anaesthetic

Gaseous

All bats should preferably be anaesthetised prior to handling for assessment. This can be achieved with the assistance of a large towel, wedging/pinning the bat in the cage so you can administer the anaesthetic via the mask.

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.

Anaesthetic Agents

Alfaxan® CD RTU 3 mg/kg – (I/M)

Propofol® 8 – 10 mg/kg – (I/V)

Due to the difficulty with intubation of Micro bats, gaseous anaesthesia is recommended.

Intubation

Cuffed endotracheal tube or catheter tip, some species are too small for intubation. Insert the endotracheal tube with the aid of an anaesthetic spray and tie in with shoelaces.

Recovery

Use a Bair Hugger® or heat mat and room temperature to maintain the patient's core body temperature throughout the procedure, using a cloacal thermometer to monitor. Once off oxygen, return the bat to the enclosure wrapped in a towel and place in a vertical position (upside down). Bats will usually get out of the wrapped towel and climb to a hanging position within 1 - 2 hours. With orphans, Vetario® or Humidicribs are ideal during post-operative recovery.

Figure 5. Infants can be placed on a rolled hand towel; once they are holding on, wrap around another towel for support. Teats without holes can be provided as dummies.

Fluid Therapy

It is important to remember to warm the fluids being administered. Using 0.9% sodium chloride, dose the patient at 5% of its bodyweight. Fluid therapy can be administered I/P or by using standard I/V infusion rates. Syringe pumps are ideal to use in small mammals if available.

Preferred routes for drug administration

- Subcutaneous – administered in loose skin at lateral neck/shoulders.
- Oral – given via a syringe, with smaller species use a cannula attached to a 1mL syringe.
- Intramuscular – pectoral muscle over breastbone is preferred, alternatively quadriceps, triceps muscles.
- Intravenous – cranial edge of wing membrane.

Euthanasia methods

Injection of Sodium Pentobarbitone® can be administered either by intravenous, intracardiac or intraperitoneal routes.

- If administered by intracardiac or intraperitoneal, the bat must be anaesthetised first.

Surgical treatment of a mammary tumour in a Red Kangaroo

C&T No. 5303

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On Wednesday 25/05/2011 I examined an approximately 15-year-old female Red Kangaroo (*Macropus rufus*) part of a Council native fauna exhibit. Staff had noticed dried blood on the animal's tail and found fresh blood on the ground near a fence.

My initial impression was the animal had run into the fence resulting in a nose bleed as there was dried blood on her muzzle. This was supported by black faecal pellets that would result from the swallowing of significant blood from a nose injury. This subsequently proved to be a misdiagnosis.

The animal was a small red kangaroo in good condition (score of 4-5). She behaved normally and no bleeding was observed at a distant examination. The staff were advised to keep her in a small (5 m x 8 m) pen for observation.

Five hours later the staff advised me there was fresh blood around the pouch. I returned fully equipped to anaesthetise and surgically repair any laceration. Although not a pet the animal was approachable and was caught by tailing her and wrapping her in a blanket. On close examination of the pouch I found the blood came from an ulcerated tumour approximately the size of a tennis ball (8 cm diameter). There was a smaller (4 x 2 cm diameter) mass nearby. The blood on her muzzle and the black faecal pellets could have been from cleaning her pouch and ingesting blood. Staff were advised it would be a major procedure to remove the tumours with low hope of success in a dusty yard. I estimated the animal to be approximately 30 Kg in weight and administered 3 mL of Betamox LA1 150 mg/mL. We scheduled her for surgery the next week at our clinic. This would involve an intramuscular sedative and transporting her approximately 1 km to our clinic.

The next week Sydney had near record rainfall so the operation was postponed.

On 06/06/11 the weather was fine. Council staff and my colleague Dr James Phelan helped catch the animal and anaesthetise her.

The anaesthetic was a combination of drugs formulated by Dr Sally Coglan[1] and used to induce anaesthesia for surgical intervention in many macropods. In this case I injected 0.38 mL of Zoletil® 200 mg/mL, 0.54 mL of Domitor® 1mg/mL and 0.45 mL of Acepromazine 2 mg/mL into the anterior thigh muscle.

The animal was suitably sedated for transportation within 12 minutes, with no excitement or running. It was obvious from the foul odour the mass had become more necrotic and extensive since the initial examination. On close examination it was apparent that the only alternative to surgery was euthanasia. The animal was a favourite with the staff and the public and we decided to attempt the surgery.

At the clinic she was induced with 5% Isoflurane via a face mask and maintained on 2% Isoflurane in 100% oxygen for the 1¾ ▶

Authors' views are not necessarily those of the CVE

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hour procedure. Throughout the operation 450 mL of Hartmann's solution was infused IV via a 20 gauge cannula into a tail vein. Heat was provided by hot water bottles and blankets throughout the procedure.

Everting the pouch allowed reasonable access to the mammary masses. The mass was highly vascular and there was minimal subcutaneous or fatty tissue under the thin skin of the abdominal wall of the pouch. There was also significant ulceration and necrosis of the mass surface (Image 1).

Each blood vessel was identified by blunt dissection and transected between two 2/0 Vicryl ligatures similar to performing a splenectomy. Blood loss was minimal.

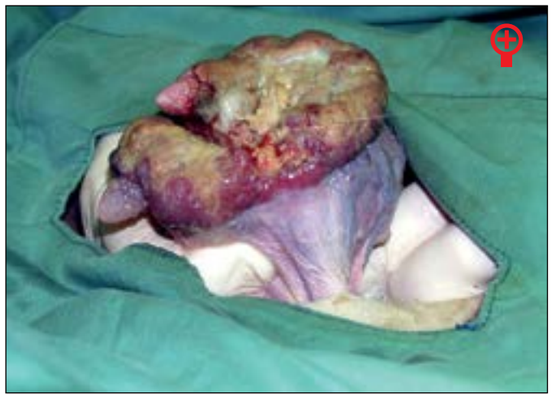

Image 1. Highly vascular pedicle of the neoplastic mass and necrotic surface

Two masses were resected. The larger mass arose from the right and left caudal teats and lateral to this was a smaller firm ovoid lump that was assumed to be a lymph node.

We did not detect enlarged lymph nodes at other sites.

Blood samples and a biopsy section were collected for histological and biochemical evaluation and tumour identification.

A skin flap was made by blunt dissection of the abdominal skin to close the defect from the wide excision. The flap was anchored at several points to the underlying abdominal muscles and to the pubic bone processes (which support the abdominal wall) to eliminate dead space and seroma formation. The skin deficit was closed with simple 2/0 Vicryl® sutures as we did not want to have to recapture the animal to remove non-absorbable sutures. Opsite® barrier spray was sprayed onto the suture line and a pressure bandage applied. The Domitor® was reversed with 0.48 mL Antesedan® 5 mg/mL.

Medical support consisted of 3 mL Betamox® LA IM., 3 mL Baytril® 50 mg/mL SC and 1.5 mL Meloxicam® SC. Equivac® TAT2 tetanus antitoxin 0.25 mL SC was also given as macropods are susceptible to tetanus.

The animal was returned to the exhibit and placed on straw with blankets and hot water bottles in a 3 x 4 metre covered shelter. She remained recumbent for 4 hours before standing and eating. The weather had turned very cold and it is possible she would not have survived without the shelter and warmth.

The next morning 07/06/11 she was standing and eating and she was confined to a small yard for observation (Image 2).

Image 2. Post operation recovery

On 15/06/11 we checked and cleaned the pouch and found the incision was healing well.

Unfortunately on 27/06/11 we were informed by the staff that the animal had been found dead in her pen. The Council staff brought the animal into our clinic and we did a necropsy to determine the cause of death.

Discussion.

The decision to operate on this animal was influenced by several factors. In a domestic animal we would have biopsied the mass and decided to resect it based on the findings. However, although this kangaroo was a captive animal she was not tame. We decided that the capture and anaesthesia of the animal, possibly twice if the biopsy results were favourable, would be more traumatic to the animal than one anaesthetic during which both procedures could be done. Cold and wet weather was also of concern in giving multiple anaesthesias.

Another consideration was the paucity of published information relating to mammary tumours in large macropods. One of the few published articles on the topic reported mammary adenocarcinomas that had metastasised to the lungs in 2 aged *M. rufus*[2]. We reasoned that from the limited data we could not rule out the possibility that the neoplasm was benign.

The pathologist's comments on the serum biochemistry that the animal *'had an inflammatory leukogram but only mild biochemical changes and mild hypoalbuminaemia'* were encouraging as we were concerned that the tumour may have spread to the liver.

Hb 115 g/L	WBC 14.5 x10^9/L
RCC 3.6 x10 ^^{12} /L	Neut 11.0 x10^9/L
Hct 0.36	Lymp 2.8 x10^9/L
MCV 101 fL	Mono 0.7 x10^9/L
MCH 32 pg	Eos 0.0 x10^9/L
MCHC 317 g/L	Baso 0.0 x10^9/L
NRBC 8 /100 WBCs	
Plat 395 x10 ^^{9} /L	
Red cells:	Anisocytosis + Polychromasia +++
White cells:	EDTA changes ++
Occasional reactive lymphocytes	
Platelets:	Normal

NOTE: WBC parameters corrected for presence of NRBC.

Fasting status: Fasting	
Sodium	142 mmol/L
Potassium	7.5 mmol/L
Chloride	103 mmol/L
Bicarbonate	26 mmol/L
Anion Gap	20 mmol/L
Urea	8.2 mmol/L
Creatinine	130 umol/L
Glucose	9.3 mmol/L
Bilirubin	1 umol/L
AST	53 U/L
ALT	19 U/L
Alkaline Phosphatase	52 U/L
Protein	55 g/L
Albumin	22 g/L
Globulin	33 g/L
Albumin/Globulin Ratio	0.7
Calcium	2.79 mmol/L
Phosphate	3.20 mmol/L
Creatine Kinase	607 U/L
Cholesterol	2.4 mmol/L
Triglyceride	0.7 mmol/L
Haemolysis	Nil
Icterus	Nil

We did not receive the histopathology results until over a week after the operation. Unfortunately the report stated '*mammary tubular carcinoma with metastasis to local lymph node'*.

At necropsy the immediate cause of death was acute haemorrhage into the lungs (Image 3).

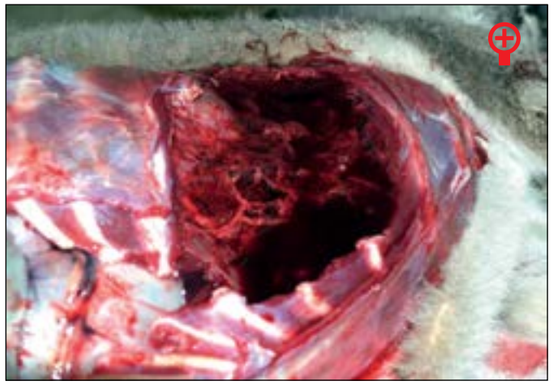

Image 3. Haemorrhage in chest cavity

There were consolidated areas in the lung tissue (Image 4) and also a large abscess (Image 5).

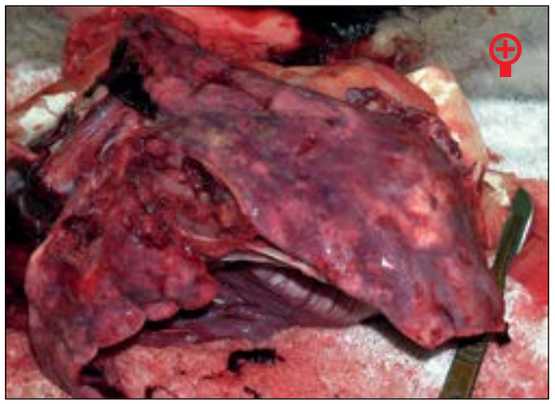

Image 4. Consolidated tumour nodules

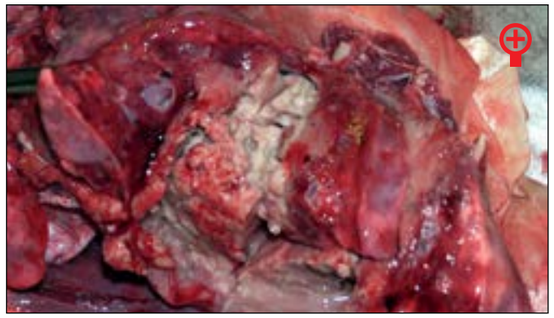

Image 5. Abscess with caseous content

The bacteria were cultured in order to detect if the animal had tuberculosis that could be of concern to the staff involved in her care. The bacteria from the abscess were identified as *Bacteroides sp.*

Drugs and materials used:-

1. Betamox LA 150 mg/mL. Norbrook Labs. Aust. P/L
2. Zoletil 200 mg/mL. Zoletil 100 Virbac (Australia) Pty. Ltd. Powder mixed in 2.5 mL of water for injection to obtain strength of 200 mg /mL.
3. Domitor 1 mg/mL. Medetomidine hydrochloride. Pfizer Australia Pty. Ltd.
4. Acetyl promazine 2 mg/mL. Acepromazine maleate. Delvet Pty. Ltd.
5. Isoflurane. Delvet Pty. Ltd.
6. 2/0 Vicryl. Polygalactin 910 braided suture. Ethicon Inc.
7. Opsite barrier spray. Smith & Nephew.
8. Antesedan 5 mg/mL. Pfizer Aust. Pty. Ltd.
9. Baytrill 50 mg/mL. Bayer Aust. & N.Z.
10. Equivac TAT 1500 IU/mL Tetanus Antitoxin. Pfizer Aust. P/L.

References

1. Dr. Sally Coglan. SCEC Pty.Ltd. Personal Communication.
2. Suedmeyer W and Johnson G. 2007. Survey of neoplasia in Red Kangaroos (*Macropus rufus*) 1992-2002 in a zoological collection. *J Zoo and Wildlife Med*, 38 (2) 231- 239.

Editor's Note

Although implied, the 'take home message' is to take a quick lateral chest radiograph before doing a big surgery in any old animal.

Invited Comment courtesy of:

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This article could be very useful to other practitioners faced with similar cases. The paucity of articles on the surgical treatment of tumours in kangaroos and marsupials in general is consistent with that of most wildlife species. Published articles tend to be based on animals in international zoos where the animals are expensive and difficult to replace so more effort is directed towards their disease investigations and treatment. Red kangaroos are easy to obtain in Australia and generally aged red kangaroos with significant tumours would be euthanised without a post-mortem or histopathological investigation. However, as owner attitudes towards captive non-domesticated animals change in parallel with those of pet owners veterinary practitioners are likely to deal with similar cases. The article provides valuable information about how the case was managed but I agree with the editor's comments about the need for thoracic radiographs prior to major surgery for tumour removal. As the animal was aged with significant widespread disease the anaesthetic regime seems to be safe and reliable.

WINNER

Surprising findings in bovine obstetrics – schistosoma reflexus

C&T No. 5304

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Figure 1. Clipping the patient for a caesarean.

One Saturday afternoon I was called to attend 'Betty' a 7-year-old pet cow, whose owner reported was having trouble with a 'breech' calving. Vaginal palpation revealed a confusing picture; I could not feel a breech calf, but something very odd was going on. I palpated around for a bit, and got an odd pulsating structure in my hand, which in a 'light bulb moment' I realised was the calf's beating heart! I explained the situation to the owners and the potential difficulty of a Caesar. With obvious affection for Betty, they asked me to give it a go.

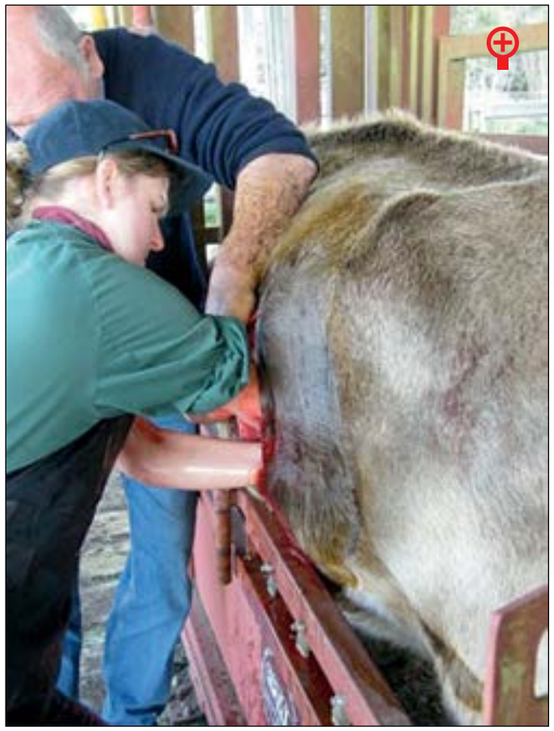

Figure 2. Performing the caesarean.

Initially this proceeded as a normal caesarean, until it was time to get the poor calf out. It was hard to get a foot out. ▶

Figure 3A and next page 3B. Pulling out the deformed calf.

Figure 4. The shistosome calf after delivery by C-section.

Then once we had a foot, it was really hard to pull the calf. The calf was struggling, and I euthanased it via direct cardiac trauma, which sounds awful, but was very effective and more humane than continuing to tug on this deformed calf with it alive. Eventually with a team of pullers we got the calf out.

I was quite worried at this point that I might not be able to close the uterus, which always seems like one of the biggest risks when a calf is difficult to extract by caesarean. I was particularly worried in this case as we had pulled the calf out with one back foot and one front foot, as it was so hard to know which bit was which.

As you can see, the poor calf was a real mess. Fortunately the uterus was not badly torn and I was able to close it (Figures 5&6).

Figure 5.

The rest of the caesarean proceeded more as per normal and I was thrilled to hear several weeks post operatively that Betty was going well, and rearing 2 foster calves. ▶

Figure 6. Closing the surgical incision.

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As an aside, for anyone who does any cattle work, I cannot sing the praises of my pictured waterproof overalls highly enough. I purchased them on a dairy prac, and it seems lots of beef vets don't have them, but they are fabulous.

Another tip from Bob Franklin, one of our senior vets, is to instil salty water (we usually use about 10L) in the abdomen of the cow prior to closing. This can both be used to flush contamination from the abdomen when there is a rotten calf, but also presumably provides the cow with some fluid support (and if anyone has a tip for making it easier to give cattle hypertonic saline in the jugular, I would love to hear it, as it is something I struggle with.)

Figure 7. Instilling salty water in the abdomen of the cow.

WINNER

Systematic BVDV management for beef Herds

C&T No. 5305

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As awareness of BVDV within the beef industry has improved, more producers are becoming amenable to getting vets out on farm to investigate their BVDV status. With guidance, many producers will implement control programs, as long as they are simple, transparent, and perceived to be profitable for them. By enabling producers to collect their own samples for antigen or antibody testing, veterinarians and producers can more cost effectively manage this disease. Swans Veterinary Services has strived to provide other veterinarians the tools to be able to do just that.

Swans pioneered ear notch testing for the identification of Persistently Infected (PI) animals from ear notch tissue in 2006. I had tried unsuccessfully to find another laboratory within Australia willing to offer the service. At the time, blood based antigen testing was the only modality offered. I had three serious issues with blood based testing:

1. It required expertise to acquire the sample
2. It lacked sensitivity in animals under six months of age due to maternal antibody interference.
3. It was expensive

Out of frustration, I imported a purpose built laboratory, primarily for the purpose of serving my own clientele. Soon, other vets became aware of my service and we began offering testing through other veterinary practices (see ***www.swansvet.com/ent.php***).

Allowing producers to harvest their own ear notch samples vastly simplified the process of screening for PIs from populations of animals. I began setting up control programs, centered on appropriate ear notch testing guided by serology. However, once again I began to find my progress obstructed by the need to physically be on farm to take blood samples for antibody testing.

Figure 1. The cow's ear shows a TEGO in action. Photo courtesy of Susan Pike Production.

ITL, an Australian biotech company contacted our laboratory to gauge our interest in using their new Australian designed TEGO device to test for PI animals. The TEGO device makes it very simple for producers to collect blood samples onto specialized absorbent cards for analysis. The disposable TEGO device is applied with a set of Allflex tagging pliers, resulting in a card which can be stored at room temperature and mailed to an appropriate laboratory for analysis. Due to the incredible simplicity, robustness, and accuracy of ear notch tissue for the purpose of detecting PI animals I did not feel they would be useful in that capacity, however I was excited about their potential for antibody testing.

We began validation trials and found that the TEGOs work quite well with the IDEXX BVDV Antibody ELISA, a kit we were already using for bulk milk tank BVDV antibody testing. Finally, I had found a tool that could simplify the serological component of setting up herd level control programs!

Experience has taught me that no farm which harbors a PI animal is safe. The more effective the exposure of young stock to PI animals prior to their first joining, the higher the proportion of immune animals during pregnancy, reducing the proportion of PI animals. Over time, fewer PI calves means reduced exposure of new young stock, potentially eventuating in groups of seronegative animals. Once these animals reach breeding age they will spend the majority of the rest of their lives either pregnant or trying to become pregnant. If they go to the bull without any form of immunity to BVDV, should they meet a PI later in life, a wreck could occur. Wrecks resulting in large numbers of PI's results in high overall immunity, and the cycle repeats itself. Without knowing the immune status of their animals and without understanding the epidemiology of the disease, it is no wonder that many producers that find themselves experiencing BVDV up close and personal often believe it has been brought in by their neighbors, when the reality is usually that they have been endemically infected for years.

The propagation of BVDV is all about timing. Animals without prior immunity, exposed to BVDV during their pregnancy from one to four months produce more PI animals. Managing BVDV effectively is all about timing as well. By providing seronegative animals immunity and removing PI animals before joining, BVDV can be successfully managed.

Antibody screening is the most sensitive way to screen for the presence of PI animals without directly ear notch testing all of the animals on the property. Quite simply, if an unvaccinated animal has immunity to BVDV then it is likely from direct exposure to a PI. If a PI has existed for a reasonable amount of time within a management group, most or all of the animals will have seroconverted to the virus.

Many times we as veterinarians may begin our BVDV investigation as part of an abortion screen. AGID testing requires a reasonable amount of serum, more than the TEGO devices harvest. The AGID is a very useful tool to prove or disprove recent exposure to BVDV, excellent for abortion investigations. The antibody ELISA on the other hand is a less expensive tool to measure the lack of, or presence of, BVDV antibodies without estimating the recentness of infection.

Often I am called upon to work up a property's BVDV status from scratch. In other instances, I am following up an investigation that had either previously detected or implicated BVDV. The TEGO devices allow me to quickly set up a risk profile for the entire herd. As veterinarians, we can help our producers to invest their resources most cost effectively by measuring the immune status of individual mobs on each individual property.

1. Mobs with a high level of immunity will not benefit from vaccination.
2. Stable mobs with low levels of immunity do not contain PI animals.

I ask my producers to collect blood using the TEGO devices from 5% or a minimum of 6 animals from each stable management group on the property. When appropriate, mobs without immunity are advised to enrol in a Pestigard vaccination program. Mature mobs with high levels of immunity may contain a PI, or the immunity could be from historic exposure. Rather than ear notch testing the adult animals from these highly immune mobs, I monitor their calves for the presence of PI animals. At calf marking, any woody calves are ear notch tested and visually marked. If the calf is a PI, their mother may be a PI as well. The calves are observed to ascertain the identity of their dam and she is eventually also ear notch tested. A new and handy tool for testing calves crush side is the IDEXX BVDV SNAP test. At the same cost, veterinarians can source the new

Figure 2. The cow and calf represent: 'A PI cow discovered by identifying its calf as a PI first.'

SNAP tests either from Swans Veterinary Services or IDEXX directly. Any PI's found at the end of each draft could be held back to identify their dam, allowing her to be tested immediately. Any adult PI animals would then be sold direct to slaughter.

After profiling the risk level of each of the mature mobs, my systematic control program focuses on annually ensuring that each new group of replacement heifers is both immune and PI free prior to mating. Screening a proportion of the unvaccinated replacement heifer mob well in advance of mating allows us to do just that. At the time of testing, the heifer replacement mob needs to be stable, without any recent additions, and well past maternal antibody interference. As a rule of thumb, they need to be at least 8 months old and have been in stable contact for at least 2 months. If they are found to have a low level of immunity, they should be enrolled in a vaccination program. If they are highly immune, the expense of vaccination can be forgone, and the heifers individually ear notch tested. Any PI animals which may exist within the mob can then be found and sold to slaughter before she begins to waste away.

Occasionally, especially in more extensive situations, or in large groups of replacement females, the seroprevalence of the group may still be maturing. If only a proportion of the animals are seropositive, there are three general scenarios:

1. Incomplete ongoing exposure (PI still present)
2. Historic PI exposure (PI present prior to heifer selection process)
3. The seronegative animals are in fact PI animals themselves

Scenario 3 can be quickly ruled in or out by performing an antigen capture ELISA on the same blood sample that was used to measure for antibody levels. If scenario 3 has been removed, scenarios 1 and 2 can be discerned by performing follow up serology on the replacement heifer group one month after the first screening. Producers should be directed to collect samples from the previously seronegative animals and an additional 5% randomly selected heifers. If all or a proportion of the previously seronegative animals have since seroconverted, there is ▶

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ongoing BVDV exposure, usually indicating the presence of a PI. The mob of heifers should therefore be ear notch tested immediately. Conversely, if the seronegative animals have been confirmed not to be PI and remain seronegative after the follow up serology, then it is likely that the partial seroconversion is from past exposure. Using the overall seroconversion rate from the first and second screenings, producers, guided by their veterinarian, can choose to vaccinate or not depending upon the estimated seroprevalence and their appetite for risk. Personally, I advise vaccinating replacement mobs with seroprevalences below 80%.

By following this process, we are ensuring that all breeding animals possess either natural immunity to BVDV or have been vaccinated, and that each new replacement heifer mob is both immune and PI free prior to mating. PI production will be greatly reduced, adult PIs will either die or be culled, and with time the entire property will likely become BVDV free. Maintaining freedom is achievable with simple biosecurity and can be further protected by maintaining a vaccination program. By continuing to annually screen their heifers as described above, we can monitor the freedom of the property, providing proof to the producer that their money is being appropriately invested in BVDV management. Should a biosecurity breech result in the production of one or more PI calves, they will be prevented from being retained as heifer replacements. The property will soon settle back down to a BVDV free status.

Lastly, and obviously, all introduced animals should be ear notch tested and preferably quarantined for 30 days prior to being introduced to any other management groups. The unborn progeny of any introduced animals should also be considered as introductions, and should be ear notch tested at birth. Direct exposure to a PI animal is the main way that BVDV is propagated, biosecurity to manage BVDV does not have to be complicated.

My goal has been to develop a range of tools that allow veterinarians to cost effectively manage BVDV, providing tools that producers can use to collect their own samples is the key to making BVDV management a profitable exercise for both producers and their veterinarians. Swans Veterinary Services provides ear notch testing, BVDV antibody ELISA testing, Bulk Milk Tank testing, specialized testing equipment, and free BVDV consultancy to veterinarians. My goal is to eradicate BVDV, one farm at a time!

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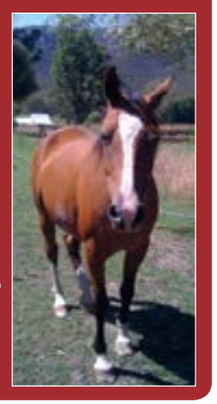
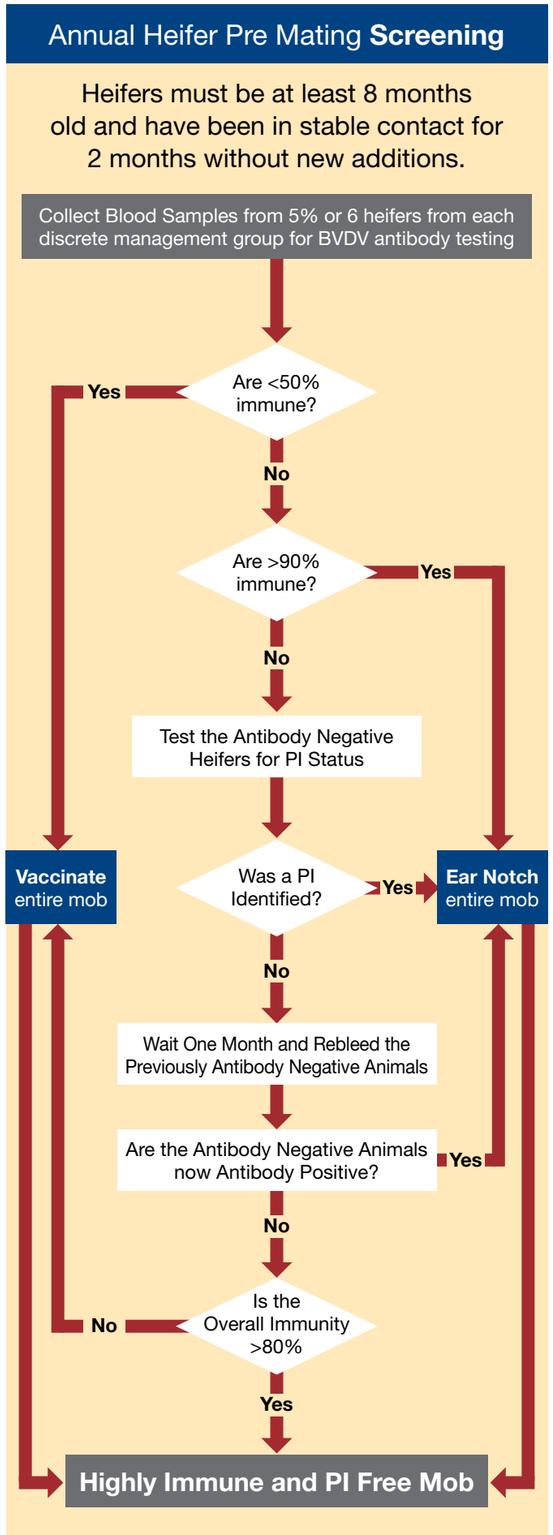


Photo courtesy of Scott Reid.



WINNER

Uterine torsion in an aged, non-gravid cat

C&T No. 5306

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Introduction

Uterine torsion is an uncommon condition in the intact female cat and is most commonly associated with pregnancy. The following case study reports on a case of uterine torsion in a non-gravid queen which presented with anorexia and abdominal distension, and had a regenerative anaemia with hyponatraemia and hypochloraemia on laboratory analysis.

Case Report

An 11-year-old entire female Domestic Shorthair cat weighing 3.9kg was presented after hours for anorexia of 1 week's duration and abdominal distension which had only been noticed the previous day but had become more pronounced. The cat was still drinking normal amounts and there was no vomiting or diarrhoea seen. A change in behaviour had been noticed over the preceding 3 weeks with the cat following her owner around more and not wanting to be left alone. The cat lived indoors and had no apparent access to outside.

Figure 1. Photograph of the cat showing abdominal distension

On examination, the cat was quiet but alert and responsive. The oral mucous membranes were pale, heart rate 200 beats per minute with a grade 1 murmur, respiratory rate 50 breaths per minute, and a rectal temperature of 38°C. The abdomen was distended (Fig 1) with a large non-painful fluid-filled tubular structure detected on palpation. There was no vaginal discharge present. Differential diagnoses at this time included pyometra, pregnancy, intussusception, intestinal obstruction, intestinal volvulus, splenic torsion, uterine torsion, and abdominal organ enlargement or neoplasia. The cat was admitted for further diagnostic investigation.

Results of haematological and serum biochemistry tests (Fig 2) showed a moderate normochromic, normocytic regenerative anaemia, moderate hyperglycaemia, hyponatraemia and hypochloraemia. Ultrasound examination confirmed the presence of a fluid filled hollow organ consistent with the uterus, and no evidence of foetal development (Fig 3). An exploratory laparotomy was scheduled following initial stabilisation.

The cat was placed on intravenous crystalloid fluids (0.9% sodium chloride solution) at a rate of 3mL/kg/hour and administered 43.75mg amoxicillin/clavulanic acid (Clavulox™ injectable) intramuscularly pre-operatively. Premedication with 0.6mg methadone/0.06mg atropine was administered subcutaneously, followed by anaesthetic induction with 10mg alfaxalone (Alfaxan™) intravenously. An endotracheal tube was placed and the anaesthetic maintained with inhalational isoflurane and oxygen via a non-rebreathing circuit. The intravenous fluid rate was increased for the duration of the anaesthetic and surgery to 10mL/kg/hour. ▶

	Result	Normal range
RBC	3.01	5-10x10^{12}/L
HCT	16.5	30-45%
HGB	4.8	9-15.1g/dL
MCV	54.9	41-58fL
MCH	16.1	12-20pg
MCHC	29.3	29-37.5g/dL
%Retic (corrected retic 1.78%)	4	%
Retic	121.7	K/uL
WBC	10.33	5.5-19.5x10^{9}/L
Neu	7.84	2.5-12.5x10^{9}/L
Lym	1.17	0.4-6.8x10^{9}/L
Mono	1.14	0.15-1.7x10^{9}/L
Eos	0.15	0.1-0.79x10^{9}/L
Baso	0.02	0-0.1x10^{9}/L
Plt	209	175-600K/µL
RBC morphology	anisocytosis 2+	
	polychromasia 1+	
Gluc	13.76	4.11-8.84mmol/L
Urea	8.3	5.7-12.9mmolL
Crea	79	71-212µmol/L
Phos	1.53	1-2.42mmol/L
Ca	1.95	1.95-2.83mmol/L
TP	59	57-89g/L
Alb	29	22-40g/L
Glob	30	28-51g/L
ALT	29	12-130U/L
ALP	<10	14-111U/L
GGT	0	0-1U/L
Tbili	2	0-15µmol/L
Chol	2.1	1.68-5.81mmol/L
Na	138	150-165mmol/L
K	4	3.5-5.8mmol/L
Na/K	35	
Cl	107	112-129mmol/L

Figure 2. Haematology and serum biochemistry results.

The cat was placed in dorsal recumbency and a ventral midline laparotomy was performed. Upon entering the abdomen, a distended uterus was visible with a 1260° torsion of the left uterine horn along its longitudinal axis (Fig 4, 5). This section of uterus was grossly congested and engorged with fluid and was still receiving some arterial blood flow into the area despite occlusion of venous drainage. The right uterine horn was also distended with fluid but to a lesser extent and still had a normal pink colouration. Ovariohysterectomy was performed with double ligation of all pedicles with 3/0 polydioxanone suture (Monodox™) – the torsion was not corrected prior to removal of the uterus. The abdomen was lavaged with approximately 500mL of warmed 0.9% sodium chloride solution and closed routinely. Analgesia (1mg meloxicam injection subcutaneously) and Vitamin B complex injection (0.5mL intramuscularly) were administered and maintenance fluids (2mL/kg/hour 0.9% sodium chloride solution) continued for 24 hours post-operatively.

The excised uterus and ovaries weighed a total of 820g. The left uterine horn was filled with thick bloody fluid. The right uterine horn contained light brown, slightly turbid fluid which seemed to be comprised of degenerate material and low numbers of normal appearing neutrophils – no obvious bacteria were visible on in-clinic microscopic examination but the presence of pyometra could not be excluded. Unfortunately, further cytological evaluation by a veterinary pathologist to characterise the fluid further and bacterial culture were declined by the cat's owner.

The cat made a rapid recovery from anaesthesia and started to eat 6 hours after surgery. Continued improvement occurred over the subsequent 24 hours and the cat was discharged. Antibiotic coverage with long-acting cefovecin (26mg subcutaneously; Convenia™ injection) was provided as the cat's owner was quite elderly and unable to administer any oral medication.

Discussion

Uterine torsion is an uncommon condition in the queen and may involve one or both uterine horns twisting along the long axis or rotation of the entire uterine body[4]. Although the exact aetiology is unknown, it is thought to occur when there is an increase in the size or weight of the uterus, alterations in uterine muscle tone, foetal movement, or with laxity or weakness of the suspensory ligaments[1,3,9]. Unlike dogs where torsion may be associated with pregnancy, pyometra or with varying stages of the oestrus cycle[4], torsion in cats is most commonly seen with a gravid uterus or in the periparturient period and until recently all recorded cases in the literature were associated with pregnancy[1,3,5,6,8,10,12]. This case was unusual in that the cat was not pregnant and had never produced a litter. To the author's knowledge there have been only 2 other reported cases of uterine torsion in a non-gravid uterus[2,9]. In both cases there was fluid accumulation in the unaffected uterine horn unrelated to the torsion – in one case mucometra and the other pyometra.

Pyometra is much less common in the queen than the bitch. This is thought to be due to the lower exposure of the uterus to progesterone[7]. In the bitch there is spontaneous ovulation and the subsequent sustained elevation in progesterone levels, even without pregnancy, predisposes to cystic endometrial hyperplasia (CEH) and pyometra. As the queen is an induced ovulator, if mating does not occur there is no ovulation and hence no rise in progesterone. Even if mating and ovulation occurs, the subsequent rise in progesterone occurs for a much shorter period of time (45 days vs more than 60 days in the bitch). Spontaneous ovulation has been known to occur in queens in response to various pheromonal stimuli which could account for CEH and pyometra occurring in queens which have had no direct contact with males[9]. Fluid accumulation due to CEH/pyometra within the uterus of the cat would potentially increase the chances of torsion occurring[9] but as there is a much lower incidence of pyometra in the cat this may account for the lower incidence of uterine torsion seen secondary to pyometra.

Mucometra results from a build up of non-inflammatory fluid by blockage of the vulva, vagina, cervix or uterus. This may be a result of inflammation and scarring, tumour or congenital abnormality. The accumulated volume of fluid can be quite large (up to 500mL) in some cases[7] and this would also increase the potential for uterine torsion.

Clinical signs associated with uterine torsion range from clinically normal to a collapsed state due to shock[4,7]. Commonly there is anorexia, vomiting, pale mucous membranes, dystocia, abdominal pain and distension, and vaginal discharge. The affected cats have ranged in age from 1 to 10 years and the stage of pregnancy from 4 weeks gestation to the peri-parturient period[3,10,12]. The clinical course in the pregnant queen tends to be relatively short from 2 hours to 3 days[4], though there are exceptions to this[8]. The case with pyometra and subsequent uterine torsion also had a short clinical course (2 days)[9] whereas the case with mucometra had a longer clinical course (1 week) and less cardiovascular compromise[2]. This may be as a result of the presence of a more severe metabolic derangement (pregnancy, dead foetuses, infection) in association with the torsion causing a more rapid progression of clinical signs[6,9], but there are so few cases with which to compare there may be other factors involved including degree of torsion or how rapidly the rotation occurs. In the cases reported, the degree of rotation has varied from 180° to 900°[2,3]. In this case there was a 1260° rotation which is greater than what has previously been reported, but despite this the cat had shown only mild clinical signs for a week and was still relatively stable and bright at the time of presentation. Although the presence of infection could not be confirmed or ruled out in this case – if present it was most likely only low grade based on the clinical presentation and the appearance of the fluid obtained from the unaffected uterine horn.

The presence of uterine torsion may be suspected based on the results of physical examination, abdominal radiographs and abdominal ultrasound but final diagnosis is often not made until the time of exploratory surgery[4,9]. Ovariohysterectomy without correction of the torsion is the recommended treatment. De-rotation of the uterus can release endotoxins and inflammatory mediators into the circulation which can have serious systemic consequences and negatively affect the outcome of the surgery[6]. Most cases of uterine torsion can be treated successfully if there is prompt surgical intervention and appropriate pre- and post-operative supportive treatment[2,3,6].

The presence of a moderate normochromic, normocytic regenerative anaemia most likely was the result of gradual sequestration of blood with the lumen of the uterus following occlusion of the venous outflow[2,3,6] – the 'third spacing effect' whereby there is a fluid shift to an area of the body where fluid doesn't normally accumulate in large amounts or becomes physiologically non-functional – i.e. outside of the intravascular and extravascular spaces. The loss of blood to the 'third space' (in this case the uterus) creates the clinical appearance of a blood-loss anaemia (usually associated with haemorrhage or haemolysis) without the cause being immediately identifiable. As the normal clinical findings associated with haemorrhage external wounds, haematuria, melaena, haematochezia, haematemesis, epistaxis, haematomas, hypoproteinemia) and haemolysis (icterus, haemoglobinuria, haemoglobinaemia, autoagglutination) are not ▶

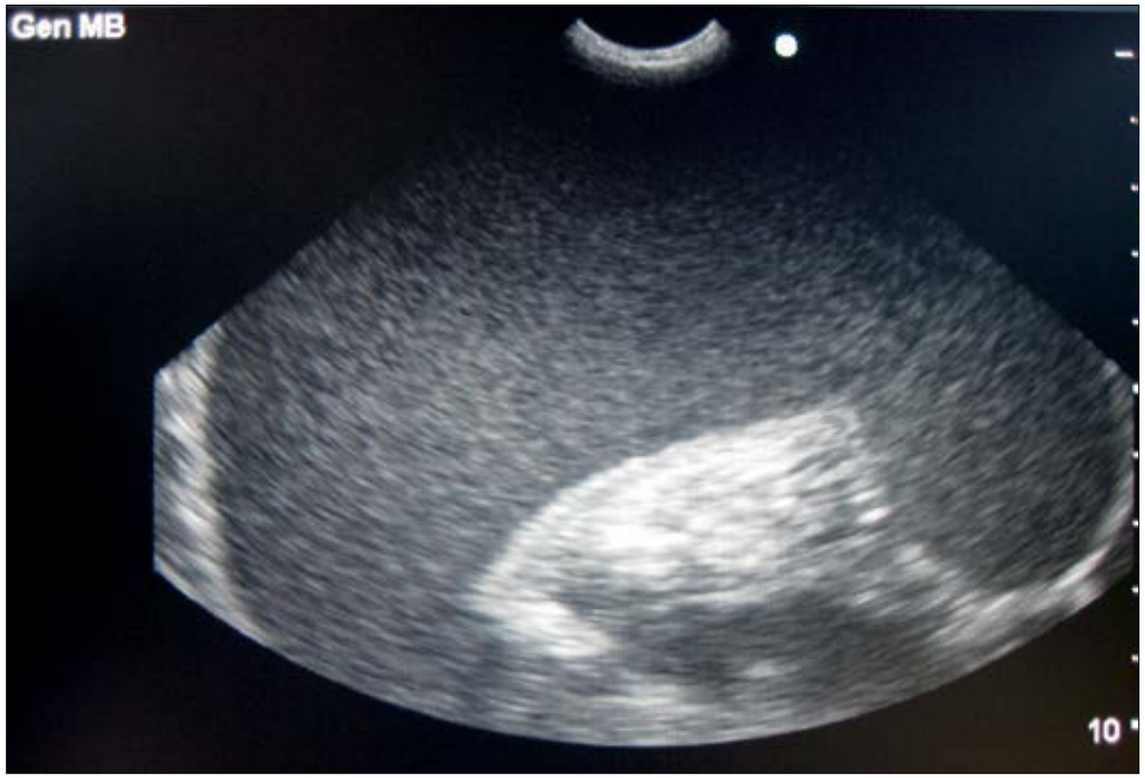

Figure 3. Ultrasound image showing fluid distension of the uterus

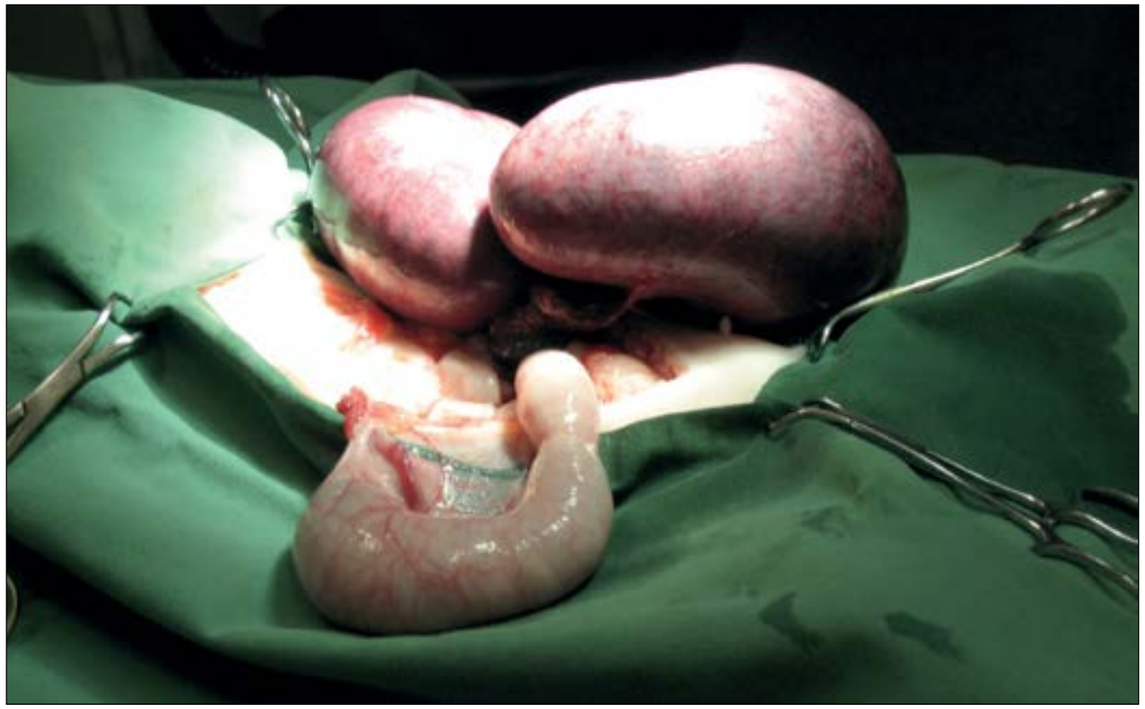

Figure 4. Intraoperative photograph showing torsion, distension and congestion of left uterine horn and fluid distension of right uterine horn

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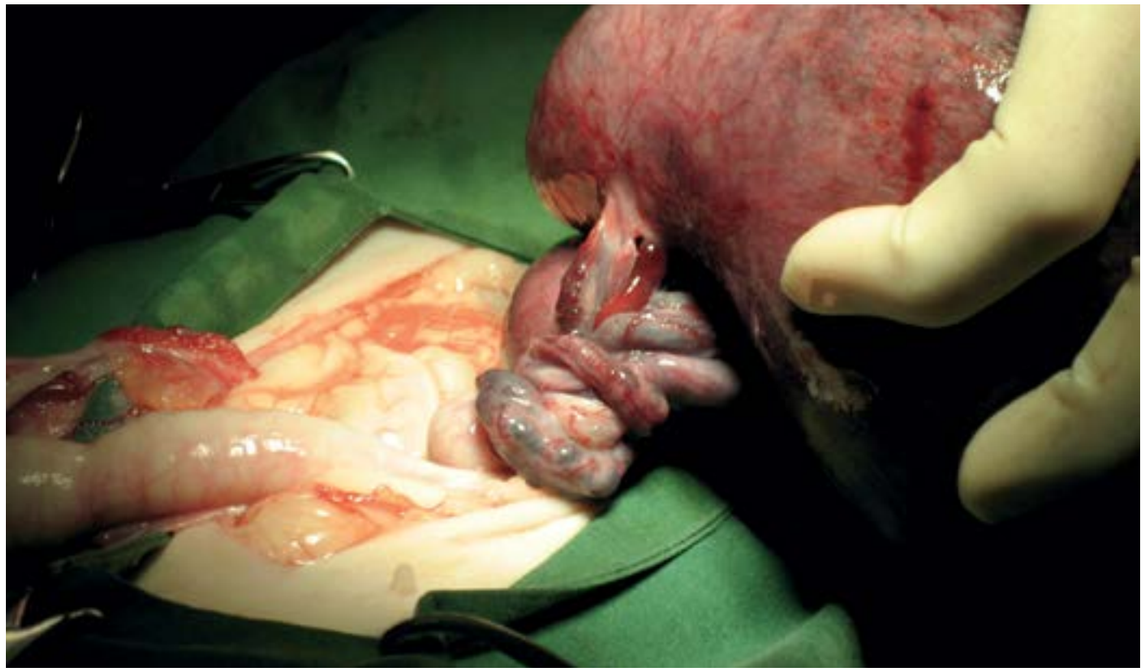

Figure 5. Intraoperative photograph of the torsion

seen – the initial identification and localisation of the condition can be difficult. The presence of an early regenerative response suggests that the sequestration had been present for longer than 3 to 5 days allowing time for a bone marrow response to occur[11]. The lack of signs of shock at the time of presentation was also consistent with a more chronic disease course with a gradual reduction in the haematocrit rather than acute blood 'loss' from the circulation. The elevated heart rate, respiratory rate, and the grade 1 murmur were considered to be secondary to the anaemia. In the absence of major renal and gastrointestinal abnormalities, the hyponatraemia and hypochloraemia were most likely occurring secondary to 'third space' loss[6,9,11] and there was a moderate stress hyperglycaemia.

Conclusion

Blood loss into body cavities and the lumen of hollow organs – the 'third space' – should always be part of the differential diagnoses when the cause of the blood loss is not readily apparent and may be more insidious in nature. Uterine torsion – although very uncommon, especially in the non-pregnant queen – should be considered as a differential diagnosis in any intact female cat presented for abdominal distension and may require urgent surgical intervention, especially if signs of shock are present.

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Transient lymphoma regression in a cat

C&T No. 5307

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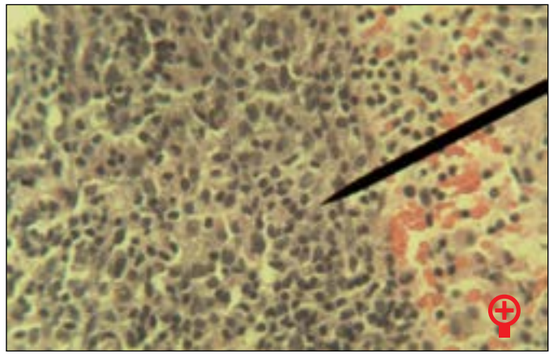
Introduction

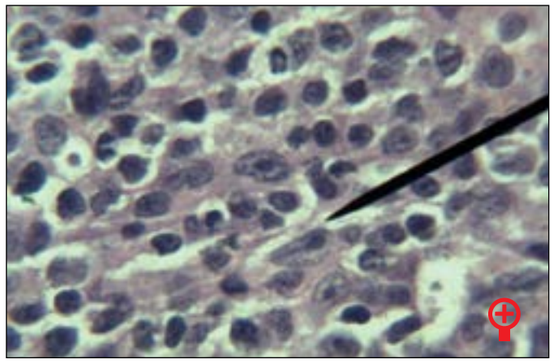
Inflammation has often been implicated in the development of neoplasia in cats – most notably with inflammatory bowel disease/intestinal lymphoma and vaccine-associated fibrosarcoma[6]. Inflammation and stimulation of the immune system may also play a role in neoplasia regression and this may be utilised in cancer treatment protocols (Immunotherapy). The following report documents a case of apparent lymphoma regression following surgical biopsy.

Case Report

A desexed male Domestic Shorthaired cat, aged approximately 5 years and weighing 8 kg was presented for a lump which had only just been noticed by his owners. The cat had otherwise been well with no changes seen in appetite, elimination, activity levels or demeanour. On examination there was a firm, non-painful, fixed mass approximately 3cm in diameter in the right cranioventral neck region. Other than excessive weight, no other abnormalities were found during the clinical exam. Fine needle aspiration of the mass was unrewarding (only blood obtained) and surgical biopsy was recommended.

Pre-anaesthetic blood tests were recommended but declined by the owner. A punch biopsy of the mass was taken under general anaesthetic and sent for histopathology (Figs 1A & 1B). Removal of the mass was not attempted at the time due to the close proximity of the major blood vessels of the neck with the mass.





Figures 1A & 1B. Histopathology Results

This is a lymph node replaced by an extensively necrotic tumour composed of pleomorphic frequently huge large histiocytic cells with frequent inclusion-like macronuclei, eosinophilic or clear cytoplasm, and a low mitotic index. They are accompanied by medium-sized and small lymphocytes. The changes are consistent with an anaplastic lymphoma. The disease may be a manifestation of T-cell/histiocyte-rich B-cell lymphoma (Hodgkin-like lymphoma). Immunohistochemistry is recommended to determine the T-cell and B-cell makeup of the neoplastic cells. This would allow a more specific diagnosis. – IDEXX Laboratories

The results showed an anaplastic lymphoma with a low mitotic index. It was recommended that the tumour be further staged to help determine the most appropriate treatment path. Further investigative options included screening thoracic radiographs, abdominal ultrasound and determination of FIV/FeLV status as well as routine haematology and biochemistry. Potential treatment options included chemotherapy, surgical resection at a referral centre, radiotherapy or a combination of these e.g. chemotherapy or radiotherapy to shrink the mass followed by surgical resection if necessary. Unfortunately, all further diagnostic procedures and treatment options for the mass, including palliative treatment with prednisolone, were declined by the cat's owner.

Three weeks after the biopsy during a follow up telephone call, the cat's owners revealed that the mass had disappeared completely 2 weeks after the surgery and that they could not find any other masses on the cat. Multiple requests to bring the cat back in for re-assessment were declined.

Six months after the biopsy the cat was represented. His weight had increased slightly since his previous visit (8.2kg) but he had had a reduced appetite over the previous 12-24 hours and had been making a snuffly noise during respiration for the previous 1 to 2 weeks. There was no nasal discharge, no facial swelling, no oral lesions and no abnormal masses detected on palpation of the throat or neck region. His rectal temperature was normal. The respiratory noise was isolated to the upper airway. The main differential diagnoses included upper respiratory tract infection (bacterial, viral, fungal infection), foreign body and, taking into consideration the previous history, nasopharyngeal mass. He was started on antibiotics and it was recommended that further investigative work-up be undertaken if there was no improvement seen by the end of the medication course.

The cat was re-examined 2 weeks later – the cat had responded to the antibiotics but the snuffling recurred once the course was completed. He now weighed 7.8kg and had a markedly reduced appetite. There was a loud stertor and no nasal discharge. The eyes were slightly reddened but there was no ocular discharge. No oral lesions were present and the rectal temperature was normal. A large smooth, non-painful mass approximately 3x6cm in diameter was palpable on the left side of the cranioventral neck region. The main differentials were nasopharyngeal neoplasia and fungal infection. Recommendations for further investigation with biopsy of the mass, endoscopic/radiographic examination of the nasopharynx and referral for MRI/CT scans were declined. Palliative care with prednisolone (on the presumption of recurrence of the lymphoma) was also declined. The only treatment option accepted by the cat's owner was a repeat course of antibiotics for symptomatic control of the snuffling.

The cat presented 2 weeks later with marked facial swelling especially across the bridge of the nose, bilateral proptosis, and loud stertorous respiration (Fig 3). The mass on the left side of the throat had increased in size. Euthanasia was elected at this time. ▶

Figure 3. Marked facial swelling.

Authors' views are not necessarily those of the CVE

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Discussion

Spontaneous tumour regression without specific treatment is a known, but uncommon, occurrence which has been documented in both human and veterinary literature and is suspected to result from stimulation of the immune system by various mechanisms[2,4,7,8].

Spontaneous regression occurs more commonly in indolent/low-grade lymphoma compared to the high-grade type. Low-grade lymphomas may not be full-blown malignancies and so potentially respond more favourably to immunotherapy; however, they may become more aggressive and malignant with progressive cytogenetic events and so become more resistant to the host's immune defences[4]. This may arise through mutations or deletions in genes encoding the tumour antigens or immunoselection may favour the growth of tumour cells with mutations in MHC genes[1]. Mutations are more likely in cancer cells due to the inherent instability of their genome and high mitotic rate.

In this case it was suspected that the combination of an inflammatory response occurring with the trauma of the surgery and the potential exposure of cellular material from the centre of the mass to the immune system was enough to induce a significant immune response in the cat to cause regression of the tumour within a short period of time. The development of a rapidly progressive nasal condition in association with a large cervical mass 6 months after the regression of the original mass was suspicious of an aggressive neoplastic condition, possibly related to the original lymphoma. However, this suspicion could not be confirmed and other conditions including fungal infections and neoplasia unrelated to the original mass were part of the differential diagnoses considered. The partial response to antibiotics was thought to be due to control of secondary bacterial infection.

Conclusion

The study of spontaneous regression of tumours has played an important role in the development of immunotherapy as part of cancer treatment, especially in humans. Tumour-specific vaccines, non-specific immunomodulation using bacterial components, interferon and various immunomodulating drugs have been used to treat cancer with varying degrees of success[1,7], even when traditional chemotherapy has failed[5].

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Delayed presentation of Tiger Snake envenomed dogs

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I would like to bring to the attention of the readers of *Control and Therapy*, the issue of delayed presentation of dogs that have been bitten by Tiger Snakes. Over the last summer I have examined 2 dogs and I have spoken to 3 veterinarians by telephone in relation to dogs that have presented with severe muscle weakness, atrophy and in some cases marked myoglobinuria. In each case the dog was seen to be ill in an initial consultation up to 10 days prior to presentation for muscle atrophy and weakness. Many of the dogs were initially examined for non-specific illness and or vomiting. In one case the dog went on to develop facial nerve paralysis. None of the dogs was suspected of having been bitten by a Tiger Snake and appropriate testing that would have confirmed the bite was therefore not run. It seems likely that many of these dogs were envenomed with a sub lethal amount dose and therefore did not go on to develop clinical signs that would have alerted the clinician to the possibility of Tiger Snake bite. These dogs then go on to develop marked muscle disease due to a toxin called Notexin that is a component of Tiger Snake venom. This toxin appears to preferentially destroy red muscle fibres. Furthermore, exercise and exertion would seem to exacerbate the damage and so strict rest is a feature of treatment of these dogs to prevent complete muscle breakdown and failure of respiratory muscles. In one such case a dog required mechanical ventilation for 10 days.

There seem to be 2 very important factors in identifying such dogs. The first is a clinical suspicion in any dog with potential exposure to snakes. The second is the measurement of creatinine kinase (CK). Any potential increase in CK should be considered as suspicious for snakebite and any continued increases should alert the clinician to the possibility of a delayed presentation. It is unknown at what time the urine will become negative following snakebite for venom detection tests but it may be as early as 48 hours post bite. This makes diagnosis in delayed presentation problematic. The muscle damage as indicated by increased serum CK will usually become evident within 24 hours and urine may indicate trace myoglobin on dipstick. This should be an early warning to consider the use of snake anti-venom. It is also difficult to predict the usefulness of anti-venom beyond the first 48 hours post bite. However, it is becoming clear that identifying dogs that are a delayed presentation may be useful as restriction of exercise may result in a reduction in the muscle damage. Restriction of any exercise for up to 2 weeks post bite is probably a good idea to try and reduce the muscle damage. Intravenous fluids may be needed in cases with pronounced myoglobinuria so as to prevent renal failure.

In several cases I have seen, the affected dogs have not had biochemical analysis run that did not include CK. This led to a failure to identify the problems as being related to a Tiger Snake envenomation. It would seem a good idea to me that commercial laboratories always include CK on biochemical analysis. Identifying increased CK in areas where Tiger Snake envenomation is likely may be the only early clue that allows prompt action.

Here is a story with a moral for you

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Yesterday early afternoon, one of my cats ('Pepe', a 2-year-old healthy male DLH) started to vomit in our living room, as cats do, so, as owners do (!), so we chucked him outside so he could throw up in the garden instead of on the carpet. Later I noticed him sitting not far from the back door. A few hours later he was in exactly the same position which I thought odd. When he came in he vomited up a lot of brown water with the smell of faeces and collapsed. Two abdominal masses on palpation, a smaller, firm caudal one and a large, slightly softer cranioventral one which felt to be in the stomach and when palpated caused more fluid to come up – some out of his nose. Straight to the hospital – ultrasound could not identify the masses because they were surrounded by gas, but clearly in the gut. 'General health profile' was unremarkable except for a marked neutrophilia. Conscious lateral radiograph unremarkable except stomach dilated, some gas but mostly soft-tissue density. His condition was deteriorating rapidly so he was put on a drip, administered a general anaesthetic and the stomach tube removed a lot of faecal-smelling brown water. On ex-laparotomy, a solid furball impaction was seen jammed in the jejunum and a very large firm furball in the stomach – so enterotomy and gastrotomy (what did you spend yesterday evening doing?).

Pepe recovered well the next day but was but spaced out on buprenorphine!

Moral – if your cat throws up, don't just throw him out!

Pepe is out all night every night, and stays in all day every day (unless we throw him out because he is about to chuck up!). Despite being a very long-haired moggy, he does not bring up furballs – my wife and I cannot remember him ever having done so. Instead, he saves them until he gets an intestinal impaction! ▶

Invited comment courtesy of

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After doing over 200 laparotomies with small bowel biopsies, it is clear to me that we (veterinarians and cat owners) have taken vomiting and hairballs far too casually. The paper we just submitted concerns 100 cats with confirmed small bowel disease. Each presented with chronic/recurrent vomiting, weight loss, chronic diarrhea or a combination. Vomiting and weight loss were far more common than diarrhea. Two of the cats in our paper had hairball obstructions. One had underlying inflammatory bowel disease; the other had lymphoma.

Chronic small bowel disease causes hypomotility resulting in vomiting and inability to move hair through the GI tract. The result is hair buildup in the stomach and small bowel. Therefore, when a hairball obstruction is relieved surgically one should take full thickness biopsies of 2 or more places in the small bowel. One should avoid the immediate area of the obstruction as inflammation would be expected there. Biopsy 10+ cm oral and aboral from the point of obstruction. This process will allow you to understand the underlying pathology in the small bowel and put you in a position to prevent further disastrous events.

Response to Gary Norsworthy's comment:

Hairball obstructions of the intestines are uncommon in domestic cats – our practice's 5 small-animal vets can only remember seeing 1 case other than Pepe. However, they do occur and as Gary points out, evidence is accumulating that hairballs and, in particular trichobezoar obstructions, appear to be associated with underlying gut pathology such as inflammatory bowel disease or lymphoma, even in the apparent absence of any other signs of underlying gut pathology. Martha Cannon, an RCVS specialist in feline medicine, recently published an excellent review of hairballs in cats making the same point (Cannon 2013).

As a follow-up to the above tale: Pepe recovered fine from his surgery and remained healthy thereafter. However, 10 months after his obstruction, even though Pepe appeared totally healthy, I detected another gastric trichobezoar on palpation. So, back to the hospital for another gastrotomy! This time, much of the small intestine appeared diffusely slightly thickened and we took biopsies of both stomach and intestine. The histopathology report was mild lymphoplasmacytic enteritis, and also mucosal fibrosis of the stomach.

What am I doing for Pepe now? He is on a commercial 'hairball control' food containing psyllium and is given some flavoured petrolatum-based laxative (Lax-A-Past, Animalcare) 2 or 3 times a week. I am not specifically treating the (otherwise) asymptomatic inflammatory bowel disease. He does not over-groom and he has regular flea treatments. I regularly palpate his abdomen. If he does gets another gastric trichobezoar, future preventative options include regular pro-kinetics or, most likely, resorting to the drastic measure of a regular 'lion clip' – which is going to look very odd on a black and white cat!

Reference

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Cutaneous asthenia: a case study

C&T No. 5310

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Ducati

'Ducati' is an 11-month-old white neutered male Domestic Short Hair Cat who was presented on 20th April 2011 for a small mobile nodular hard mass on dorsal thoracic region caudal to shoulders. No other skin signs observed. Ducati was otherwise healthy, HR180, pink and moist mucous membranes. Chest and abdomen are normal. Fine needle aspirate results reveal small numbers of cocci. Ducati was started on 62.5mg Clavulox drops PO twice daily for a week. Differential diagnoses included abscess, cyst, foreign body reaction and neoplasia.

Client communication on 26th April to owner revealed that Ducati's mass had responded slightly to antibiotics and owner thought mass was smaller but harder now. Ducati was rechecked and admitted for boarding, lumpectomy and histopathology on 30th April as mass had not resolved completely. Physical examination revealed that the mass had grown larger and more 'lumpy' and harder in consistency.

Lumpectomy was planned for 2nd May. Prior to the anaesthetic, the surgery vet found another mass subcutaneous nodular mass with poorly defined margins in right inguinal region. We then thought that the masses are more likely to reflect a systemic disease process, rather than locally occurring non-related diseases. We changed the diagnostic plan to obtaining samples for biopsy and submitting these for histopathology, bacterial and fungal cultures after obtaining permission from the owner.

Post-operatively, Ducati was started on Temgesic® orally and Metacam® orally for pain relief and Clavulox®, Baytril®, Flagyl® for antimicrobial therapy. Histopathology results returned as 'immature organising fibrous tissue – non-suppurative, non-neoplastic, suggestive of infection'. Bacterial and fungal culture later returned as all negative. We then requested the pathologist to look for organisms with special stains. Initially we thought it may be something strange like mycobateria so we contacted Richard Malik . He was immensely helpful with this case and soon agreed to meet Ducati to try and help us find a diagnosis.

By the time Richard saw him, Ducati's previously biopsied mass in inguinal area had grown to 3 times its original size and other new masses erupted under the left hindlimb and over the left antebrachium. Under Richard's advice, we also did Xrays of the thorax and abdomen and both of these were normal. The special stains (PAS, 2N) the pathologist performed for us also returned negative.

On 15th May, Richard examined Ducati and performed ultrasound and fine needle aspirates on various masses on Ducati. No organisms were found and some of the masses seemed to be just seromas/scars so Dr Malik thought that Ducati may have a rare manifestation of feline cutaneous asthenia. After this enlightenment, we stopped all antibiotics as Ducati was very healthy clinically and the masses showed no response to anti-microbial therapy for the last few weeks.

As he continued boarding with us, Ducati's masses became smaller but harder. He also developed a new lump on the nuchal crest on the 31st May. Despite the changes, Ducati remained a healthy cat otherwise. He was discharged from boarding on 14th June and is living a happy life at home.

Cutaneous asthenia (collagen dysplasia, dermatosparaxis)

This is a rare congenital hereditary disease in cats and dogs similar to Ehlers-Danlos-Syndrome in humans. It is known to occur in many other species of animals such as mink, rabbits, horses, sheep and cattle (known by different names in different species – for example, the horse equivalent, hyperelastosis cutis, is becoming increasingly more common as it runs in cutting horse lines of Quarter horses). Note that this is NOT acquired skin fragility syndrome seen in older cats induced by drugs (e.g. prednisolone), endocrinopathies (hyperadrenocorticism) and other disease. Both diseases have similar presentations but signalment and history usually helps to differentiate them.

Cutaneous asthenia is characterised by defects in connective tissue of the dermis. It has been proved that defects in collagen structure are connected to a shortage of pro-collagen peptidase and increased in collagenase activity. In cats, it is both autosomal dominant and recessive and has been found in Burmese/Himalayans/Persians/other long-haired breeds and Domestic Short Hair cats. No sex predisposition is found in any species.

Clinical signs are mainly confined to the integument. Affected animals have thin, hyperextensible skin easily torn and prone to injuries, like Ducati, our case. They develop bleeding wounds/haematomas/seromas mainly on the head and the neck. Most case reports in dogs, cats, horses and cattle had skin signs in the more dorsal parts of the animal (such as the trunk) rather than the extremities and the abdominal surface. This may be due to increased tensile strength in these locations. Cutaneous asthenia can manifest in joints (joint laxity in dogs and cats), blood vessels (increase fragility), ocular system (e.g. lens luxation) or present as hernias in cats.

Diagnosis is based on clinical signs and ancillary tests. It is important to perform skin extensibility index in a physical examination. The length of a skin fold is measured on the back by pulling skin away from the spine until able to elicit pain (Figures 11B & 12). The length of an animal is its length from occiput to its tail base.

$$\text{Skin extensibility index} = \frac{\text{Length of a skin fold}}{\text{Length of an animal}} \times 100\%$$

Average values are over 14.5% in dogs, over 19% in cats and 19.2% in rabbits. Values higher than these would increase level of suspicion for cutaneous asthenia. This is regarded as the most important clinical feature.

Sometimes histopathology (H&E and special stains like Van Fieson, Mallori, Masson) helps to confirm the diagnosis. Histopathology should reveal abnormal structure of collagen fibres that lay at a distance from one another and are fragmented, shortened and irregular in appearance. Definitive diagnosis usually involves usage of electron microscopy , which shows irregular structure of collagen fibres.

Prognosis is poor as there is currently no cure and skin can become increasingly fragile with age. Many animals are euthanased due to young age and severe clinical signs. Anecdotally, Vitamin C has been used in treatment of cutaneous asthenia with no drastic improvement. Doses are 50mg per cat daily and 500mg per dog daily and it's thought that vitamin C may increase collagen in the skin.

Post-Script: The patient was lost to follow-up as both authors left the clinic. However, Anita commented that although she can't remember her records, they did measure the skin flexibility and estimates it was approximately 20-25%. ▶

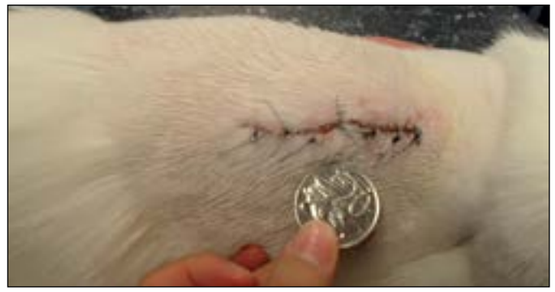

Figure 1. Size of surgical wound as compared to a 20 cent coin.

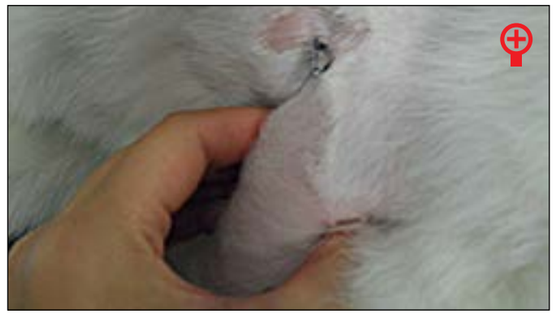

Figure 2 & Figure 3. Very stretchy skin as shown in the pre-op and post-op images above.

Authors' views are not necessarily those of the CVE

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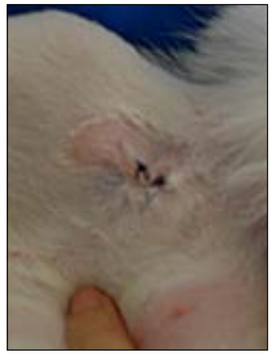

Figures 4 & 5. Pre operatively, large lumps can be seen.

Figure 6. Dorsal lumbar lump.

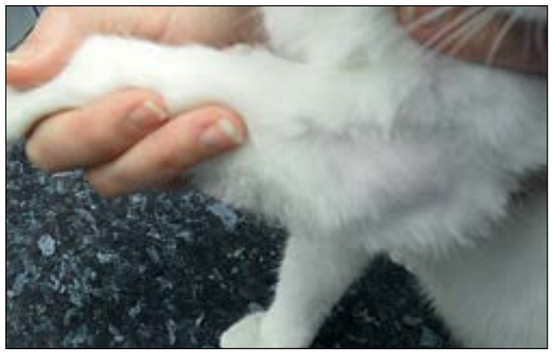

Figure 7. Left elbow lump.

Figure 8. Nuchal crest lump.

Figure 9. Nuchal crest lump lateral.

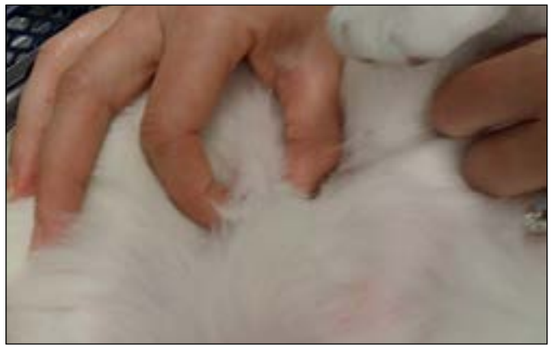

Figure 10. R inguinal lump.

Figure 11A. Burmese cat with CA (courtesy of Dr Amanda Burrows)

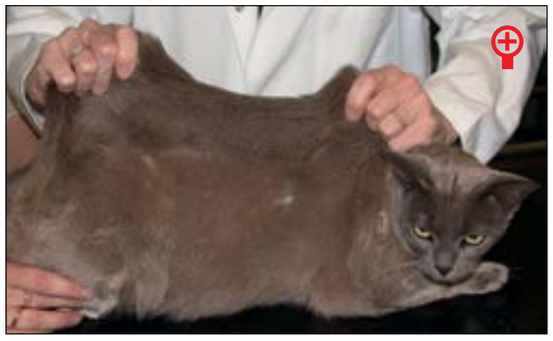

Figure 11B. Hyperextensibility in affected CA Burmese cat (courtesy of Dr Amanda Burrows)

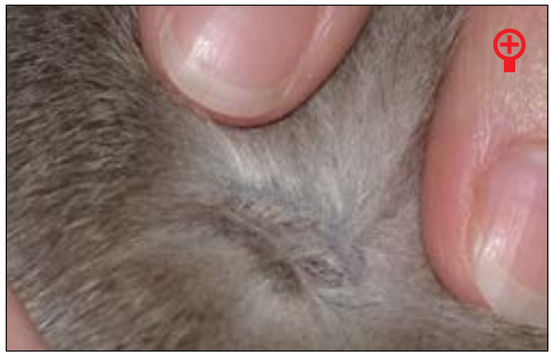

Figure 11C. Close-up of cicatrial alopecia/scar in affected CA Burmese cat (courtesy of Dr Amanda Burrows)

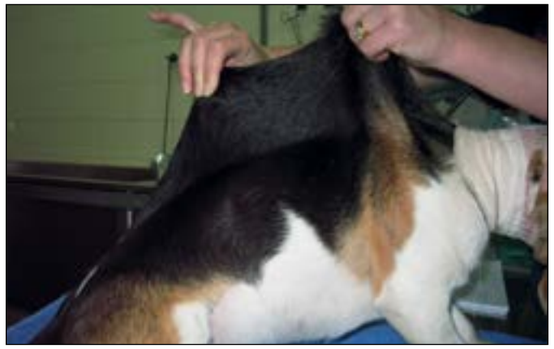

Figure 12. Photograph of a dog with cutaneous asthenia. Note how far the skin 'tents'.

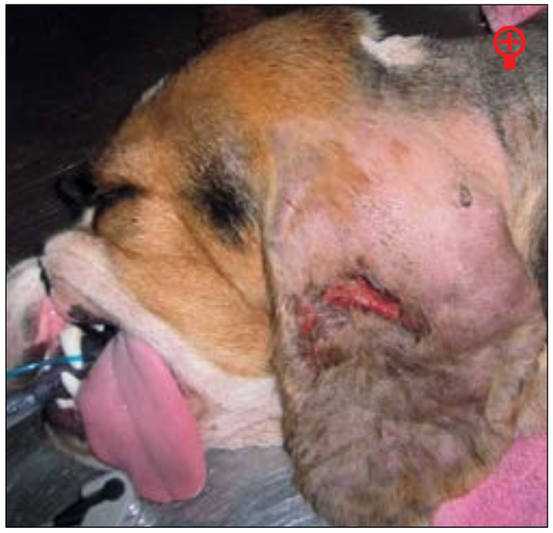

Figure 13. Photograph of a wound on a dog with cutaneous asthenia – this occurs because the skin is so fragile.

Editor's Note

Thanks to Dr Amanda Burrows BVMS MANZCVS FANZCVS Veterinary Dermatologist, Registered Specialist in Veterinary Dermatology at the Skin, Ear and Allergy Clinic at Murdoch University Veterinary Hospital for supplying images 11A, B & C.

A blade of grass causing respiratory distress and epistaxis in a cat

C&T No. 5311

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A 3-year-old female spayed domestic short-hair cat presented to the Alamo Feline Health Center for evaluation of respiratory difficulty of 19 days duration. The cat had an acute onset of sneezing with epistaxis.

The cat presented with paroxysmal episodes of severe sneezing which had been occurring for three weeks. These episodes began after the cat was let outside for the afternoon. When the cat came back inside, the owner noticed vomitus containing grass and blood on the floor. The owner also noted that the cat was experiencing severe bouts of sneezing. Prior to presentation at the Alamo Feline Health Center, the cat was evaluated by the owner's regular veterinarian. On presentation the cat was bright, alert, and responsive. Physical examination revealed epistaxis and dysphagia. The patient received a dose of Convenia®. One week later the cat was reassessed by the same veterinarian. The patient was subjectively improved, but still experienced bouts of dysphagia and unilateral epistaxis. At this time the cat was administered an antihistamine, but there was no response. The cat was subsequently referred for further evaluation and rhinoscopy. Physical examination was unremarkable except for the respiratory system; where a lesion was localized to the nasal cavity or nasopharynx. Epistaxis was still evident from the right nostril. CBC and Chemistry testing using the Abaxis VS2 and HM5 were normal. Endoscopy of the nasopharynx was performed using a 3 mm flexible rhinoscope. No foreign material was found, but a purulent discharge emanated from the right choanus. Antegrade rhinoscopy was then performed using a 1.7 mm rigid arthroscope. A green object was visualized briefly approximately 1 cm from the right external naris. It was not possible to grasp this foreign body using forceps. However, retrieval of grass in this manner may result in tearing of the grass blade and retention of the caudal portion. A nasal flush was performed using 60 milliliters of warm saline in an attempt to dislodge the object from within the nasal cavity, but flushing did not yield positive results. Therefore a dorsal rhinotomy was performed the next day to explore the anterior right nasal cavity. Anesthesia was induced by the inhalation of isoflurane in an induction chamber and maintained using isoflurane for the duration of the procedure. Following a dorsal midline skin incision and dissection to the underlying bone, a 1.5 cm incision was made to the right of the midline using a power drill. The foreign body was blindly grasped with curved mosquito hemostats (Figures 1 a, b, c). ▶

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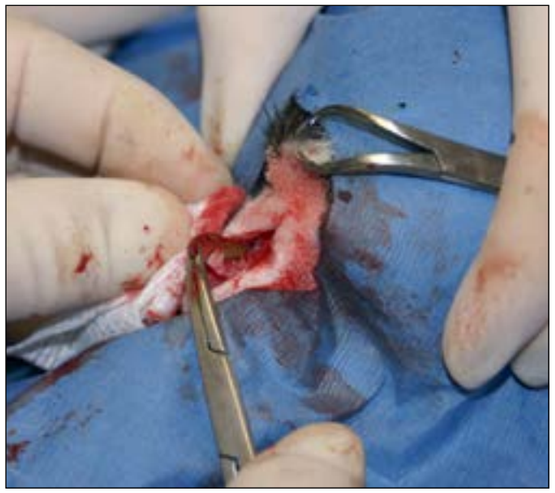

Figure 1a: Progressive extraction of the grass blade through a rhinotomy incision.

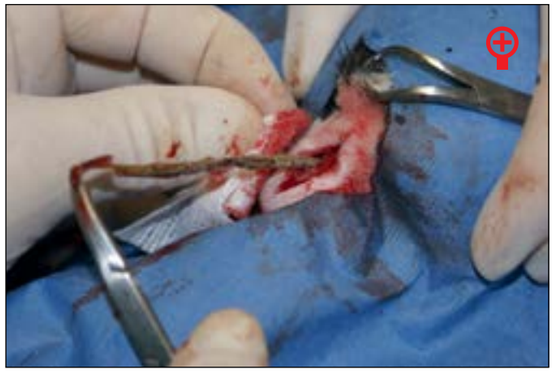

Figure 1b: Progressive extraction of the grass blade through a rhinotomy incision.

Figure 1c: Progressive extraction of the grass blade through a rhinotomy incision.

The foreign body was identified as a 6 x 0.25 cm grass blade, that was surrounded by inflammatory exudate. The incision was closed using simple interrupted suture pattern using 4-0 PDS®. The patient given buprenorphine immediately post-operatively and every 12 hours thereafter for three days; it was discharged the next day. Recovery was uneventful (Figure 2).

Figure 2: The patient one week following rhinotomy.

Discussion Nasal disease in cats can be infectious, allergic, neoplastic, congenital (due to anatomic defects), traumatic, or referable to a foreign body. Nasopharyngeal conditions such as polyps or nasopharyngeal stenosis may lead to similar clinical signs, in particular stertor, and should be included in the differential diagnosis.[1] The type and location of nasal discharge assists in narrowing the list of differential diagnoses. Unilateral nasal discharge is usually seen with nasal foreign bodies and nasal neoplasia. Bilateral nasal discharge is much more common and may be due to all of the listed causes above.[2] Nasal or nasopharyngeal foreign bodies occur infrequently in cats and are usually due to blades of grass lodged within the nasal cavity.[3,4] Foreign bodies commonly reported in the feline nasopharynx include grass awns, grass blades, fish bones, tablets, and sewing needles; basically, anything that can be swallowed, can be deposited into the nasopharynx following "inaccurate" emesis. The foreign body typically reaches the nasal cavity or nasopharynx during vomiting or regurgitation. This may also occur through direct antegrade introduction into the nasal cavity, although this is much less common in cats than dogs, on account of the smaller size of the feline nares. Most foreign bodies, including barbed grass awns, seeds, and blades of grass, favor one way migration and are difficult to expel backwards out of the naris.[5] When the foreign body embeds into the nasopharyngeal region, peracute clinical signs of stertor, inspiratory dyspnea, snoring, dysphagia, and epistaxis occur. These signs worsen as inflammation progresses.[2] This case presented with a history consistent with nasopharyngeal deposition of grass following vomition. The acute onset of signs, initial dysphagia, presence of epistaxis, and antecedent vomition of grass blades, strongly suggested this etiological diagnosis. The presence of a foreign body can be diagnosed by radiography, endoscopy, nasal flush, blind exploration with forceps, or rhinoscopy.[6] Additionally, a magnetic resonance imaging (MRI) or computerized tomography (CT) scanning may be used to gain better resolution of foreign material within the sinonasal cavity. Radiographic signs in cats with rhinitis tend to be extremely variable. Radiopaque foci may be identifiable in cases of foreign bodies, but only if they are of bone, glass, hard plastic, or metal origin (i.e., sewing needles, metallic airgun pellets).[7] Most nasal and nasopharyngeal foreign bodies result in little to no radiographic change. Some will demonstrate a unilateral fluiddensity.[5] Due to the radiolucency of grass blades, radiology would have been of no benefit in the present case. Nasal flushing of the nares may be performed by packing the caudal oropharynx with gauze and flushing the nasal cavity with warm saline through a [3.5] F. urinary catheter. This technique may dislodge small particles from the nares.[2,5] Rhinoscopy is commonly used to search for foreign bodies in the nasal cavity. Rhinoscopy is a valuable tool in viewing the internal structures of the nasal cavity, identifying anatomic abnormalities, and locating nasal foreign bodies. There is, however, a direct relationship between the diagnostic benefit of rhinoscopy and the size of the patient undergoing the procedure. Retrieval of a foreign body via direct visualization is preferred whenever feasible; however, this approach is usually thwarted by the anatomic limits of the feline patient's nasal cavity, the comparative dimensions of the indwelling instrument, the location of the lesion, and the presence of mucus and hemorrhage.[2,8] Instruments must not be advanced blindly beyond the medial cantus of the patient's eye to insure that the instrument does not penetrate the cribriform plate. Inadvertent penetration of this nasal anatomic barrier to the brain has obvious and potentially fatal consequences.[8] In this case, only a "hint" of the foreign body was able to be visualized in the rostral right nasal cavity by rhinoscopy, but anatomic and instrument limitations prohibited retrieval of the foreign body with visual guidance. This "hint", in addition to the cat's history and the presence of purulent material in the nasopharynx, was deemed sufficient evidence to recommend surgical exploration of the right nasal cavity. In conclusion, the differential of a nasal or nasopharyngeal foreign body must be considered in cases with acute onset of severe upper respiratory signs, especially when the signs are unilateral. History and clinical signs may provide sufficient evidence to pursue further diagnostics regardless of the lack of abnormal radiological findings. Prognosis is excellent with removal of the foreign body.

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[6]Henderson SM, Bradley K, Day MJ, *et al.* Investigation of nasal disease in the cat – a retrospective study of 77 cases. *J Fel Med Surg* 2004; 6: 245–257.
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WINNER

External markers of internal disease; the 'not-so-humble' nail clip

A nail clip should be mandatory before an MRI or CT!

C&T No. 5312

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I know, we vets hate nail clips and fob them off to the Nurses as fast as we can. But there is a mine of information on a nail clip – it's a WELLNESS profile right in front of you, as good as any expensive blood screen!

Figure 1. Popping the hood/bonnet - SLOD

So next nail clip, open your eyes, your ears and engage your sense of smell and touch.

We have lost count of the number of second opinions on our practice for recalcitrant lameness and/or weakness where nothing had been found on extensive work-ups – except no-body checked the nails!

A recent second opinion was a dog with suspected spinal neoplasia. My partner Mark Weingarth shaved the over-long hair and discovered the cause of lameness and could replicate it for the client – the over-long hind leg dewclaws were interlocking and binding the dog's hind legs, causing the collapse. The consult air was blue when the client realised the contrast myelogram/CT scan they would still be paying off should have been a $25 nail clip!

Equally, old arthritic dogs not responding as well to NSAIDs just need their matted feet and overly long nails clipped to allow the animal to walk with good grip and pain free.

So – External Markers in a nail clip:-

- There is a feel and shine and a clicking 'sound' you get when clipping healthy nails. LEARN WHAT THE NORM IS by doing nail clips on healthy animals.
- In Diabetes and hypothyroid you lose that 'click' sound and when you check you find that the nails are softer, the blood vessel narrower and when you question the client, - yep, the patient is often drinking or urinating more or gaining weight. Poor/deficient diets can cause scurfy nails.
- Less near the tip as older growth and may not be affected if the SLOD onset is recent.
- Canine Symmetrical Lupoid Onchodystrophy is more common than you think (Seavers, A. Sept 2009. Spotlight on SLO - Symmetrical Lupoid Onychodystrophy Treatment, *The Veterinarian*, Pg53-54.) and the nails show intense pain and ridging and splitting on the length of the shaft cranially – less near the tip as older and may not be affected if the condition is recent. Once you see the 'popping the hood/bonnet' picture (Figure 1) you will never miss this condition again.
- Yeast infections in the nail bed suggest allergy/atopy/contact/allergy/autoimmune/canine hepatocutaneous syndrome depending on the age and breed of the dog.
- Shredded nails can mean separation anxiety as well as recent trauma.
- Long over-grown nails means the animal is left alone for long periods of time or never walked. They can also mean that the dog won't let the owner near the nails to clip them, in which case behaviour modification and large emery boards are needed.
- Long nails also suggest the dog can't walk due to pain in the toes or because the nails are so long that they physically bend the digits out of correct placement alignment. Abnormally worn nails imply neurological issues.
- Nails are a bit like the markers of internal disease that indicate a bitch licking her vulva/dog his prepuce are often the first signs of diabetes or bladder stone, as well as the normal contact/fold issues.

Read next C&T edition in Sept for a huge gallery of images provided by my colleagues from around the world of not only canine but also feline nail disease presentations that may present as a lameness/neuro cases when they are in fact primary dermatology/neoplastic/bad management conditions.

Authors' views are not necessarily those of the CVE
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WINNER

Secnidazole as a one-dose for *Giardia*

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I have been using secnidazole over the last few weeks with some excellent responses in young and old cats with diarrhoea. I see a lot of *Giardia* down here in Frankston. Not all the cats have been *Giardia* positive on antigen fecal test but responses so far have been excellent with a **single oral dose** compared to my poor response experiences to daily metronidazole or ronidazole for weeks on end. I had it compounded into capsules by West Lindfield Pharmacy (Sydney).

I have posted a note on http://vetbook.org at **http://vetbook.org/wiki/cat/index.php/Secnidazole**

The article that inspired me to try the drug was: Da Silva AS, *et al,* Secnidazole for the treatment of giardiasis in naturally infected cats, *Parasitol Int* (2011), doi: 10.1016/j.parint.2011.06.024.

No toxicity noted as yet – dosing at 30mg/cat stat (a little on the underside of recommended dose for cats).

I treated a 13-year-old DSH with chronic bloody diarrhoea unresponsive to dietary change with Convenia, depomedrol and metronidazole. I was going to do a laparotomy with the expectation of a colonic adenocarcinoma, so trialed secnidazole over the weekend and saw the cat again 3 days post-treatment. Faeces were normal for first time in about 6 months! I am unsure if it was an anti-inflammatory effect of the drug or the haematochezia was a consequence of coccidiosis (not something I thought of in a geriatric cat).

Editor's Note

Giardia *is now the most preventable gastrointestinal parasite of dogs and having a once only treatment is a big advance on treating every day for 3-5 days because it means the vet can give the treatment at the time of an annual health check. Therefore, we believe there is a case for treating every dog and every cat in Australia once a year – just as a routine preventative.*

The average dose is 30mg per cat. This medication may be available from compounding pharmacies at an approximate price.

Invited Comments courtesy of

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We too were intrigued by this publication regarding secnidazole as a single modality treatment for *Giardia* in cats following on from its excellent results in human giardiasis. We too see many cases of *Giardia*, mostly in purebred cats. These are confirmed by fresh faecal exams, faecal antigen IDEXX SNAP tests or Faecal PCR's (many cats with *Giardia* also have intercurrent *Tritrichomonas* infections).

We have treated 20+ confirmed cases with secnidazole 30mg/kg single dosing since reading this publication. I would say our clinical impression is that it is certainly a useful medication with about 90% cure (post dose antigen testing) or positive clinical response. But we have likely seen more treatment failures compared to our impressions of the clinical efficacy of 5 days of fenbendazole 50mg/kg PO 5 days.

I would see this difference as being in part due to the necessity for environmental control of giardiasis in multi-cat households with re-infection being common. Repeated dosing may be required for improved efficacy to catch any environmental re-infections. The largest benefit of secnidazole is the single dose, readily compounded into a single capsule dose – but at a cost. Fenbendazole is cheap but can be difficult to administer to cats due to its yucky taste.

We did initially hope that secnidazole may show clinical efficacy against *Tritrichomonas foetus*, but this certainly has not been the case, with ronidazole 30mg/kg PO SID 14 days being the only proven therapeutic at this point.

We certainly do use secnidazole in empiric therapy where giardiasis is highly suspected or as a rule out option in cases of chronic diarrhoea in young cats.

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I, like Jim, have also found secnidazole to be a great one-dose treatment for feline giardiasis. I have only used the dose recommended by Da Silva *et al.,* which is 30mg/kg (rather than 30mg/cat). I recommend testing cats for *Giardia sp.* before and after treatment with secnidazole to both confirm a diagnosis and demonstrate resolution of the infection. Thus far in 2 cases (out of 9) I have found cats still testing positive for *Giardia sp.* after treatment with secnidazole; however, it is unclear whether this was due to re-infection or drug failure. In cases of persistent giardiasis I recommend re-treatment with secnidazole @ 30mg/kg, and if this is still unsuccessful then metronidazole @ 15mg/kg BID for 7 days or fenbendazole @ 50mg/kg SID for 5 days. The environment should also be carefully investigated for sources of possible re-infection, for example the stocking density, disinfectant used, and the frequency of litter cleaning. I also recommend testing for *Tritrichomonas foetus* in cases of giardiasis as co-infection is common. Ronidazole @ 30mg/kg SID x 14 days remains the treatment of choice for infection with *T. foetus*, despite cases of neurotoxicosis being occasionally reported.

Without doubt *Giardia sp.* and *Isospora sp.* are the most common gastrointestinal parasites seen in Australian cats. Last year I tested 160 asymptomatic shelter cats and found prevalence rates of 10.0% (16/160) and 9.4% (15/160) respectively. These prevalence rates are almost identical to those found by Bissett et al in a 2009 Australian study.1 This finding suggests that all cats should be routinely treated with a routine worming product, as well as doses of secnidazole and toltrazuril to cover giardiasis and coccidiosis. To my knowledge secnidazole has no effect on *Isospora sp.*

1. Bissett SA, Stone ML, Malik R, Norris JM, O'Brien C, Mansfield CS, Nicholls JM, Griffin A, Gookin JL. 2009. Observed occurrence of *Tritrichomonas* foetus and other enteric parasites in Australian cattery and shelter cats. *JFMS*;11:803-807.

WINNER

Enamel hypoplasia in an 8 month old dog: a case study

C&T No. 5314

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'Tinta', a Female Staffordshire Terrier Cross presented to the RSPCA as a puppy of approximately 8 weeks of age. The animal had a history of non-contagious skin disease resulting in hair loss, and was fostered by a carer until the animal was old enough to be desexed. The dog was desexed at approximately 16 weeks of age, and then fostered further until the skin condition resolved. Ultimately, the skin condition was thought to be allergic in origin.

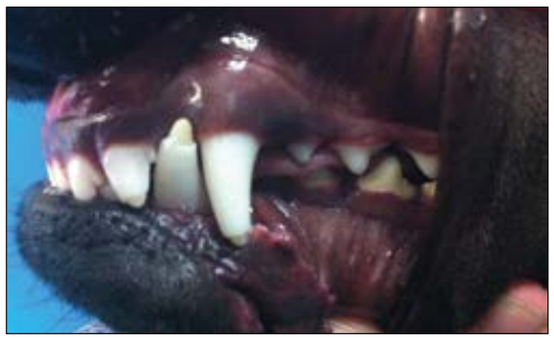

Image 1: Tinta's teeth (left view) at initial examination (7mths old)

The first time I saw Tinta was at 7 months and 3 weeks of age when I was checking her suitability for rehoming. The skin problem had since resolved. On physical examination Tinta's vitals were within normal limits and her examination was unremarkable, apart from one issue – her teeth. Tinta had a condition known as 'Enamel Hypoplasia' which refers to the abnormally reduced formation of enamel on the teeth, leaving exposed dentine. It is sometimes also referred to as enamel hypocalcaemia (as it is defects in calcification of the teeth that lead to this enamel defect). The condition was affecting almost all teeth, with the canines and incisors most obviously affected (See Image 1). The distal tips of the teeth were lacking enamel cover, with exposed brown dentine at the tips of the teeth (which is notable by the reduced diameter of the dentine compared to the rest of the enamel-covered tooth).

Enamel hypoplasia usually becomes evident at the time of (or shortly after) permanent tooth eruption, which explains why it was not evident at the time of desexing, or during previous examinations. Common causes include sustained pyrexia during adult tooth formation, trauma to the tooth root (but with this usually only one or a few teeth are affected), Distemper Virus, poor nutrition and some toxicities. There is thought to be a genetic component to this disease. Tooth roots can be either normal, abnormal (lacking density or floating) or completely absent.

Dental radiographs were then performed for Tinta to determine the viability of the tooth roots, as this would affect Tinta's prognosis. These were performed approximately 10 days after my initial examination. Left and right lateral oblique radiographs were taken of the dental arcades, and all tooth roots were determined to be present and normal (normal radiodensity, anatomy and size). However, since the previous examination 10 days ago the teeth had rapidly deteriorated, with a significantly increased loss of enamel, particularly on the carnassial teeth and canines, and marked deposition of dental tartar on all teeth which was not previously present (See Image 2). Exposed dentine will tend to wear faster, and can also be very sensitive. If affected teeth are viable, the dental tubules should seal themselves off with tertiary dentine internally which means they will not be sensitive later in life.

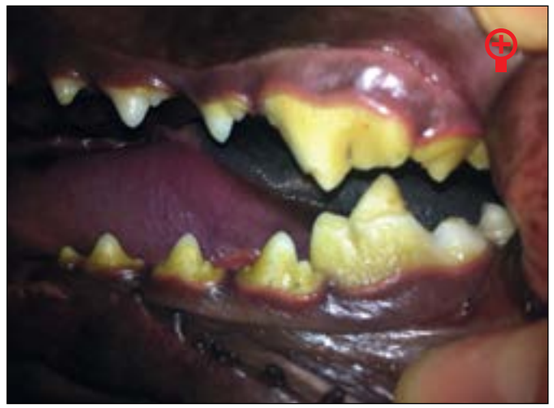

Image 2: Tinta's teeth at second examination (7mths old), just 10 days later than Image 1.

With this amount of dentin exposed, and such rapid enamel deterioration, the teeth would most definitely be painful for Tinta and prognosis for quality of life without major treatment was poor. There was no guarantee in this case that the dentinal ▶

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tubules would seal off with tertiary dentine; this case seemed to be quite severe.

Affected teeth are prone to plaque and tartar accumulation, and vigilant dental hygiene and care is critical in avoiding complications. Animals often require regular dental cleaning. If uncomplicated, enamel hypoplasia can be managed with dental care combined with soft toys and no bones or hard ingestible items. Animals may also benefit from regular fluoride therapy, which assists in reducing tooth sensitivity and hardens enamel. One complication that can occur is infection of the dentine, which occurs due to its porous nature, leading to pulp necrosis.

Some animals may require enamel scrub to smooth tooth surfaces, using white stone burrs or finishing discs. Bonding agents are also available to seal exposed dentinal tubules and protect tooth surface. Metal insert use is an option, however is thought to have a poor long term prognosis. If the tooth roots are absent, the affected teeth would not be viable and would usually require removal. Tinta would be a good candidate for bonding agent use, fluoride therapy and possible metal inserts. If the teeth continued to deteriorate at the same rate it is possible Tinta will need multiple teeth extracted within the next 12 months.

Image 3. Tinta

In many cases the prognosis for a healthy life is good, if the tooth roots are normal, and many animals can live a relatively normal, comfortable life in the care of an observant owner.

Acute skin necrosis in a Kelpie

C&T No. 5315

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Marilyn treated this case whilst working at Springfield District Vets.

Day 1: 'Jack', a 6-year-old, male, neutered, red Kelpie presented to the clinic for being 'not quite right'. He had been lethargic, inappetant, and no vomiting or diarrhoea had been noted. On presentation his abdomen was comfortable; he was tachycardic and was pyrexic with a temperature of 40.7˚C. The only other abnormality found on examination was an oedematous swelling on the left side of his ventral thorax measuring 15cm x 5cm, which the owners said they had noticed in the last few days, which looked very similar to a previous case of suspected spider bite.

In-house general health profile (CBC and Biochemistry) and urinalysis was performed. There were no abnormalities on CBC and Biochemistry, except for a stress leukogram, and the urinalysis showed a USG of 1.010, WBC +1 (but not demonstrated on microscopy).

A fine needle aspirate of the swelling revealed a few neutrophils, although free fluid was not able to be aspirated.

The dog was treated with a carprofen injection and re-checked the next day.

Day 2: The next day Jack's temperature was within the normal range (38.6˚C) and he seemed slightly improved in himself, but was still not eating. The swelling on his ventral chest appeared to have increased in size. A fine needle aspirate of the area was repeated, and this time blood constantly dripped from the site, after the needle aspirate was performed. An activated clotting time was performed and was within normal limits (clotted at 100 seconds). Jack was admitted for intravenous fluid therapy of Hartmann's Solution at twice maintenance rates and a bandage was applied to his chest. He was also started on a course of oral Noroclav® and Carprofen®. By the afternoon, the swelling had extended caudally, to just cranial to the prepuce.

Day 3 to Day 5: Jack stayed in hospital over the weekend and started eating chicken. His temperature remained within normal range however the swelling had increased markedly. I thought at first the cause was IV fluids going extravascular. However the original area of swelling was now alopecic; this area was clipped and revealed a large area of erythematous and black skin. The black areas were moist. At least 3mL of haemopurulent fluid was aspirated, and a Diff-Quik® stained smear revealed cocci +++, some rods and degenerate neutrophils +++. The owners were informed that the area should be drained and cultured; however, they declined culture. To save on funds, Jack was sedated with acepromazine and methadone and local anaesthetic was injected into 2 sites, and the skin incised with a scalpel blade. The area was lavaged with saline and penrose drains were placed.

The owner was advised that more tissue could necrose, so surgery to remove the blackened skin was withheld at this stage. The plan was to continue Noroclav® tablets for 3 weeks, and Carprofen® as required.

Day 7: A re-check was performed 2 days later, and more tissue was breaking down cranially with fluid draining from this site. The owners had been bathing the area with Dettol®, which they were told not to do. A repeat cytological exam was performed, which revealed similar findings. Jack was re-admitted and a course of Enrofloxacin® was added to the therapeutic regimen and was to be continued for 2 weeks, until the Noroclav® course was finished.

Day 8: The eschars that had developed were sloughing off, and surgery to resect the necrotic tissue was performed. Jack was given a pre-med of acepromazine and methadone, induced with thiopentone, intubated and maintained on isoflurane in 100% oxygen. When the skin was being prepped, the eschars were mobile and could almost be pulled away prior to surgery. Large amounts of necrotic skin, subcutaneous tissue and some muscle was removed; the subcutaneous tissues were lavaged with copious amount of saline and penrose drains were placed. The owners were given a guarded prognosis for healing uneventfully.

Day 11 and Day 15: Some of the penrose drains were removed 3 days later and the rest 5 days later. The suture line was looking fine.

Day 24: The sutures were removed 14 days later and all healed well. A definitive diagnosis was never achieved, but I thought that a spider bite was most fitting, but am not sure how exactly to definitively diagnose this.

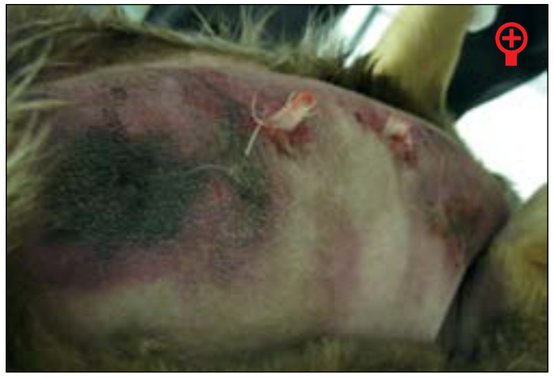

Figure 1. Day 5 after placing penrose drains in ventral most areas. Note the erythema surrounding the black skin.

Figure 2. Day 8, eschars sloughing off, prior to surgery.

Figure 3. Surgical resection of all compromised areas.

Figure 4. Day 11, 3 days after surgery before removing some of the penrose drains.

Figure 5. Day 24, Sutures were removed 14 days after surgery and had healed well.

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Fulminant ascites options – drugs, batteries and scalpels

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A. Zaroxalyn-Metolazone

Zaroxalyn-Metolazone, a thiazide-like diuretic, is a popular drug used by overseas vets for severe ascites cases. My literature search suggests it might be a safer option than spironolactone as the potential gastro-oesophageal side-effects of spironolactone are not reported with Zaroxalyn so perhaps a good option for German Shepherds (GSDs).

(I have had GSDs on spironolactone where the inappetence and cachexia was put down to cardiac causes when it fact it was an undiagnosed gastric ulcer. By the time I saw the GSD it was too late to reverse and the dog bled to death 48hrs later).

Works well in renal failure cases.

However, advised not to be used in Sulphonamide sensitive cases. So that rules out Dobermans and additionally for me that would mean caution in Labradors as they can throw a nasty sulphonamide-induced hypothyroid crisis.

A USA colleagues use 5 mg/per dog per day along with the Lasix they are already on.

Given daily until the ascites resolves then cut dose to every 2-3 days.

Monitor renal function closely doing the first check 5-7 days after starting. Efficacy is superb.

(Trocoxil the long acting NSAID recently released has the same sulphonamide sensitivity warning on it).

B. Mechanical means

I had to euthanise a severe CHF dog. Its massively and recently fluid-distended body being taken home for burial would no longer fit in the special home burial bag the family had personalised and prepared in advance…

So I made a small single stab incision to the side of linea alba to let the fluid drain..And it drained...And drained.. For over 40minutes.. at the speed of a running tap and the width of the top of a biro pen as a constant flow.

The flow was steady and given the distress caused from the massively distended abdomen this dying animal had been exhibiting – it had been unable to sit or lie down or eat much and in fact had spent the previous day trying to sleep standing up – the thoughts struck me that:-

- Given for years we have used decompression to assist in acute abdomen re pressure release**, if women can lose 25% of their *sequestered* fluid volume when giving birth - and that can be over a relatively short period of time for some labours – and not have issues and given how uncomfortable even milder forms of bloated fluid retention abdomen is in menstruating women or IBD people, perhaps it's better to remove the transudate passively but fast over 40mins with minimum restraint rather than slowly over hours with a 3-way tap system and manual restraint? Say to make such an animal comfortable until the owners came back from holiday to be with such a pet if the animal deteriorated whilst owners were away?

I wondered if anyone used stab incisions or small trocar (with local anaesthetic block) to drain such a volume in a terminal patient? One quick nick would allow the animal to just have the fluid drain away with minimal restraint and handling whilst it happened, as opposed to the restraint/sedation/handling needed for needle taps.

The responses to my questions posed above came from vets who do similar approaches with:-

- A small lap incision (~ 1cm) under local in severe ascitic patients, and the fluid just pours out. They don't like trochars or 3-way taps because they take forever and block up with Omentum.
- A 12 G IV catheter (fenestrated to help keep it from getting plugged with Omentum)
- A 16 to 18 G catheter in a big dog that needs some load off the diaphragm. Can fenestrate the 2.5 inch catheter if preferred to use over the shorter one.
- An I/V line.
- A suction pump with suction very low. Adjust the strength of the vacuum as needed to maintain flow but not too much to plug the needle. Wall suctioning used whilst the gauge remains in the green area. In 20 minutes you can remove about 500-800 mL.

  ONLY ACTIVELY DRAIN UNTIL THE animal breaths more comfortably and/or when the skin on the abdomen is no longer stretched like a drum head.

  This does not usually require any sedation (often on cardiac doses of morphine already and may be slight sedated) or just simply ill enough, but can local bleb of lidocaine all the way to the peritoneum.

  Due to the high pressure in these abdomens the hole fails to close immediately after drainage and catheter withdrawal and continues to drip for 1-2 days in decreasing amounts until the pressure is not high anymore. Furosemide and heart drugs do the rest. Somewhat messy but no big deal according to the cardiologists (you can be sure they checked). Usually the dogs are in hospital and on urination pads, often O_2 dependent so not dragging the drip around. These dogs are often stable by day 3. Some cases have dropped from 72 to 50kg over 10days with scant abdominal effusion then.

  Watch for kinking/blocking due to Omentum (which sometimes you can blow away if you have a stop cock on your extension tubing and reposition for continued drainage).

  Each time the owners should be warned that the dog could go into shock and die, but this has not happened yet.

There was a report in *JSAP* 2012* where an automated pump was inserted under muscle and with 2 catheters intra-abdomen to direct the fluid into the bladder. It worked for a couple of months and whilst only a prototype currently, it has promise.

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Animal Pain 1 2 3 4

C&T No. 5317 Download Penny's pain medication charts

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Vets don't recognise and treat pain in their patients enough!

Pain is never helpful in relation to healing and it is our duty to reduce it as much as we can. Narcotics are cheap and writing a few words in a red book is not difficult. I recently had the misfortune to dislocate my elbow falling from my horse (entirely my fault). I did not howl and pace in agony – I sat very still and concentrated on breathing with my eyes shut. If I was a dog or cat in a clinic cage the vet/nurse would have a quick look and say I was OK. I was NOT OK! I was in severe pain and when I finally had IV morphine (with metoclopromide) at the hospital it was an incredible relief. I was still totally aware of what was happening but the pain had gone from a 10/10 to a 3/10. Our patients can't tell us this and by looking at them it is not obvious.

We **need** to think how would I feel in this situation? Surgery is painful, fractures/dislocations are painful. Blocked cats, dog bite wounds are painful. Penny Hocking BVSc, at our clinic, has made easy to use charts that are stuck on the prep room walls – you just need the weight and you have dose/route/frequency there.

NSAIDs such as meloxicam and carprofen are not enough for orthopaedic procedures. Most vet clinics have IV fluid pumps now so making a constant rate infusion is not hard and provides pain relief **all** night instead of for quarter of the night with an injection. Please think if I was – hit by a car/chewed up by a big dog etc. – would I want to rest comfortably for the next 24 hours with some IV narcotics or should I just take a few panadol?!

There are some good books on pain and Penny is happy to share her charts by contacting admin@sheppvets.com.au. She has changed the way we treat pain but my personal experience of pain will change how perceptive I am to animal pain. I have been under-treating it for 18 years – I never used pain relief for routine surgeries in my first job – how unacceptable is that now?!

My next 18 years will be far more pain orientated. How many owners will decline to pay for pain relief? So, please treat pain as a priority.

Reply to C+T No. 5172 – Elevations in ALT and gall bladder ultrasound changes in anaphylaxis – 2 case reports

(Issue 265 Dec 2011)

C&T No. 5318

Rick Atwell (Retired Professor)
BVSc PhD FACVSc
M. 0409 065 255

1 2 3 4 5

Rollover or Download to read C&T No. 5172

I had the privilege to observe over 300 dogs go into (most likely) anaphylactoid shock. Apart from seeing unique behaviour (hiding, seeking darkened areas) in an isolated (strawed) horse stable, you could easily palpate the progressive enlargement of the liver, along the left costal arch, and feel its rounded edges develop. Repeated auscultation, at times, revealed there to be no audible (to my ears) heart sounds in standing dogs, along with the expected shock signs e.g. reduced arterial pulse amplitude etc. At necropsy such dogs had very congested livers (histologically, the hepatic veins were clearly in spasm) and acute, small-volume ascites (blood tinged). They also had acute percentage changes in ALT values over time[1]. No liver U/S was performed but the reported gall bladder changes (5172) would be consistent with liver engorgement. The clinical signs of shock, acute hepatomegaly (with round edged lobes) and altered audible haemodynamics were pathopneumonic. The heart U/S (Video tapes – J. Seton, PhD Uni Qld) revealed poor filling and poor contraction, perhaps explaining the altered intra-cardiac sound generation (relating to chamber volume and to valve closure and compression velocities).

Comparative changes in ALT values were used to classify dogs into mild, moderate and severe 'shock' cases, induced by oral DEC dosages in dogs with (*D. immitis*) microfilariae. While the exact shock mechanism (of the leukotriene prostaglandin-interplay) was never well established, these percentage ALT changes were an objective indication of a 'shocked' liver (i.e. dog specific, most shocked organ).

In people (also see Anaphylaxis – A. Litster, PhD; Uni Qld) mast cell elastase can be used to verify a truly anaphylactic reaction (i.e. acute profound mast cell release of multiple species-specific mediators – elastase being of diagnostic use (but the test is time-limited, to 6-8 hours, after the signs of shock).

My son had such an acute Type I (?) reaction to a drug of the muscle relaxant family – the surgeon was not aware of the test, its availability or of the concept of species variability with regard to different organ targets and mediators. If the test is available it may be more useful as usually there is no control ALT level to verify percentage elevation in dogs with variable normal (pre-shock) resting ALT levels. Such changes may also be time–release dependent, as they could be affected by the rate of arterial perfusion, the degree of hepatic vein constriction and altered (liver) venous return. Skin testing (subsequent to recovery) is the standard verification test to identify reactivity to defined chemicals (as for muscle relaxants) in people, who can then wear a drug susceptibility wrist band.

Reference

[1]Sutton RH, Atwell RB, Boreham PF. 1985. Liver changes, following diethylcarbamazine administration, in microfilaremic dogs infected with *Dirofilaria immitis*. 1985. *Vet Pathol*. Mar;22(2):177-83.

Reply to C&T No. 5273 Chronic cystitis driving me crazy (Dec 2012, Issue 269)

C&T No. 5319

Invited Comment courtesy of:

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1 2 Rollover or Download to read C&T No. 5273

Refractory *Proteus mirabilis* urinary tract infection (UTI) in a CKCS

'Rosie' certainly represents a frustrating UTI case and a large amount of clinical work has been put into gaining a resolution. For all intents and purposes, Rosie appeared to present initially with a **simple uncomplicated UTI** (otherwise healthy dog) but time revealed it to be difficult to cure.

Whilst not a common cause (<10% of cases) of recurrent or persistent bacterial cystitis,[1,2] *Proteus spp* can certainly prove difficult to eradicate in some patients. In Rosie's case, the infection most likely falls into the category of **relapsing** UTI because sterility of the urine was documented on one occasion (and was possibly achieved at other times) but an intriguing possibility is that of **refractory** deep bladder wall infection not reflected by that single negative urine culture.

So why did 'Rosie' fail to respond to apparently reasonable therapies when many other dogs would have been cured?

An answer certainly calls for **speculation** but some points to consider are: ▶

Authors' views are not necessarily those of the CVE

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- It appears that Rosie was not as 'normal' as she initially seemed i.e. an underlying predisposing condition was likely present. The owners later confessed to long-term (>4 months) dosing with low-dose (0.04 mg/kg PO SID) prednisolone. Whilst we can't be sure that this predisposed Rosie to relapsing bacterial cystitis, an increased incidence asymptomatic UTI was found in dogs with pruritus treated with similarly low doses of prednisone long-term (>6 months).[3] In addition, Rosie's body condition score was not mentioned but 14 kg seems excessive for a CKCS. Perivulvar dermatitis and vulval recession can predispose to recurrent urinary tract infection[4] and obesity may contribute to these conditions. Whilst hyperadrenocorticism is more common in older dogs, it can certainly predispose to urinary tract infection[5] and the diagnostic investigation for hyperadrenocorticism was excellent in this case. Additionally, abnormalities of Rosie's urinary tract cannot be ruled out and further studies such as the suggested contrast studies and/or cystoscopy, which would allow visualisation of the urinary tract, including polyps, neoplasia, uroliths, and collection of samples for histopathology and culture, could also facilitate greater understanding of Rosie's predisposition to bacterial cystitis. Identifying underlying conditions and correcting them is the mainstay of management of refractory UTI.
- Amoxicillin-clavulanate (AMXC) is a time-dependent antibiotic so proportion of time (> 50% of the dosing interval is recommended) that the urine concentration of antibiotic exceeds the minimum inhibitory concentration (MIC) is the most important parameter to consider when planning a treatment regimen.[6] Anecdotal success in recurrent or persistent UTI cases and indeed the current recommendation by ISCAID[7] dictate that AMXC be administered 3 times daily (12.5-25 mg/kg PO q8h) for treatment of bacterial cystitis (we have found the high end of that range to be successful for treatment of multidrug-resistant *Escherichia coli* cystitis in dogs although vomiting can be an issue at doses >20 mg/kg TID). Advantage over amoxicillin (AMX) has not been established and in a case such as this in which the isolate is sensitive, AMX could have been dosed at 11-15 mg/kg PO q8h initially, proving more cost-effective. In light of the fact that the isolate was sensitive, it possible that in Rosie's case, use of AMX or AMXC on a TID dosing schedule would have been more effective in eradicating the infection.
- Choosing AMXC to treat the *P. mirabilis* infection in Rosie for a third course was rational based on the sensitivity data to hand, and indeed sterile urine had been achieved at least once previously, but given the history of relapsing infection perhaps another antimicrobial could have been chosen? One possibility for persistence is the presence of deep bladder wall infection, supported by the subjectively thickened bladder wall noted on several occasions (biopsy and culture of the bladder wall would be required to confirm). Much is known about the ability of *E. coli* to invade the bladder wall but it appears likely that *Proteus* spp. may possess similar capabilities and both species have been reported to cause a rare histiocytic and difficult-to-cure bladder inflammation known as malakoplakia.[8] The bladder wall status when the urine was determined to be sterile following 2 courses of AMXC was not reported, but even so, bladder ultrasound is an insensitive way to predict bladder wall infection. Additionally, although AMXC is known to achieve good concentrations in the urine of animals with normal renal function, it may not be achieving sufficient concentrations in the tissue of Rosie's bladder wall to eradicate the bacteria, or even longer treatment duration could be necessary.[6] It is possible that treatment with an antimicrobial with superior tissue penetration e.g. a fluoroquinolone such as enrofloxacin, at a high dose (10-20 mg/kg SID for 4 weeks), would achieve a cure.
- A further potential complicating factor in Rosie's case is that the bacteria may be producing a biofilm, providing additional protection from antimicrobial attack and making a cure more elusive.[9]
- Chronic low-dose antimicrobial therapy in 'Rosie' could impact resistance development, particularly as urine sterility may not have been achieved prior to commencement and persistent bladder wall infection remains a possibility.

Tips for monitoring dogs with relapsing or refractory infection- from ISCAID guidelines[7]

http://www.iscaid.org/assets/content//documents/ISCAID_Urinary_guidelines.pdf.

A diagnosis of recurrent UTI should never be based on clinical signs or urine sediment examination alone. Bacterial culture and susceptibility testing should be performed in all instances to confirm recurrent UTI.

- Culture urine (cystocentesis sample) **5-7 days after commencement** of therapy. Bacterial growth indicates potential treatment failure and should prompt immediate re-evaluation. Referral or consultation with a specialist at this point is recommended.
- Urine culture **7 days** (3 weeks for cefovecin) **after therapy** is completed is also required and if positive, then in-depth investigation of predisposing factors for relapse or reinfection should be performed. Unless there is clear evidence for the reason for failure, retreatment without any other investigation is not recommended.
- If there is a **lack of clinical response to treatment or if clinical signs of UTI recur after apparently successful treatment** then the animal should be managed again as above, with particular emphasis on determination of underlying causes. Referral is strongly recommended.

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Reply to Treatment of penile lesions in bulls

C&T No. 5282 (Mar 2013 Issue 270)

C&T No. 5320

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Rollover or Download to read C&T No. 5282

I must applaud Heather and the photographer, Tracey, for the fantastic photos of the treatment of penile and or preputial injuries in bulls. In the past I have had similar lesions resolve with slings as long as the owner is prepared to put in the work. I have done several on my own and am aware of the time that is needed for treatment.However as I have no current cases, all I can offer is a very basic drawing (however I did get a better artist than me to help so that the image wouldn`t look too laughable.)

My main comment however is that this bull(s) must have been extraordinarily co-operative as all my patients manage to either rotate the sling or have it slip forward or back while moving around out in the paddock.

So I used to make the sling of a complete wrap around piece of hessian; ideally this was 2 chaff bags sewn together loosely with hay baling twine and a bag sewing needle (or in an emergency your prolapse needle). These days it is very difficult to get hessian chaff bags as they are nearly all the woven plastic or poly material which would be abrasive and totally unsuitable. I have seen some produce stores still do stock hessian products so it shouldn`t be impossible to find.

Then to stop it slipping back I tie twine around the NECK with a non-slip knot to keep it stable. I tie the sling to this with 3 separate twines, 1 along the back and 2 along either shoulder. The ties can be looped into the sling with a slip knot to allow the sling to be removed easily.

Then a BRITCHING string is tied around the back end slightly above the stifle level to stop the sling moving forward.

The bull can urinate through the bottom of the sling so I always advise to line the bottom with fairly soft material like a piece of old sheet.

I also advise the owner to make **two slings** so one can be removed daily, the lesions can be cleaned and a fresh sling can be put on while the old one is hosed and hung on the fence to dry.

Other Things I Would Do:-

1. As above I would treat and clean **daily**. I found if the bull was always fed hay or some grain mix in the crush during treatment it was easier to keep getting him in.
2. The prednoderm would probably be fine although I used to use a non greasy udder cream like Hibitane or the old Ceatro (neither of which I have seen around for some time). I just felt they would attract less dirt onto the lesions.
3. I used to always clip the **preputial hairs** away to try and reduce some contamination but also to try and lessen the chance of the hairs catching on prolapsed tissue when it started to retract into the prepuce.
4. I would have probably given about 3 doses of long acting penicillin 48 hours apart e.g. 50 mLs Benacillin i/m, but am totally in agreement in using tolfedine or similar anti-inflammatory at the start.
5. I would have preferred to keep slinging until the prepuce had healed entirely, as in Figure 3 of the article I feel there is risk of re-injury.

Prevention of adhesions after prolapse resolves

Sometimes the penis can retract up into the prepuce while it is still quite indurated.

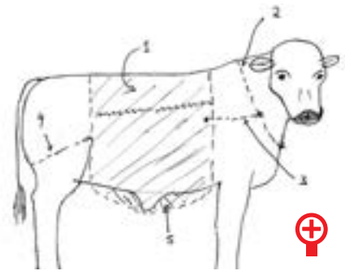
I used to get the farmer to squirt in a steroid ointment like Mastalone but this was always quite expensive and probably went nowhere in an area as large as the prepuce.

I eventually worked out to use a soft hose or old stomach tube with a stirrup pump. Fill the prepuce with water then hold off the tip of prepuce with your fingers then with the other hand massage the prepuce and slosh all the liquid around. I would repeat 3 or 4 times and keep treating daily until I was sure no adhesions were forming.

A couple of times I sedated the bull to examine the penis before stopping treatment.

Key to picture is:-

1. The sling ideally made of 2 chaff bags sewn together then loosely tied together so it can be removed easily.
2. The neck twine tied with a reef or other non slip knot.
3. One of 3 ties going back from the neck rope to the sling.
4. The britching tie attached to the sling on both sides.
5. Where the small piece of old sheet is placed in the sling to try and stop the damaged tissue being abraded on the hessian.



Answer to What's YOUR Diagnosis?

C&T No. 5290
(Mar 2013 Issue 270)

C&T No. 5321

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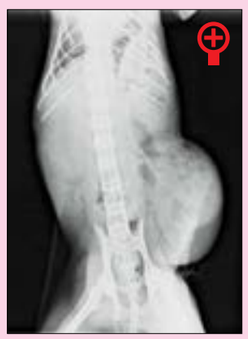

The radiographs show a stomach that has herniated through the left? side abdominal wall, and a diaphragmatic hernia.

***Note:** Marilyn is the Winner and entitled to a CVE proceedings of her choice. www.vetbookshop.com*

Authors' views are not necessarily those of the CVE

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How to best monitor diabetes in cats

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Monitoring clinical signs and response to therapy

Establishing a practical routine for the cat's owner

Many owners of diabetic cats welcome the opportunity to monitor their pet's response to therapy, although compliance can be very variable. Compliance is markedly improved if there is close rapport between the owner and the clinician managing the case and appropriate individualisation of the cat's therapeutic and monitoring regimen. The veterinary clinician must invest time to educate the owner about feline diabetes and its management, as well as to provide support and guidance while the owner becomes accustomed to the treatment and monitoring procedures and establishes a practical routine.

The primary aims of therapy in diabetic cats are to achieve resolution of clinical signs and to optimise the chance of diabetic remission. Diabetic remission is most likely when the blood glucose concentration is kept below the renal threshold. Higher blood glucose concentrations cause 'glucose toxicity' which results in dysfunction of any remaining beta cells. This effect can be reversible and so there is often recovery of beta cell function once a diabetic cat achieves persistent negative glucosuria. **Decreased insulin requirement is typically seen 1-2 weeks after the onset of negative glucosuria and it is important to then reduce the exogenous insulin dose to avoid hypoglycaemia.**

It is very helpful if owners can test urine glucose daily in addition to regularly monitoring signs such as the volume of water drunk and body weight. Most cats will readily cooperate with urine glucose testing. Cats that use a litter tray will often urinate immediately if their owner changes the litter while the cat is in the room. Cats that do not use a litter tray will typically allow urine sampling if their owner accompanies them when they are let out into the garden in the morning. A dipstick can be applied to urine-soaked litter or soil while it is still wet. If the urine patch has dried out, tap water can be added to allow application of a dipstick. This will dilute the sample but still allow determination of positive versus negative urine glucose. Some owners prefer to use a litter tray with a sieve-like bottom that allows urine to collect in a lower tray. **It is necessary to counsel owners that no importance can be attributed to the amount of glucose recorded on the dipstick – that is, there is no important difference between 1+ and 4+ results; it is simply a question of positive or negative glucose. Persistent negative glucosuria identifies periods when the exogenous insulin dose should be decreased.**

Monitoring daily water intake provides a very useful guide to the current level of glycaemic control in diabetic cats. Average blood glucose concentration correlates positively with 24 hour water intake. It is helpful for the veterinarian to educate their clients on the 'normal' water intake of cats because most owners are not familiar this. Normal young adult cats eating a 100% diet of dry cat food drink 80 to 150 mL daily (20 to 30 mL/kg/day), while they drink little or nothing when eating a diet of canned food. **If a diabetic cat drinks more than 40 mL/kg/day or is lethargic or losing weight, then adjustment of the insulin dose is probably required.**

Owners of diabetic cats should be encouraged to keep detailed records of their cat's progress.

1. Appetite, general demeanour and behaviour. This should be recorded every day.
2. Meal amount and composition should also be recorded every day.
3. Insulin dose. This should be recorded twice each day.
4. Water intake. This should ideally be measured every day when the cat is receiving insulin and once a week when the cat does not require insulin. The key is to determine how many millilitres of water the cat drinks over a known number of hours. If the water bowl is shared with one or more other cats, then the volume drunk by all cats should be measured. Owners will need a measuring jug with 10 mL increments.
5. Urine glucose and ketones. This ideally requires collection of a sample of urine every day. This can be either a liquid sample or some wet kitty litter or soil that has been moistened using tap water.
6. Body weight. Ideally, a diabetic cat's body weight should be recorded once each week. It is important to use the same scales each time the cat is weighed. Scales designed for weighing adult humans are not suitable for cats. Scales designed for babies are a much better option.

In addition to appraisal of the owner's insulin dosing technique, compliance of both the owner and the patient with the feeding recommendations must be routinely evaluated. Appropriate nutritional strategies can complement insulin therapy in diabetic cats and so it is often beneficial to make changes to the diet or feeding regimen. The lowest carbohydrate foods are the best for diabetic cats. **Most canned or wet cat foods have a very low carbohydrate content.** The only dry cat foods that are permitted are the ones specifically formulated for diabetic cats, such as the Hills m/d® dry food or the Royal Canin Diabetes dry food. **Meals may be consumed at any time and do not need to be matched with insulin injections.**

Measurement of long-term glycaemia: fructosamine

Measurement of fructosamine is an additional way of assessing glycaemic control in diabetic cats, although **monitoring clinical signs is usually sufficient.** Plasma fructosamine provides ▶

an approximate measure of average blood glucose concentration over the preceding 2-4 weeks and thus is an indicator of longer-term diabetic control. **Measurement of fructosamine is most useful when there is little available information about recent clinical signs of the diabetic cat**, or when results of serial blood glucose measurements do not match with the reported clinical signs.

Comparison of serial measurements of fructosamine in an individual diabetic cat allows evaluation of glycaemic response to management changes. A major limitation with these measures is that they represent average glycaemia and give no information about the degree of fluctuation around that average. Therefore they do not indicate the risk of hypoglycaemia on the current insulin regimen.

Monitoring blood glucose concentrations at home

Some owners are interested in performing blood glucose monitoring at home, particularly those who desire more autonomous control over their cat's diabetes. Owners who choose this method of monitoring sometimes need to be advised against over-zealous blood glucose measurement and interpreting the results themselves. Other owners are unwilling to add home blood glucose monitoring because they feel it is an added source of stress. **The majority of uncomplicated diabetic cats can be well managed if owners closely monitor clinical signs and urine glucose and do not require additional blood glucose testing.** Complex cases however benefit greatly by the introduction of home blood glucose concentration measurements to the monitoring regimen.

Samples can be obtained either from the marginal vein of the lateral pinna or by collection of capillary blood from the medial pinna. Application of petroleum gel prior to blood sampling facilitates beading of blood on the skin surface and thus allows sampling using smaller volumes.

Owners should be encouraged to purchase a **veterinary blood glucose meter** because human glucose meters give significantly lower results in cats when compared with both veterinary meters and laboratory reference methods. Veterinary glucose meters currently available in Australia are the **AlphaTRAK meter** (VetQuip Pty Limited) and the **g-Pet meter** (The Vetservice Group in New Zealand).

There is considerable day-to-day variability in blood glucose measurements in diabetic cats. S**ingle, sporadic measurements provide little useful clinical information for monitoring glycaemic control.** The major advantages of home-monitoring of blood glucose concentration are that measurements can be easily obtained at any time and can be repeated if equivocal results are obtained, the cost is minimal compared with a veterinary visit, and the effects of hospitalisation on appetite and stress hyperglycaemia are avoided. **Serial blood glucose concentration curves that follow the same protocol as those obtained in hospital can be performed at home.** Results must always be related to the cat's clinical signs; interpretation requires an understanding of the complex interactions involved in glucose homeostasis in diabetic cats.

Serial blood glucose concentration curves are most useful in cases where the clinical history is poor. However, it is important to recognise that they are an unreliable clinical tool for evaluation of insulin dose in diabetic cats because there is a large amount of day-to-day variability in results. It is advisable to always consider additional indicators of glycaemic control, such as changes in the cat's water intake, body weight, and urine glucose concentrations, when appraising insulin dose.

A practical approach is to use knowledge of the cat's clinical signs to guide the timing of home-generated blood glucose curves. For example, if there is marked variability of 24 hour water intake and/or urine glucose results, owners can be advised to perform a glucose curve on a day when the cat has negative glucosuria or does not drink much water. This approach would increase the chance of detecting hypoglycaemia. **If clinical signs consistent with hypoglycaemia occur at home, owners accustomed to measuring their cat's blood glucose concentration can quickly confirm whether or not hypoglycaemia is present and so facilitate timely treatment.**

Continuous interstitial glucose concentration monitoring systems

Continuous interstitial glucose concentration monitoring systems such as the Guardian REAL-Time Continuous Glucose Monitoring System (Medtronic) can also be used in the home environment or in hospitalised patients to monitor glycaemia. **Important advantages of continuous monitoring systems over intermittent measurement of blood glucose are that they facilitate detection of brief periods of hypoglycaemia and provide information overnight or when the owners are not at home.** One limitation is that they must be calibrated with blood glucose concentration, so there is still a requirement for some blood sampling during monitoring.

Problem-solving difficult cases

Difficulty monitoring glycaemic response – results do not match the cat's clinical signs

Glucose homeostasis in cats is a dynamic process that can change very rapidly. Cats with diabetes have dysfunctional glucose homeostasis and so there is even more pronounced variability of blood glucose concentrations, especially in response to stress or illness. **A common error when managing feline diabetes is to assume that the condition can be 'stabilised' and that measurements taken on one day are representative of diabetic control on other days.** A more appropriate approach is to establish an ongoing monitoring regimen that will detect the changing trends in the cat's response to treatment. Improved activity, resolution of polydipsia and polyuria, and weight gain are all indicators of improved glycaemic control. It is important to realise that **negative glucosuria can occur at any time once clinical signs have resolved, and may occur within days, weeks, or even months of the last insulin dose adjustment.** Once the blood glucose concentration is consistently below the renal threshold, the cat's insulin requirement will usually decrease substantially. However, **the rate of diabetic remission appears to be optimised if the insulin treatment is withdrawn gradually, with a dose decrease every 1-2 weeks.** In contrast, sudden cessation of insulin can result in recurrence of hyperglycaemia, return of glucose toxicity, and increased requirement for exogenous insulin.

Micro-management of feline diabetes with frequent insulin dose adjustment should be avoided. In general, insulin dosage adjustments should not be made any more frequently than once every 1-2 weeks. The exception is following an episode of hypoglycaemia, which should always prompt reduction of the insulin dose (unless there was a dosing error). When presented with a cat that has shown a very variable response to insulin and inconsistent glycaemic results, it is often helpful to carefully review the history with the owner to determine if there were any periods when there was resolution of clinical signs. It is important to also note the insulin dose administered before any occurrences of hypoglycaemia. **A reasonable approach is to resume the treatment regimen that the** ▶

cat was receiving when there was good glycaemic control and to carefully monitor the cat's response for a minimum of 2 weeks. This information can then provide baseline data for an ongoing regimen of monitoring and treatment adjustment.

Unexpected hypoglycaemia

Daily recording of urine glucose provides a useful 'early warning system' for the risk of clinical hypoglycaemia for most diabetic cats. Decreasing the insulin dose within 2 weeks of the onset of negative glucosuria will thus ensure that hypoglycaemia is avoided in the majority.

The owner's insulin administration protocol should be carefully evaluated whenever hypoglycaemia occurs to identify dosing errors. It is strongly recommended that insulin injections are administered at strict 12-hour intervals. Irregular timing of insulin injections can lead to overlap of insulin action with that of the previous dose. If it is not possible to administer an insulin injection on time, then the best approach is to miss that injection and resume insulin administration at the next injection time. Missing a single injection will usually have negligible consequences. In contrast, late administration of insulin can lead to increased insulin action (and therefore over-dose) if the following insulin injection is administered on time. The usual meals can be fed whenever an insulin injection is missed.

For cats that are prone to hypoglycaemia, **longer-acting insulin preparations such as glargine insulin are recommended** as these will likely have a smoother action and so minimise periods of both hyper- and hypo-glycaemia.

Feline diabetes that is complicated by concurrent disease and/or medications that are causing insulin resistance will typically present with very unpredictable blood glucose results that vary anywhere from 1.5 mmol/L to 30 mmol/L despite consistent insulin dosing. Such cases will often roughly follow a 3-day cycle fluctuating between hyper- and hypo-glycaemia and might present with intermittent hypoglycaemic seizures. Measurement of blood glucose concentration by owners immediately prior to each insulin injection can greatly help to guide insulin dosing in these cases. An example of dosing recommendations that were developed for an individual diabetic cat that has chronic pancreatitis/triaditis and requires prednisolone and chlorambucil therapy is as follows. Note that these recommendations are based on blood glucose measurements using a veterinary glucose meter.

- Measure blood glucose prior to each insulin injection:
- If blood glucose is 15.0 mmol/L or greater, give 5 units
- If blood glucose 10.0-14.9 mmol/L, give 4 units
- If blood glucose is 6.0-9.9 mmol/L, give 3 units
- If blood glucose is 3.8-5.9 mmol/L, give 2 units
- If blood glucose is 3.7 mmol/L or less, give no insulin.

Such an approach provides owners with a practical tool to avoid clinical hypoglycaemia and achieves reasonably good control of the clinical signs of hyperglycaemia in cases where diabetes has proved very difficult to manage. If owners of such complicated diabetic cats are unwilling to perform blood glucose testing at home, then the safest approach is to recommend consistent treatment with an insulin dose lower than that which has previously caused clinical hypoglycaemia and to closely monitor clinical signs and urine glucose.

It is important to note that **feeding in cats will not typically cause a postprandial increase in blood glucose concentration and so is not a reliable treatment for hypoglycaemia.** Low carbohydrate foods such as those typically recommended for diabetic cats will not be helpful in this situation. However, liberal application of glucose syrup or honey to the oral mucosa will usually effectively control clinical signs although repeated dosing might be required. The glucose syrup or honey can also be administered per rectum, which is usually the safest option in a cat with severe seizures. Once the cat has recovered and is able to eat, glucose syrup or honey can be added to cat food. Cats cannot taste sweet foods and appear not to notice the presence of honey if it is mixed thoroughly with the food. **If oral glucose is not successful, the hypoglycaemia should be managed initially with an intravenous bolus of 50% dextrose at a dosage of 1 mL/kg (0.5 g/kg) administered slowly.** This can be followed with a constant rate infusion of 2.5% dextrose solution at 6 mL/kg/hr with the flow rate adjusted based on subsequent blood glucose concentrations.

High insulin requirement (>5 units/cat) or an unexpected increase in insulin requirement

Most diabetic cats require doses of 5 units/kg or less of glargine insulin administered every 12 hours. Occasionally an individual cat will need 6 or 7 units before good glycaemic control is achieved. Insulin doses should always be based on estimated ideal body weight rather than actual body weight in underweight or overweight cats. **Once persistent negative glucosuria is achieved and insulin requirement decreases, many cats will go into remission and the remainder will typically require 3 units or less of insulin every 12 hours.**

An unexpected increase in insulin requirement is recognised when a cat that has been in long-term diabetic remission needs insulin therapy again, or when a cat that has needed only a low insulin dose for months unexpectedly requires higher doses.

The major differential diagnoses for a diabetic cat to require more than 7 units of insulin every 12 hours or for an unexpected increase in insulin requirement are:-

- **Error in insulin handling or administration**
- **Obesity**
- **Concurrent disease or drug therapy**
- **Compensatory hyperglycaemia secondary to insulin overdose (Somogyi phenomenon).**

Error in insulin handling or administration

Insulin can become inactivated if exposed to temperatures >30°C or light for prolonged periods. **An expedient method of ruling out the possibility of inactivated insulin when investigating insulin resistance is to change to a new vial of insulin.** Insulin suspensions must be thoroughly mixed prior to administration or doses might vary greatly.

Although experienced owners of diabetic cats rarely report difficulty with administration of insulin to their pet, it is important **to review their injection technique for errors whenever insulin resistance is investigated.** A wide range of insulin syringes are available and inadvertently changing to a different type of syringe can lead to dosing errors. **Dosing errors are less frequent with insulin dosing pens than with needles and syringes.** However, **insulin dosing pens must be primed prior to administration of each dose** to ensure there is no air in the system. It is also important to check that the dosing dial has returned to the 'zero' position after each dose.

Obesity

Obesity is a common reason for a diabetic cat to have increased insulin requirement. Reversible insulin resistance is induced and **if weight loss occurs insulin requirement can rapidly return to normal and care must be taken to avoid hypoglycaemia.**

Weight gain is initially a desirable outcome when treating diabetic cats as it is an indicator of good glycaemic control. However, once this is achieved, overweight and obese-prone diabetic cats should be carefully managed to attain and maintain an ideal body condition. **Both body weight and body condition should be monitored long-term, and calorie restriction will be necessary in some cats.**

Concurrent disease or drug therapy

Every diabetic cat that is not overweight and has increased insulin requirement should be assessed for concurrent disease. Any medical condition can theoretically cause insulin resistance and urinary tract infection, hyperthyroidism, and chronic renal failure are commonly recognised. Acromegaly should be considered if all other diseases are ruled out. **The classic textbook signs of acromegaly are not always present in affected diabetic cats, with some showing no sign other than high insulin requirement.** Diagnosis is typically based on increased serum IGF concentration, although the laboratory that was running this assay in Australia has recently stopped offering it. To optimise the chance of detecting increased IGF concentration, cats must first be treated with insulin for at least 4-6 weeks. This is because portal insulin induces IGF production and acromegalic cats can give normal IGF results when they are insulin deficient. It has been suggested that the incidence of acromegaly in diabetic cats might be underestimated, although this has not been confirmed in Australian cats. Anecdotal information suggests that the prevalence in Australian diabetic cats is likely not more than 10-15%.

Compensatory hyperglycaemia secondary to insulin overdose (Somogyi phenomenon)

Compensatory hyperglycaemia secondary to insulin overdose (Somogyi phenomenon) can cause insulin resistance in diabetic cats. There is typically a period of good glycaemic control that is followed by deteriorating glycaemic control despite increasing insulin doses. The period of good glycaemic control may be very brief and is sometimes missed, especially if dose adjustment is based only on results of blood glucose or fructosamine concentration testing without careful consideration of the cat's clinical signs.

In insulin-treated diabetic people, hypoglycaemic events occur more frequently during the night than during the day and the same might be true for cats. Therefore insulin-induced hypoglycaemia can be missed with day-time monitoring. Continuous subcutaneous glucose monitors are a useful diagnostic aid for detecting night time hypoglycaemia. **Compensatory hyperglycaemia often appears to persist for several days following insulin-induced hypoglycaemia.**

For diabetic cats with poor glycaemic control that are receiving insulin doses greater than 1.5 units/kg where administration/dosing errors and concurrent disease/drugs have been ruled out, it is recommended that the insulin dose be decreased to 0.5 unit/kg and the response to this change monitored. If insulin resistance was due to compensatory hyperglycaemia secondary to insulin overdose, there is typically marked clinical improvement within 1-2 weeks. If there is another cause of insulin resistance, clinical signs typically become much worse within a few days and the previous insulin dose can be resumed and the investigation for another cause continued.

Perspective 97

Management of an extensive necrotic wound using various hydrocolloids and a mesh graft

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My co-author Shelagh Lamb is not a vet but a human nurse who works as a sales rep for Hollister Australia and who also happens to be the owner's sister. Shelagh's contribution was massive, both in accessing donated materials and guiding us in their use. In addition to Hollister's products, Shelagh found alternatives from other companies where appropriate. I included Shelagh as co-author as she directed me to a lot of research on hydrocolloid use in people and was present at almost all of the early bandage changes to guide me in application. Without her, I doubt we would have had the outcome we did.

Abstract

Moist wound healing and the use of hydrocolloids have become accepted practice in human medicine and are being increasingly employed (off label) in veterinary medicine. The large number of bandaging products available with differing compositions and properties makes learning when to use these products very confusing. The following article outlines the management of an extensive necrotic wound in a 5-year-old Whippet using various hydrocolloids and a mesh graft. The products used are described along with the practical benefits and difficulties encountered in their use. Throughout the course of this case the author was assisted by a nurse qualified in human wound care management.

As problems were encountered solutions were adapted from common practices in management of human patients with burns, stoma wounds and skin grafts. Although there are differences in the way humans and dogs heal, the guidance received from the human medical field was invaluable in resolving this difficult case.

Abbreviations: bpm beats per minute, breaths per minute
SC subcutaneous
IV intravenous
PO per os

Key words: hydrocolloids, necrotic wound, mesh graft, dog

Authors' views are not necessarily those of the CVE

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Case report

A 5-year-old male desexed Whippet was presented on Good Friday 2011, with sudden onset nonweightbearing lameness in his right hind leg. Clinical examination revealed moderate to marked swelling of the caudomedial thigh proximal to the stifle. The skin in this area was bruised in appearance. Temperature was 40.9℃, heart rate 140 bpm, respiratory rate 24 bpm. Regional lymph nodes were not enlarged. No other clinical abnormalities were detected. No cause of the bruising could be found. Supportive treatment with antibiotics (Amoxicillin / clavulanic acid 20mg/kg q 12hr,) and pain relief (Meloxicam 0.1mg/kg po q 24hr) was started.

Over the next 3 days the dog's condition progressively worsened with swelling of the leg extending to the distal limb. Large areas of the skin appeared necrotic. The limb was cold to touch and serous fluid ooze was apparent in some areas. Enrofloxacin 10mg/kg q 24hr was added to the medical regime. Multiple releasing incisions were placed along the limb to help relieve pressure and a compression bandage was applied. This helped reduce the swelling a little but did not prevent necrosis of the skin from the caudal thigh and medial stifle extending distally to the hock.

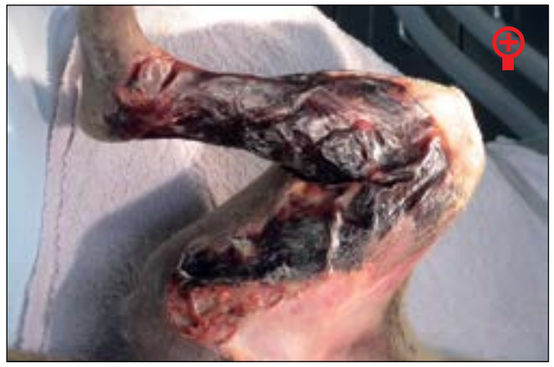


Figure 1. Necrotic skin caudomedial thigh and medial aspect of hindleg.

The decision was made to aggressively debride under general anaesthesia and manage the resulting wound using moist wound healing techniques. Moist wound healing creates optimal conditions for faster wound healing.[1,2] Wound fluid contains proteases, protease inhibitors, growth factors and cytokines in the appropriate physiologic ratios for each stage of healing.[1,2] By using a moisture retentive dressing to retain this fluid in the wound, cell proliferation and function are enhanced.[2]

Water loss through intact skin is 4 to 9 g/m^2/hr and increases to 80 to 90 g/m^2/hr in partial and full thickness wounds.[2] The occlusiveness of wound dressings is measured by moisture vapour transmission rate (MVTR)[2]. Dressings with a MVTR less than 35 g/m^2/hr are considered moisture retentive.[2] The closer the MVTR of the dressing is to the transepidermal water loss of intact skin, the greater the likelihood of a positive wound healing outcome.[2] Hydrocolloids have an average MVTR of 11.2 g/m^2/hr.[2]

Moisture retentive dressings have been shown to:[2]

- encourage white blood cells to remain in the wound where they perform selective autolytic debridement of necrotic tissues.
- Maintain physiological temperatures which support protease, growth factor and cell function
- Be more comfortable than non-occlusive dressings
- Prevent entry of urine and other fluids
- Allow longer intervals between bandage changes and faster healing
- Reduce scarring
- Reduce the incidence of infection by:
  – Providing a barrier to bacteria
  – Preventing tissue desiccation and necrosis
  – Increasing the viability of white blood cells and their enzymes
  – Maintaining a low oxygen tension which:
    » Deters bacterial growth
    » Attracts white blood cells
    » Favours collagen synthesis and angiogenesis

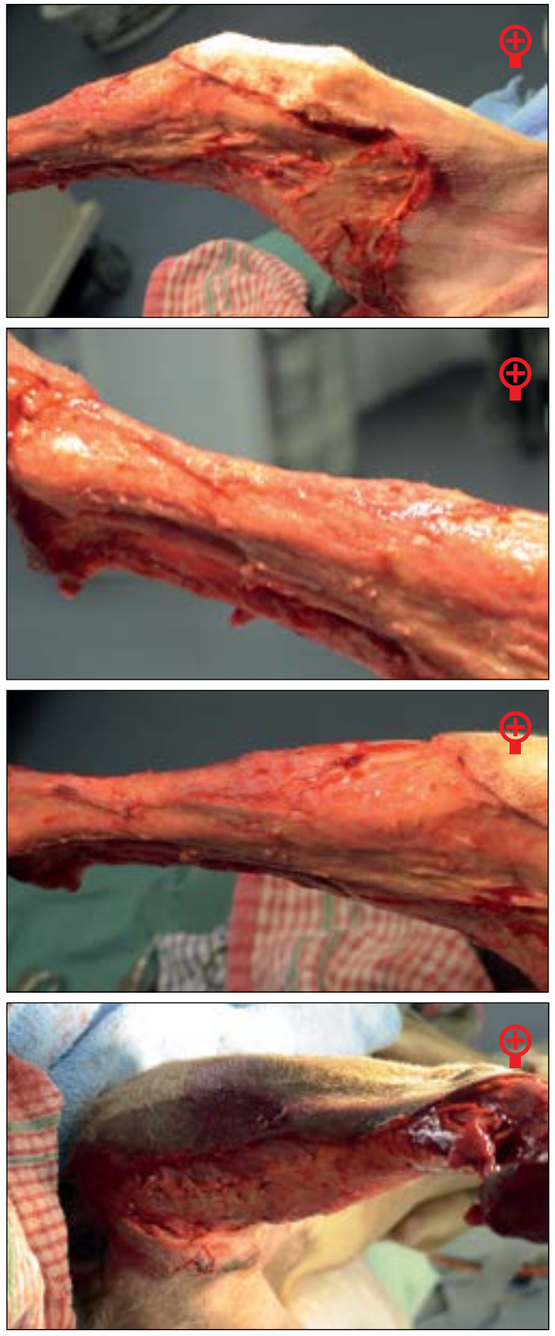


Figure 2 A-D. After debriding.

Histopathology

A biopsy was taken from 2 areas at the margin of normal and abnormal tissues during debriding. Histopathology results revealed a 'severe neutrophilic and fibrinous cellulitis with cutaneous infarction'. The pathologist's report read as follows:

'One of the sections is characterised by extensive necrosis involving the epidermis, dermis, adnexal structures and extending into the subcutaneous tissues. The subcutaneous tissues are expanded by large numbers of neutrophils that are variably degenerate together with abundant fibrin and oedema. A number of vascular structures in the adjacent soft tissues contain fibrin thrombi. The other section has a margin where there is a defect extending into the subcutaneous tissue. There are abundant neutrophils extending into the subcutaneous tissues and these are intermingled with fibrin and oedema. A number of vascular structures are occluded by fibrin thrombi and some appear to be organising. Streams of neutrophils within the expanded and oedematous connective tissue extend beneath intact epidermis but inflamed deep dermis. Obvious infectious agents are not seen with routine and special stains.'

After online consultation with other veterinarians through the Veterinary Information Network (www.vin.com) we began treatment with pentoxyfiline 15mg/kg q 12hr.

Pentoxyfilline is a xanthine derivative which enhances peripheral blood flow and tissue oxygenation. [6,7] Its mechanism of action is not fully understood but may involve relaxation of smooth muscles of the peripheral vessels causing vasodilation. pentoxyfilline also increases flexibility of the red blood cell and promotes platelet de-aggregation. These two effects contribute to a decrease in blood viscosity and improved movement of blood through peripheral blood vessels. In human medicine pentoxyfilline is primarily used for the treatment of chronic occlusive peripheral vascular disorders of the extremities.[8] More recently pentoxyfilline has been investigated for its potential use in aiding healing of difficult wounds. Some trial work has also shown that the use of pentoxyfilline may decrease pain associated with poor perfusion of wounds.[7,8,9] In dogs pentoxifylline has been used to enhance healing and improve microcirculation.[6] Other listed uses are vasculitis, vasculopathies and contact dermatitis.[10]

Meloxicam was discontinued when pentoxyfiline was started as there may be an increased risk of bleeding when used with NSAIDs.[10]

Wound Management

Day 1. (Day of debriding)

Immediately after debriding, Medihoney® was applied to keep the wound moist and a tie down bandage was applied using melolin® and veterinary gamgee®. This was held on with cotton tape through loops made with suture material attached to the surrounding skin. Honey has been used for many years for its bactericidal properties.[11] Its high osmolality reduces oedema and attracts macrophages.[11] Medihoney® can be messy to apply and bandages need to be changed at least daily or more frequently when it is used.[11] The author has observed it appears to cause pain when applied to conscious patients.

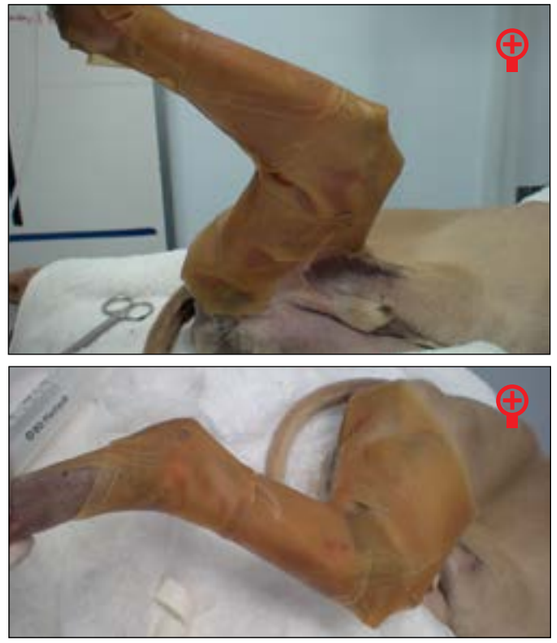
The logistics of ongoing treatment was discussed with the owner. Our plan was to manage the open wound until maximal contracture had occurred. Due to the size of the wound it was unlikely that contracture and epithelialisation alone would result in complete healing. A graft would be required to at least cover the area between the stifle and hock which had lost all skin. Options for a graft included a caudal epigastric flap and a free mesh graft. Pavletic indicated in his book that greyhounds and other thin skinned breeds (such as whippets) did not tend to do well with flap grafts due to increased risk of wound dehiscence and partial flap necrosis.[1] A mesh graft would require recruiting a large amount of skin from the lateral thorax, flank or neck of the whippet. This was not a dog with a lot of loose skin. A large donor site would mean significant tension in closing the donor site wound. If dehiscence occurred this would result in another open wound to manage. Our aim therefore was to reduce the size of the potential recipient bed as much as possible and achieve good granulation before considering a graft.

If we continued using Medihoney® as our moist wound dressing daily bandage changes would be required. This would be expensive and logistically difficult for the owner. Hydrocolloids were considered a better alternative as they allowed less frequent bandage changes.

Hydrocolloids consist of a mixture of absorbent and elastomeric polymers that interact with wound fluid to form a gel that maintains a moist wound environment.[2] They come in powder, paste or sheet forms.[2] The sheets are backed with a film that is impermeable to fluid, gas and bacteria creating an occlusive dressing when a good seal is maintained on the peri-wound skin.[2] The powder and paste forms fill irregular and deep wounds.[2]

Day 2. The tie down bandage was replaced 24 hours later using Medihoney®, again while we waited for hydrocolloid supplies to arrive. This style of bandage allowed free movement of the limb but was messy as excessive exudate and honey leaked through the secondary and tertiary layers very rapidly. The dog did not tolerate this well and continued use of this bandage type would have required 2 to 3 bandage changes per day.

Day 3. For the first application of hydrocolloid we used Restore Plus dressing (Hollister)®. This product did not adhere well to the dog's skin. To achieve the air tight seal we needed, overlapping layers were applied allowing the product to adhere to itself. The product was also a little too thick to be easily moulded to the shape of the leg. This may not have been a problem in a larger dog. Soffban® synthetic orthopaedic padding was used as a secondary layer to provide padding that would both help immobilise the limb and provide absorption for any excess exudate. Elasticon® was used as a tertiary layer and extended up over the hip in a spica splint type pattern to reduce slippage. This bandage was left in place for 5 days.



Figures 3A. Restore Plus - medial aspect & Figure 3B: Restore plus - lateral aspect.

Day 8. A good granulation bed was present with a visible gap between the deep and superficial digital flexor tendons. ▶

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The author was concerned that granulation may not occur across this gap so packed the area with Restore Calcicare Alginate (Hollister)®. Intrasite hydrogel (Smith and Nephew)® wound dressing was applied over this to hold it in place. Restore Plus (Hollister)® was then applied with the same secondary and tertiary layers as previously.

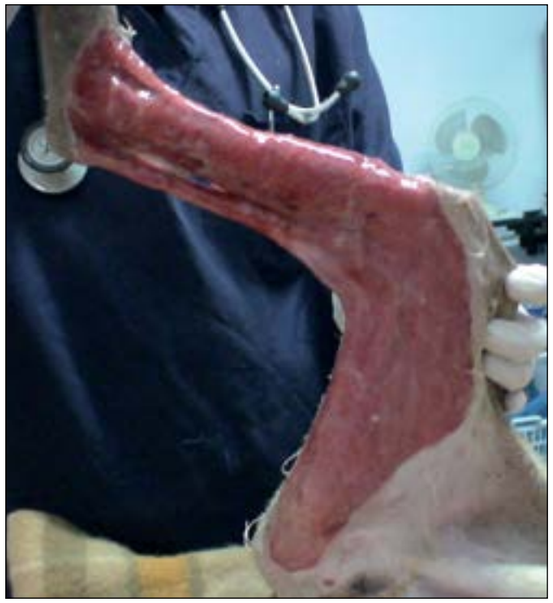


Figure 4. First bandage change.

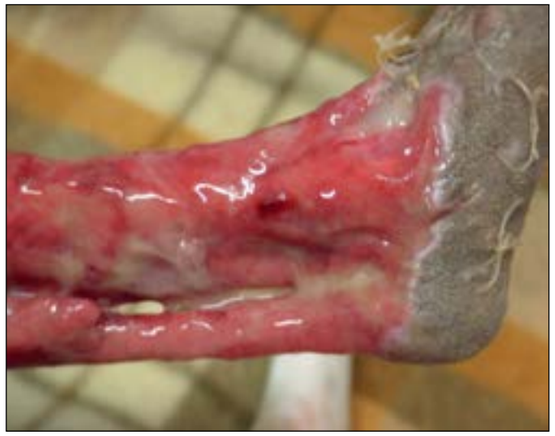


Figure 5. Hock region at first bandage change.

Day 12. Obvious contraction of the proximal end of the wound had occurred. The dog had been chewing at the top of the bandage resulting in removal of some of the hydrocolloid in this area. This had led to the proximal end of the wound drying out. In contrast the distal end of the wound was granulating well and had become very vascular. The alginate dressing was removed revealing that the area between the superficial and deep digital flexor tendons had closed.

As the hydrocolloid dressing absorbed exudate and the gel in contact with the wound liquified, it produced an odour which was intolerable to the owner. This odour was characteristic of many hydrocolloid dressings and not indicative of infection. In order to maintain compliance we changed to a Coloplast® product similar to the Restore® which had been found by the nurses advising us to have less odour associated with its use in humans. This product adhered to the peri-wound skin better than the Restore® had and was easier to mould to the shape of the leg but was still relatively thick.

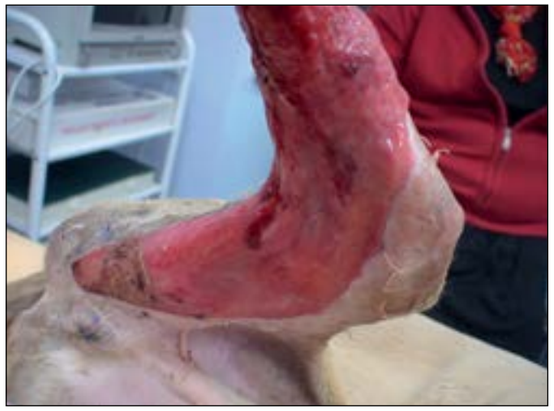


Figure 6. Proximal wound - contraction and desiccation.

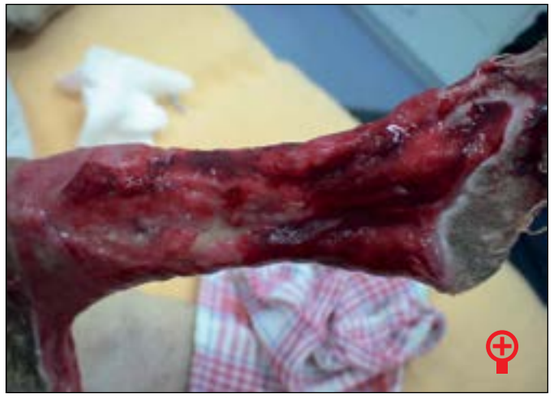


Figure 7. Hock showing active granulation.

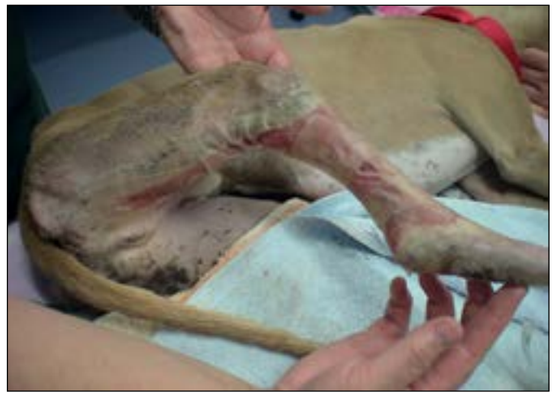
Day 16. The coloplast® appeared to have left a sticky residue on the peri-wound skin. The wound itself however was progressing well and an attempt at delayed closure of the proximal part of the wound was made. In retrospect this was probably a little premature as there was still some tension across the wound and the wound dehisced after only 4 days.

Day 20. The peri-wound skin was becoming inflamed where adhesive residue was left behind and excess exudate was leaking. To give the skin a rest we applied Adapt barrier seals (Hollister)® along the wound margin. This product is designed for use in human stoma patients as a protective barrier for the skin around a stoma wound. The barrier seals are a ring of mouldable material which can be cut and stretched to the desired shape. It adhered well to wet or dry skin and left minimal residue on the skin. Intrasite hydrogel (Smith and Nephew)® was used as our primary layer. For our secondary layer we used Profore #1 (Smith and Nephew)®. This is an absorbable orthopaedic padding that is easy to tear and appears to absorb exudate well. It has a slightly stiffer feel than Soffban® and was easy to conform to the shape of the leg with minimal slippage between bandage changes.

Coloplast® was replaced the following day. Adapt barrier seal® was placed around the wound margins again and Coloplast® adhered to this instead of the skin.

It was noticed that 24 hrs before each bandage change the dog would start chewing at the bandage. We decided this was an indication that we needed to increase the frequency of changes from every 4 days to every 3 days.

Day 27. This bandage change we started to use Comfeel Plus strips (Coloplast)®. These are thin transparent hydrocolloid sheets that conformed very well to the shape of the leg and adhered well to the skin. This product was the easiest of the hydrocolloids to use. After several bandage changes the dog seemed to develop a hypersensitivity to the product where it adhered to the periwound skin. By using the Adapt barrier seals® on the wound margin and applying the Comfeel Plus strips (Coloplast)® onto this we were able to minimise inflamation.



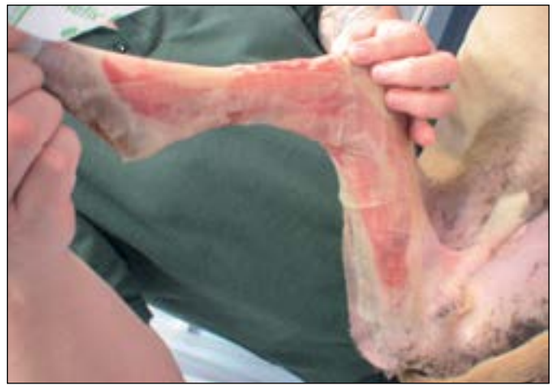
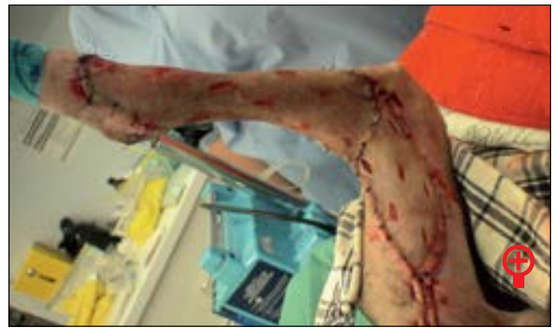


Figure 8A & B. Comfeel Plus® strips applied.

Day 36. By this stage we felt we had sufficient contracture to be able to perform a mesh graft. The donor site was planned by cutting a template in the shape of the wound and mapping this to the skin of the lateral thorax and neck in 2 pieces.

The recipient site was prepared 24 hours before planned surgery by cleaning and clipping the peri-wound skin to remove adhesive residue. The granulation bed was lightly flushed with sterile saline and the epithelialised margins were debrided. A thin layer of silver sulfadiazine ointment was applied and Atrauman (Hartmann)® dressing applied over this. The product information sheet states that Atrauman® is a fine weave hydrophobic dressing that has low adherent properties. Exudate is able to easily move through it but the fine weave prevents new tissue from growing into the weave thus reducing the risk of maceration. Atrauman® is also available in a silver impregnated form which could have been used here in place of the silver sulfadiazine cream. Conforming bandage was used to hold this in place, with Profore® and Elasticon® completing the bandage.

Day 37. The dog was premedicated with methadone 0.1mg/kg SC, midazolam 0.2mg/kg SC and glycopyrrolate 0.01mg/kg SC. Anaesthesia was induced with propofol 4mg/kg IV and maintained with isoflurane. Intravenous lactated ringers 10mL/kg/hr was administered throughout surgery. The graft was harvested and applied following procedures described by Pavletic.[12] The area between the stifle and the hock was addressed first. The graft for this area was harvested from the dorsolateral thorax. The donor site was kept moist using saline soaked laparotomy sponges while the mesh graft was prepared and applied. The proximal end of the caudal thigh wound was then closed with horizontal mattress sutures. The graft for the remaining portion of the wound affecting the caudal stifle was then harvested from the cranial margin of the previous donor site extending up the lateral neck. Once this had been prepared and applied the donor site was closed with simple continuous intradermal 3/0 Biosyn® sutures to hold tension and Simple interrupted 3/0 Premilene®. This closure left a small deficit which was covered with Mepilex Border® – a foam dressing with a polyurethane backing film that adhered well to the surrounding skin without adhering to the wound. It required no secondary layer and kept the small open wound moist and protected.



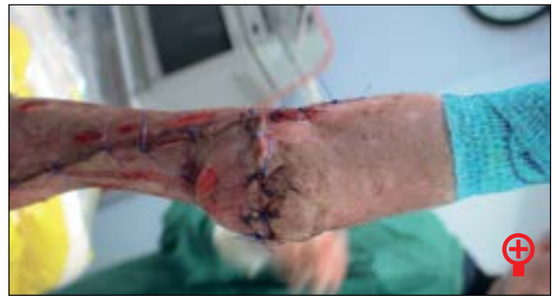


Figure 9. Mesh grafts applied.

Bandaging of the mesh graft needed to provide protection for, and prevent movement of, the graft and absorb exudates to avoid separation of the graft from the wound bed.[12] To achieve this we used Kendall AMD foam dressing (Covidien)® as a primary layer. This is a very soft polyurethane foam dressing impregnanted with polyhexamethylene biguanide, an antimicrobial agent effective against Gram negative and positive bacteria, fungi and yeasts.[13] Care was taken to prevent overlapping of the edges of the dressing so that there was no 'bunching' that may rub on the graft. The dressing was cut to shape and the edges sutured together to sit flat. To prevent movement, tacking sutures were placed to hold the dressing to the peri-wound skin. Conforming bandage was applied over this for extra support. To prevent movement of the stifle, multiple layers of Profore® were applied in a modified Robert Jones style bandage with the outer adhesive Elasticon® layer extending up over the hip.

Day 44. The bandage was left in place for 7 days in order to leave the graft undisturbed as long as possible while staying within the period of antimicrobial effectiveness of the Kendall™ AMD® dressing.[13] The dog was sedated with Medetomidine 0.01mg/kg IV and Butorphanol 0.1mg/kg IV to prevent movement during the bandage change. Care was taken to ensure the dressing had not adhered to the graft before lifting. ▶

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The margins of the graft were necrotic. The majority of the graft skin was a similar colour to the rest of the dog's skin suggesting good survival of the graft. The bandage was replaced using the same materials and left in place for another 5 days.

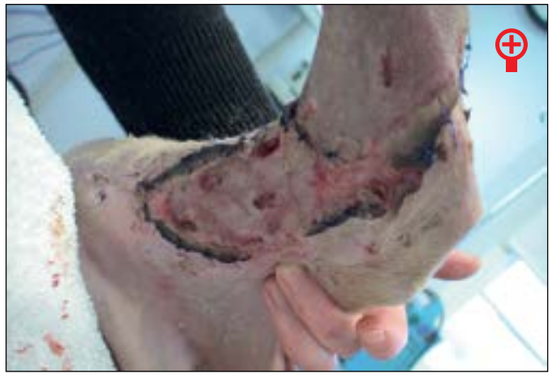
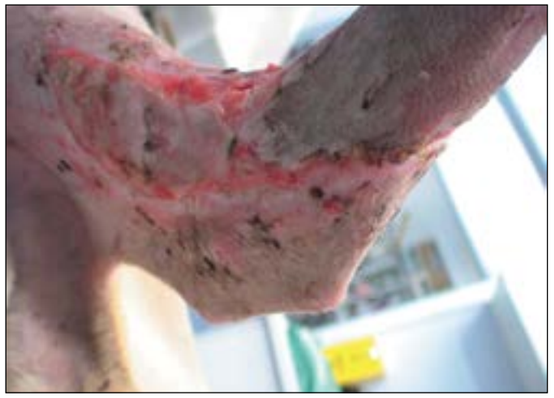


Figure 10A-C. 7 days post graft.

Day 49. *13 days post grafting, the sutures were removed.*

Figures 11A-B Overall graft 'take' appeared to be very good.



Figures 11A-C. 13 days post graft.

Mepilex border® was placed over the caudal stifle where some breakdown of the graft had occurred and there was still moderate exudation. Atrauman® was placed over the rest of the healing graft and conforming bandage used to hold this in place. A Relevo absorbent pad® was applied over this followed by Profore® and Elasticon®.

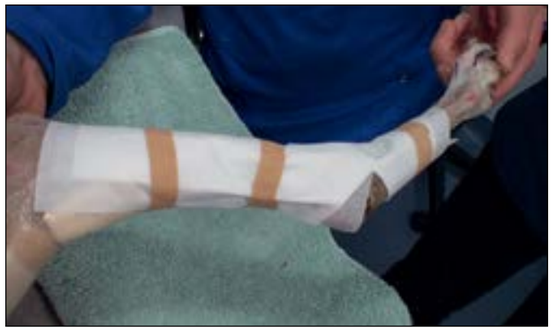


Figure 12. Mepilex Border and Relevo.

Day 51. 17 days post graft there was only a small wound remaining at the caudal stifle. This was covered with Mepilex border®. In this high movement area the Mepilex border® did not adhere as well as it had on the thorax. A layer of Elasticon® was applied to keep it in place. At this point we also gave the dog more freedom to move around.

Day 57. We encountered problems with breakdown of the caudal stifle wound and maceration of newly epithelialised areas at the proximal tibia. The caudal stifle area was heavily exuding and became infected. This was addressed initially by suturing KendallTM AMD® dressing over the area and restarting Amoxicillin/clavulanic acid 20mg/kg q 12hr. Exudate was absorbed and trapped well by the Kendall™ AMD® dressing.

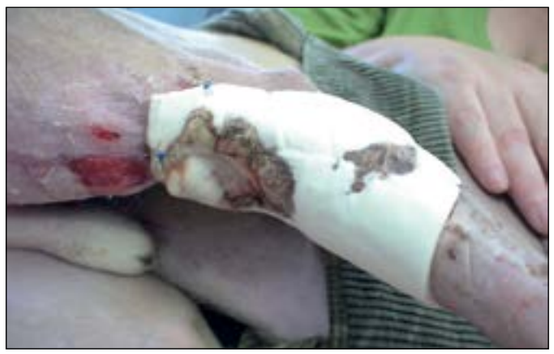
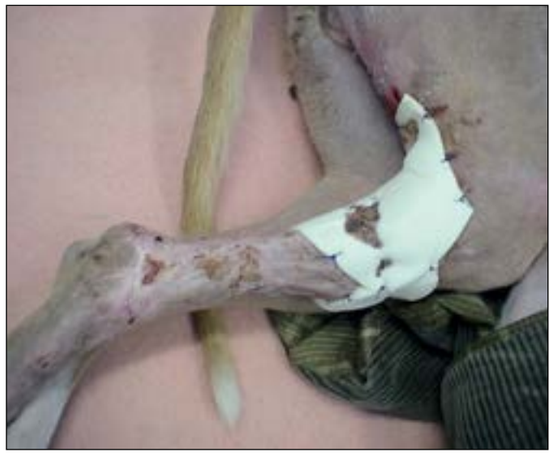


Figure 13A. Kendall AMD in use.



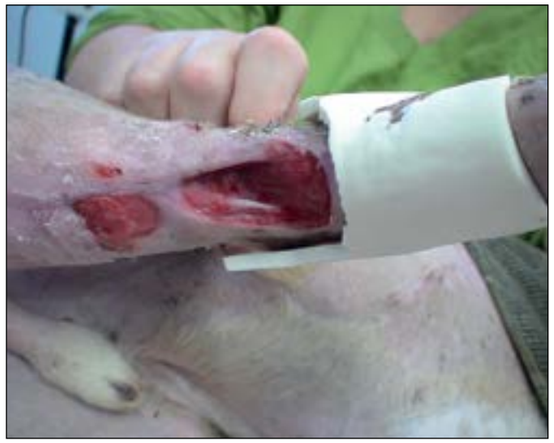
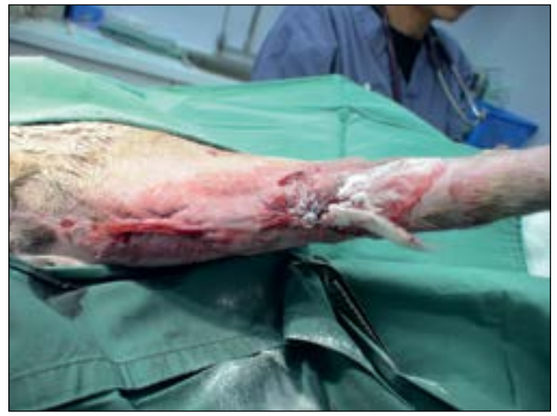


Figure 13B & C. Kendall AMD in use.

Once the exudation was under control we started using Comfeel Plus® strips again. We continued to have problems with breakdown at the proximal tibia and caudal thigh and appeared to be making no progress with the caudal stifle. It became evident that there was a draining tract preventing healing. This was packed with Restore Calcicare Alginate (Hollister)® to absorb exudate within the tract. Adapt Powder® (Hollister), a hydrocolloid powder, was applied over the wound. The caudal thigh wound was closed with 3/0 premilene in a horizontal mattress pattern. Relevo absorbent pad® was applied over this and held on with Hypafix®(Smith & Nephew), a hypoallergenic self adhesive fabric sheet. This allowed the dog to have reasonable mobility despite being bandaged.

Figure 14A. Draining tract management.



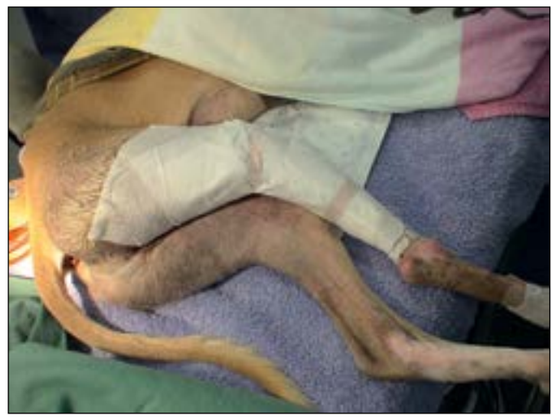


Figure 14B-C. Draining tract management.

Sutures were removed 14 days later and the remaining wound continued to be bandaged every 48 hours with Adapt Powder®, Relevo absorbent pad® and Hypafix®. ▶

Figure 15. At suture removal.

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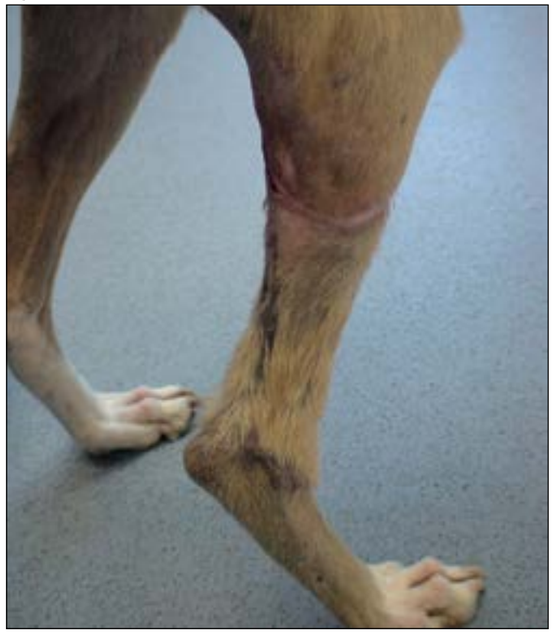
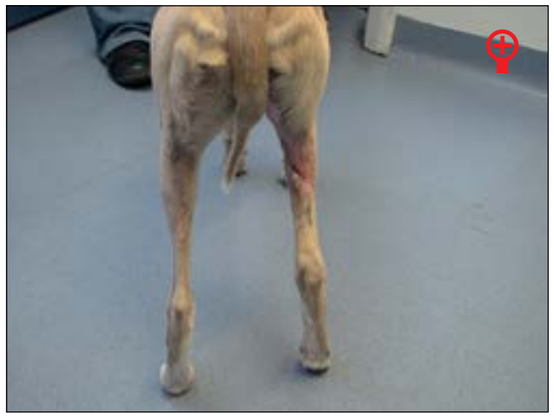
The end result 25 August 2011:

Figure 16A-C below.





Discussion

Although there are differences in the way humans and dogs heal,[1] the help of the human medical field in resolving difficult cases like this can be invaluable. The owner was fortunate to have a member of his family experienced in stoma care and human wound management available to work with the vets involved in this case and also able to access the various products used. All dressings tried worked well when used in the appropriate setting. Hydrocolloids are best in low to moderately exuding wounds, while the foam dressing was best applied when the wound was exuding heavily. Overall the authors found the Comfeel Plus® strips the easiest to use. This product has been shown to enhance the rate of epithelialisation and granulation tissue morphology when used in first intention healing surgical wounds in dogs.[14] The dressing is flexible and adhered well. The authors have been told by their human nurse contacts that skin irritation is sometimes seen in human patients when the product is used over several weeks. The authors experienced this problem in this patient but were able to manage it using Adapt barrier seal® as a protective layer between the skin and the dressing.

The Kendall™ AMD® dressing is very soft and worked well in protecting the mesh graft while absorbing exudate from under the graft. It did not adhere to the graft and was easily removed without disturbing the graft. Suturing the dressing to the peri wound skin helped prevent movement of the dressing while secondary layers of the bandage were applied.

In retrospect, if the authors had used the Adapt Powder® earlier in the management of the post graft wound breakdown, resolution of this wound may have occurred sooner. Although the Comfeel Plus® was maintaining a good granulation bed, the secondary dressings used to cover it were causing maceration at the margins. Comfeel Plus® does not necessarily require a secondary dressing, however these layers were used here to prevent the dog from removing the hydrocolloid dressing. Use of an Elizabethan collar had proven futile as he learnt to get around it or get it off. The Adapt Powder®, Relevo absorbent pad® and Hypafix® combination was well tolerated and allowed us to minimise the size and thickness of the dressing. This in turn gave the dog increased mobility and appeared to improve his overall demeanor.

Adhesive removal wipes were also used when removing adherent bandages from the skin. These appeared to significantly reduce the discomfort normally seen with removal of adhesive bandages.

Acknowledgements. The authors would like to thank the vets who provided advice on the Veterinary Information Network message boards and the many anonymous donors of bandaging materials and wound dressings used during the treatment of this patient.

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