THE UNIVERSITY OF SYDNEY
Centre for Veterinary Education
Professional Development Leaders

C&T

CONTROL AND THERAPY SERIES

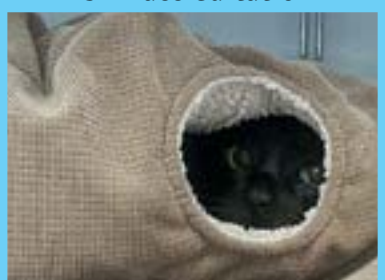
December 2013 ISSUE 273
Australia's Leading Veterinary Forum

Feature Article

Thoracodorsal axial pattern flap & novel non commercial closed suction drain for the reconstruction of chronic non healing axillary wounds in cats

Second 'bumper' issue in 2013!

Creating a 'Cat Friendly Clinic' & working towards ISFM accreditation



Go to e-book to view the Koala Restraint Film Clip

***Australaps superbus* known as the lowland copperhead**

Centre for Veterinary Education
Professional Development Leaders

C&T
CONTROL AND THERAPY SERIES

The Centre for Veterinary Education (CVE)
T. (02) 9351 7979
F. (02) 9351 7968
E. cve.publications@sydney.edu.au
www.cve.edu.au
Level 2, Vet Science Conference Centre,
B22, Regimental Crescent, The University of Sydney, NSW 2006.
Print Post Approved No. 224792/0012

DECEMBER 2013 ISSUE 273 — Australia's Leading Veterinary Forum

CONTENTS

PUBLISHER
The Centre for Veterinary Education (CVE)
Director
Hugh White BVSc MVSc MACVSc

EDITOR
Elisabeth Churchward
T. 9351 7979
E. cve.publications@sydney.edu.au
Veterinary Editors
Hugh White
Richard Malik

ADVERTISING ENQUIRIES
Elisabeth Churchward
T. 9351 7979
E. cve.publications@sydney.edu.au

DESIGNERS
BrandQuest

PRINTERS
University Publishing Service

Environmental policy
The C&T is produced using soya-based inks and paper from sustainable forests.

COVER IMAGE
Cover Image & Winner of Best Pic 'Share and share alike – Casper with bandicoot cobber' (Courtesy of Nicole Laing, VetWest Animal Practice, WA)

Another year is drawing to a close and once again it is an opportunity to reflect on what we have achieved in the past and what we would like to achieve in the future. How do you feel? Have you achieved your goals over the last 12 months and are you excited about the coming year, or do you feel that you are on a treadmill going nowhere with a gnawing fear that no matter how hard you try, things will not get any better next year?

Despite all the advances in technology and the ready availability of information, many people find themselves working just as hard, or even harder than ever. Competition is always increasing – whether it is suppliers competing in so many ways for your hard earned dollars, or aggressive competitors trying to get an even greater share of the pie. In veterinary practice there are so many conflicting demands on our clients' time and money. Sadly, veterinary services are often regarded as discretionary spending. A range of external influences beyond our control affect our traditional markets – from the impact of mining and coal seam gas developments on rural enterprises, to the reported decline in dog and cat ownership across the country. Compounding this situation is the annual production of around 500 veterinary graduates from 7 veterinary schools around Australia, with many wanting a career in clinical practice, often on their own terms!

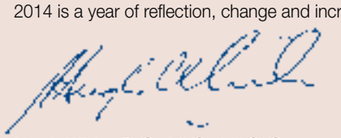
The same picture can be found in the continuing veterinary professional development (CPD) market, where an increasing number of CPD suppliers are competing for your custom. With the rapid expansion of webinars, short on-line courses and other internet resources, veterinarians can now source information in their own time, from wherever they may be. Social media is said to keep everyone in touch all of the time, yet how much of this provides the depth of discussion, debate and thought that accompanies more traditional interactions?

At the CVE, we strive to provide what you, the veterinarians, want and critically, what you really need. We continue to offer traditional avenues for learning – conferences, seminars, workshops and distance education, as well as webinars and short on-line courses. The feedback that we get from so many people is that nothing is as good as real time face-to-face experiences. Seminars and conferences provide both professional and social interaction with specialists and colleagues, while workshops provide a stimulating environment for both hands-on learning and in-depth thinking. **Our aim is not just to provide information, but to equip people with knowledge and skills, which will not only further their careers but stimulate them to continue the process of life-long learning.**

The C&T forum is one of the flagships of the CVE, which has been refined and developed over the years. It is now regarded by many practitioners as one of the most interesting, relevant and readable veterinary publications. This edition is no different, offering a diverse range of articles on both large and small animal topics and wildlife, as well as a large Perspective on Toxicology. Once again I would encourage you to access the interactive, on-line version as this enables you to view the videos and expand images.

I encourage everyone over the summer break to sit back and reflect on where they have been and where they are going, as generally it is not possible to see the wood from the trees when you are on the daily treadmill.

From everyone at the CVE we wish all of our members, speakers, tutors and readers all the best for the festive season and hope that 2014 is a year of reflection, change and increasing knowledge.



Hugh White BVSc MVSc MACVSc
DIRECTOR

Creating a 'Cat Friendly Clinic' & working towards ISFM accreditation

In September's issue of C&T, Andrea Harvey – one of CVE's Feline Distance Education tutors, feline Specialist at Small Animal Specialist Hospital (SASH) in Sydney and Australasian Representative for The International Society of Feline Medicine (ISFM) – introduced the ISFM 'Cat Friendly Clinic' accreditation scheme which has been recently launched in Australasia with the support of Royal Canin and CEVA. Andrea discussed the importance of stress in cats and the impact of this on feline patients and the veterinary clinic. Here, Andrea provides some practical advice for how veterinary practices can become more 'cat friendly'.

For more detailed information apply for a FREE 'cat friendly clinic' information pack which includes a full veterinary guide, all the details of scheme and how to apply for accreditation, by visiting: http://tinyurl.com/isfmcfc (or go to the International Cat Care website for further information: www.icatcare.org)

5 TOP TIPS FOR REDUCING FELINE STRESS IN THE HOSPITAL

- Minimise exposure to noise, dogs and general 'activity' – keep cats to quiet areas and be as quiet as possible at all times
- Less is more when it comes to restraint: be gentle with handling, utilise minimal restraint and use of bedding to reduce direct physical contact
- More haste is less speed when working with cats: go slow to go fast
- Provide cats with an opportunity to hide (utilising bedding, boxes, bottom half of cat carrier etc) wherever possible
- Keep cat carriers covered and on a raised surface

REMEMBER THE KEY WORDS WHEN WORKING WITH CATS:
quiet, calm, gentle, hide, height

10 steps to developing a 'cat friendly clinic' clinic

1. **Appoint someone in the practice as the 'feline advocate'**
Designate a vet or nurse who has a natural empathy with cats, and an understanding of their behaviour, and is willing to educate others in the practice, to lead the efforts to implement 'cat friendly' changes. This is a very effective way to start educating other staff, identifying required changes and implementing them. It also gives a point of contact for any feline concerns in the practice from staff or clients.

2. **Engage the whole veterinary care team**
In order for changes to be successfully implemented, it is crucial that all staff at all levels (receptionists, nurses, animal attendants, vets, management team) understand the reasons for changes and the benefits that they will bring. This means having at least a basic understanding of what makes cats stressed, the negative effects of stress on the cat, on the veterinary care team and on the client, how stress can be overcome and the positive effects of doing so. Some basic education may be essential for the whole team to really 'buy in' to the 'cat friendly' ethos. Most hurdles at this stage come from a lack of understanding of staff. There is also growing feedback from clinics participating in the scheme suggesting that being a cat friendly clinic makes a huge difference to clients, and results in an increased feline caseload, more willingness for investigative procedures to be performed, and increased uptake of preventative healthcare, and these can also be motivating factors for practice owners/managers.

3. **Understanding what makes cats anxious, recognising the anxious cat and reacting appropriately**
Understanding what makes cats anxious and recognising the subtleties of feline body language are key to enabling staff to react appropriately in order to reduce the cat's fear and not heighten it. Cats are generally sensitive to unfamiliar people and situations and their 'body language' may be misunderstood. The AAFP/ISFM Feline Friendly Handling & Nursing Care Guidelines should be a 'must read' for all staff involved in cats; they not only discuss cat handling but also cover all the basics about feline body language.

4. **Developing the right attitude and approach to feline patients**
This begins with the receptionist who answers the phone and makes the initial appointment or welcomes the client and their cat at the reception desk. Much can be done at this initial stage to impress the client with the practice's understanding and empathy for cats, and to help facilitate a 'low stress' visit. This will also apply to the vet or nurse examining the cat, any staff involved with their care during hospitalisation, and those that discharge the cat and show the owners how to medicate it, and what they need to monitor for at home. A few tips for each of these stages include:-

   - Train receptionists at the time of booking an appointment to make suggestions for easing the stress of transporting their cat to the clinic. This may include discussing the type of carrier used, leaving it out in the house for as long as possible before their appointment so the cat gets used to the carrier being part of their home environment, putting a comfortable bed in it and covering the carrier
   - Ensure that receptionists notice if waiting cats are about to have an encounter with a dog and advise clients to cover carriers, put them at a height, move to a separate waiting area etc
   - All staff need to be aware of the cat's sensitivity to sound, sight and smell and avoid sights, sounds and smells that will cause unnecessary distress. This involves keeping cats separate from dogs, and keeping cats in quieter, less busy areas of the hospital. It is important that all staff are quiet, calm and gentle around feline patients

5. **Handling cats to reduce and prevent anxiety**
Adopting a 'less is more' approach is critical to cat handling – cats generally respond well to minimal restraint. Many cats are frightened, but if they can be gently reassured rather than heavily restrained, this will help prevent most cats becoming defensively aggressive. Often fear is overlooked as a cause of aggression and these cats can be frequently 'mis-handled' only resulting in contributing further to their fear. Lots of tips and guidelines on best handling techniques are included in the AAFP/ISFM Feline Friendly Handling Guidelines, but a few key tips include:-

   - Always approach a cat in a calm and gentle manner and avoid direct eye contact. Gentle stroking and rubbing the cat's own pheromone centres (above the bridge of the nose and the preauricular area) can really help them to relax
   - A comfortable bed can encourage cats to sit or lie down; avoid slippery surfaces. Use bedding to allow cats to partially hide if they wish, during examination and minor procedures
   - Allow cats the opportunity to get out of the carrier on the floor and walk around the room for a minute before examination – this is allowing them to assess the environment and realise there is no danger
   - When cats don't want to get out of the carrier themselves gently lift out of a top opening carrier, or remove the top and door and examine the cat in the bottom half of the carrier
   - Be flexible and let the cat choose its preferred place and position for being examined/blood sampled etc. Try to adapt your techniques to the individual cat
   - Scruffing should not be used routinely as a first method of restraint and cats should never be lifted and held up by the scruff. Grabbing and immediately scruffing or heavily restraining a cat can be highly intimidating and often provokes defensive aggression

6. **Evaluate the cat's journey through the clinic and identify areas that may be important sources of stress**
Once you have a good understanding of cats, and what makes cats anxious, a wealth of information can be gleamed by taking a bit of time on a normal clinic day to walk through the practice looking at everything from the cat's perspective.

   When you walk in to the waiting room, what do you see and hear? Do you have to be near dogs? Do clients put carriers on the floor? Can a dog come up and sniff them? Do the receptionists show an understanding and empathy for the feline patients? Are they making suggestions for minimising stress? Is there provision for placing carriers at a height and covering them over? Are the cats having direct visual contact with other cats whilst waiting? When the cat is taken into the consult room do they have to walk directly past dogs?

   In the consult room, are carriers being put on the floor and cats allowed to get out of their own accord and walk around? Are they being immediately 'dragged' out of the carrier? Are they being examined calmly and gently, avoiding firm restraint and direct visual eye contact? If they are admitted, is there a cat only ward? What can cats hear and see in the ward and treatment area? Do they have places to hide (bedding, boxes etc) within their cages? Are procedures done in a quiet area? Are quiet clippers used? What can the cat hear and see when minor procedures such as blood sampling are being performed? Are procedures being done in a large, busy or open areas which subconsciously necessitates firmer restraint because you can't risk the cat jumping off the table? When cats are discharged, are owners given appropriate guidance on how to medicate their cat etc?

7. **Identify changes that can be made throughout the clinic to address the sources of stress identified**
Some changes that would be ideal may be physical changes, like creating a separate cat ward, whilst other changes may be simple, such as providing bedding that cats can hide in, or purchasing quiet clippers and soft Elizabethan collars. Some changes will be related to staff training and policies. The important thing here is not to be put off or overwhelmed if you have identified lots of potential changes and not all may be possible. Compromises always have to ▶

Authors' views are not necessarily those of the CVE
Back to TOC page

be made and it is surprisingly often the smaller, simplest changes that can make the biggest differences. The good thing with cats, being small, is that it isn't usually too difficult to find a small quiet space for doing examinations, minor procedures etc, even if this is utilising a consult room rather than using a busy treatment area.

Get creative with ideas; for example your waiting room may not be large enough to create a separate cat area, but perhaps there is somewhere that you can create some shelving so that cats in their carriers can be separated from dogs by utilising 3 dimensional space? Or maybe you can create some separation in time rather than space, for example by having some designated cat only consulting times. Display clear notices asking clients with dogs to keep them away from cat carriers, and reinforce this by asking dog clients to be considerate of cats in the waiting area. If there is a busy period, can a waiting cat be moved behind the reception desk or into a spare consult room?

Towels can be provided to cover cat carriers so they don't have direct visual contact with other animals.

The ISFM Guide to Creating a Cat Friendly Clinic gives much more detailed practical advice and guidelines, along with criteria required for accreditation

8. **Prioritise the most important changes**
Sometimes clinics give up because they are overwhelmed and don't think it is possible for them to achieve the criteria required for cat friendly accreditation. Each clinic will differ in what the most important changes are that are needed; for some it may be staff training, for others it may be setting up a cat ward. Prioritising just 2 or 3 changes that you are going to make first helps to focus everyone and make it a more manageable goal. It helps to implement some simple things quite quickly (e.g. obtaining 'cat friendly' bedding, waiting room changes), because more often than not staff and clients notice the beneficial effects on the cats quite quickly and this can motivate everyone to keep going with further improvements.

9. **Plan the implementing of changes**
It is really helpful to have a realistic plan for achieving your goals and to work through things in a step-by-step manner. It may also be useful, particularly in larger clinics, to have clinic protocols on things such as feeding, bedding, procedures, handling, pain scoring etc to ensure that all staff follow the changes. Sometimes plans fall down because all the staff are not motivated to implement changes, or too busy to think about them. Whatever the priorities, getting all staff 'on board' first is critical and this may involve some training with the feline advocate as well as reading the AAFP/ISFM feline friendly handling and nursing care guidelines. It is important to set realistic targets and timelines and hold regular meetings to ensure that planned changes keep moving along. The ISFM also runs distance education courses in feline friendly nursing which also may be of benefit to some clinics.

10. **Don't just stop there!**
Once you have implemented the changes that you identified would reduce feline stress in the clinic, don't stop there! Things are constantly changing and it is important that you continue to evaluate everything in the practice from the cat's perspective. Other areas in the clinic may change and what used to be a quiet area may become busy. There will be new staff that will always need training when they join the practice, new equipment may become available, and there are constantly new tips and ideas that staff may come up with, or you may learn at a CE event. Every practice is also different and you need to find what works best for the cats in your clinic, so sometimes there will be some trial and error experimenting with new ideas. Just be ready to always be assessing cats for signs of anxiety and be ready to change and try something different if you are still encountering a lot of anxious cats. Listen to the cats, they will tell you when you have got it right!

Further reading

ISFM Guide to Creating a Cat Friendly Clinic (available on registering interest at http://tinyurl.com/isfmcfc)

Cat friendly clinic supplementary information (will also be provided on registering interest at http://tinyurl.com/isfmcfc, and are also free to download from www.icatcare.org)

Rodan I, Sundahl E, Carney H *et al* (2011) AAFP and ISFM Feline-Friendly Handling Guidelines. *J Feline Med Surg* 13(5):364-75

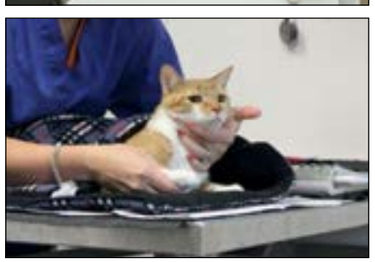
HC Carney, S Little, D Brownlee-Tomasso *et al* (2012) AAFP and ISFM Feline-Friendly Nursing Care Guidelines. *J Feline Med Surg* 14 (5), 337-349

Arriving at the 'cat friendly clinic'

News

Provide cats with an opportunity to hide (utilising bedding, boxes, bottom half of cat carrier etc) wherever possible

Signs can be used around the hospital to help to remind staff and clients of any new 'cat friendly' protocols and help improve compliance.



Using top-opening carriers, allowing cats the opportunity to get out of the carrier themselves, minimal restraint and use of comfortable bedding during examination and minor procedures, and avoiding direct eye contact are all simple things that go a long way to reducing feline anxiety in the vet clinic.

Back to TOC page

CVE 2014 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.** Listed dates are subject to change. Refer to www.cve.edu.au, for any updates.

EVENTS IN 2014

17-20 Feb	Emergency Conference	Sydney
16 Mar	Dermatology	Hobart
10-11 Apr	Feline Tales	Melbourne
4 May	Dentistry	Adelaide
1 Jun	Dermatology	Canberra
16-19 Jun	'Cradle to Grave' Conference	Melbourne
15-18 Sep	Canine and Feline Internal Medicine Conference: Special focus on diseases of the abdomen	Hawaii
19 Oct	Dentistry	Port Macquarie

Note - Please go to **www.cve.edu.au** to read about other events and workshops which had yet to be confirmed when this issue went to print.

TimeOnline

TimeOnline courses are short courses delivered wholly online giving you the flexibility to study when and where you wish and complete your course at your own pace.

Each TimeOnline program is worth 10 CVE points.

The courses are available to veterinarians and support staff.

To find out what courses we have coming in 2014 please visit **www.cve.edu.au/timeonline** or email us **cve.timeonline@sydney.edu.au**

Xmas Closedown

The University of Sydney and the CVE close down at COB on Tuesday 17 December 2013, reopening on Thursday 2 January 2014.

(Image courtesy of Jenny Parker from Shoot-ya-pooch Pet Photography www.shootyapooch.com)

www.vetbookshop.com

CVE is a distributor for Wiley-Blackwell Publications and several CSIRO Publishing titles. A 15% discount on titles from these publishers is currently available to financial CVE members. For more information on titles available plus details of Tables of Contents pages for CVE proceedings, please visit **www.vetbookshop.com**

… and more C&T articles and Perspectives needed
Thanks to every author who contributed articles or comments to the *Control & Therapy Series* (C&T) and to those who supplied images and visuals. Without your generosity the Series would cease to exist.

We are delighted to once again produce a 'bumper' 64-pager and thank all our readers and contributors for supporting the *C&T Series*.

Winners

Major Prize

Entitling the recipient to one year's free membership of the CVE

- **Matthew Tay:** Thoracodorsal axial pattern flap & novel non commercial closed suction drain for the reconstruction of chronic non healing axillary wounds in cats

CVE Publication Prize Winners

- **Cathy Opie:** Spinosad reaction
- **Donald Wiggins:** My favourite tool!
- **Heather Shortridge:** Unilateral thrombus in a cat
- **Virginia Grice:** Malicious freezing of a kitten

Winner of Best Picture

- **Nicole Laing:** Share and share alike – Casper with bandicoot cobber (cover image)

Winner of Best Film Clip

- **Mark Hynes** – Dental mystery in a Boston Terrier

‹e-book›

We need your email to forward you the link to the ebook version of this issue

Contact the CVE today to check your email status:-

Contact **cve.membership@sydney.edu.au** or call Jacqui Kennedy on (02) 9351 7979.

Visit **www.cve.edu.au/candtebook** to access the last 4 e-book issues

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.'

News

Congratulations to all our 2013 DE Participants

On behalf of our Distance Education Tutors and DE Co-ordinators, we would like to congratulate the following participants for their dedication to continuing veterinary education and pursuing Tom Hungerford's *Goanna Track to Success.* Tom Hungerford OBE BVSc FAVSc HDA, our first Director, was a legend in the veterinary profession and famous for this *Goanna Track* philosophy i.e. vets should take one area of veterinary practice and become thoroughly familiar with all aspects of it – conquer it completely. Then, you train others to run this aspect and proceed, like a goanna, to the next challenge and do the same again.

Beef Production Medicine
TUTOR: Paul Cusack
Sarah Bettridge, QLD
Sara Clark, QLD
Tim Gole, QLD
Libby Harriman, ACT
Peter Launders, ACT
Felicity Miller, VIC
Suzanne Van Es, TAS

Behavioural Medicine
TUTOR: Kersti Seksel
Hannah Beveridge, QLD
Miranda Bourque, United States
Katriona Bradley, Hong Kong
Tawnya Copland, Canada
Julie Culver, United Kingdom
Ruth Daling, United Kingdom
Margaret Giles, NSW
Joanna Goldman, NSW
Megan Kearney, NSW
Euan Kilpatrick, VIC
Nicole Lobry de Bruyn, WA
Sneha Mata, NSW
Joanna McLachlan, NSW
Pierrette Mercier, Canada
Selina Neill, QLD
Helen Purdam, ACT
Michael Rae, WA
Gillian Shippen, SA
Charlotte Smithson, WA
Rayya Takieddine-Malaeb, VIC
Pauline Taylor, Hong Kong
Eleanor Tuffley, NSW
Jessica, WAllace, NSW

Cardiorespiratory Medicine
TUTOR: Nick Russell
TUTOR: Niek Beijerink
Sarah Bembrick, NSW
Sarah Coall, NSW
Abbie Couper, VIC
Sheila Gadaloff, QLD
Khor Hua, Malaysia
Nutrajgorn Jearranai, Thailand
Clifton Jones, QLD
Ukritkarn Konglertmongkol, Thailand
Aomusa Kuaha, Thailand
Marea, Sawtell, NSW
Shalini Sinnan, NSW
Napapen, Wachiratada, Thailand
Wipada Weerakijpanich, Thailand
Kelly Yeo, VIC
Angel Yip, Hong Kong

Clinical Pathology
TUTOR: Sandra Forsyth
Deanna Crea, ACT
Kylie Green, SA
Karina Harding, ACT
Emily Haywood, VIC
Viviane Itaziki, Malaysia
Tiffany Jacobs, WA
Amanda Johnson, NSW
Blair Kennedy, NSW
Heidrun Kraft, Hong Kong
Myffawny Lawrie, QLD
Alison Neef, NSW
Lesa Potten, ACT
Erni Sulistiawati, Indonesia
Charles Tilley, NSW
Karin, VIChukit, Thailand

Dermatology
TUTOR: Ralf Mueller
TUTOR: Sonya Bettenay
James Blackshaw, NSW
Rosemary Cox, VIC
Jennifer Griffiths, SA
Kelly Harper, NT
Tristan Ingle, ACT
Ho Yi Candy Lam, Hong Kong
Fiona Macintosh, QLD
Caroline McCarthy, VIC
Elana McKeon, VIC
Pimpinun Rojanai, Thailand
Pattaraporn Vechsuruck, Thailand
Sompoach Vuthikornudomkit, Thailand
Jane Whitley, VIC
Rebecca Williams, QLD
Vorapun Wiwatvisawakorn, Thailand
Mark Yostos, NSW

Emergency Medicine
TUTOR: Sandra Forsyth
TUTOR: Trudi McAlees
Jason Arlaud, VIC
Timothy Biffin, NSW
Shelley Brown, ACT
Jenny Brown, NSW
Jeffrey Clyne, SA
Rebecca Coleman, WA
Jessica Cooke, NSW
Natsuko Druery, NSW
Louise Flinker, VIC
Lauren Fraser, SA
Laetitia Geiger, NSW
Storm Gifford, NT
Lee Grant, TAS
Rhianna Jones, ACT
Camellia Leong, Singapore
Jun Loh, VIC
Sheree Maas, VIC
Thananchanok Narongraks, Thailand
Katherine Nash, QLD
Andrew Neutze, NSW
Jodie Northcott, ACT
Michelle Peters, VIC
Jun Pushatrirat, Thailand
Stephen Reid, WA
Ellene Rossouw, SA
Amy Simon, NSW
Philippa, Wallace, WA
Helen Wilson, ACT

Equine Reproduction
TUTOR: John Chopin
TUTOR: James Roger
Luke Annetts, QLD
Jessi Flynn, QLD
Sarah Goodwin, NSW
Chris Hallett, NSW
Simone Herbert, WA
Megan Jolly, NSW
Holly Ludeman, WA
Alexandra Pearce, VIC
Betty Rindfleish, NSW
Jodie Smith, NSW
Jennifer Watts, NT
Ian Wilbraham, QLD

Feline Medicine
TUTOR: Carolyn O'Brien
TUTOR: Andrea Harvey
TUTOR: Sarah Caney
TUTOR: Wayne Mizon
TUTOR: Samantha Taylor
TUTOR: Sheila Wills
TUTOR: Elise Robertson
Sarocha Akarakijwiroon, Thailand
Tamara Albers, NSW
Amelia Alexander, NSW
Sarah Anderson, NSW
Loke Yuen Ang, Malaysia
Helen Beban, New Zealand
Krissda Boonaramrueng, Thailand
Elisabeth Buist, United Kingdom
Karim Chammas, NSW
Jo-Ann Chan, Singapore
Joyce Chi Taiwan, Province of China
Hiu Ying Choi, Hong Kong
Catherine Conmy, Ireland
Maria da Fonseca, Portugal
Caroline Daly, Hong Kong
Catarina Eliasson, Sweden
Ana Fernandes, Portugal
Caroline Ficker, United Kingdom
Summer Fu, Hong Kong
Laura Graveling, Canada
Eva Heger, Sweden
Tiffany Tsz Ting Ho, Hong Kong
Xiangting Huang, Singapore
Megan Irvine, New Zealand
Reza Kamgarpour, QLD
Hyun Jeong Kim, NSW
Melanie Kuehn, VIC
Wai Pong Lau, Hong Kong
Wing Yeung Lau, Hong Kong
Laura Lee, Singapore
So Kei Liu, VIC
Cheryl Lordan, SA
Gwenda Lowe, Singapore
Karen McCormick, QLD
Claire Mellor, United Kingdom
Geetha Nellinathan, Singapore
Jenni Nystedt, Finland
Gitty Otte, The Netherlands
Rachel Payne, WA
Piyawun Phurahong, Thailand
Sarunya Prapitpa, Thailand
Kerrie Rodgers, VIC
Maayke Ruiter Netherlands
Chantal Samaratunga, QLD
Elizabeth Smit, TAS
Barbara Sortini, Italy
Sharon Stevens, United Kingdom
Thanikran Suwannachote, Thailand
Leah Taylor, United States
Mary Thomson, Canada
Sasithorn Trairatthanom, Thailand
Avence Tsao Taiwan, Province of China
Dora Urban, Hungary
Euapong Varatorn, Thailand
Donald Wiggins, United Kingdom
Lynne Wilkins, New Zealand
Ann Williams, QLD
Claire Williams, United Kingdom
Anne Woolley, VIC
John Yang, NSW
Ivy Yue, Hong Kong

Internal Medicine: Keys to Understanding
TUTOR: Darren Merrett
TUTOR: Kate Hill
TUTOR: Jennifer Brown
Flora Adams, VIC
Samantha Allmark, QLD
Rebecca Ardrey, QLD
Heidi Beroll, Canada
Julia Brougham, NSW
Kumiko Chono, VIC
Cherry Chung, Hong Kong
Nicola Cotton, VIC
Susanna Gamage, NT
Nadthagarn Glaewketgarn, Thailand
Lin Hoi, Hong Kong
Saila Holopainen, Finland
Margaret Jenner, VIC
Annie Kicinski, NSW
Karin Kuh, Hong Kong
Angus McMillan, SA
Rebecca Mifsud, VIC
Brigit Pitman, NSW
Praewpailin Putta, Thailand
Ema Rankin, NSW
Julie Sich, NT
Jiradet Trairatthanom, Thailand
Elly Whittle, QLD
Chuan Wong, Singapore
Jeffrey (Ho Luen) Yip, Hong Kong
Ricky Yuen, Hong Kong

Internal Medicine: A Problem Solving Approach
TUTOR: Jill Maddison
Alex Avery, New Zealand
Ruth Barrett, VIC
Patcharasarin Buathet, Thailand
Jessica Chan, NSW
Justin Choo, Hong Kong
Osathee Detkalaya, Thailand
Maria Di Genova, VIC
Kellie Gray, WA
Susanne Hafner, QLD
Nicola Hooper, Hong Kong
Kristie Karikios, NSW
Helen Kwan, Hong Kong
Reanne Kwok, Hong Kong
Kin Fung Lam, Hong Kong
Kee Chee Jacqueline Lam, Hong Kong
Pui Pui Liem, NSW
Amanda Miller, NSW
Naomi Morgan, NSW
Mei-Pamela Ong Sheau, Malaysia
Hester Raijmakers, NSW
Sarah Robson, VIC
Leah Wright, VIC

Ophthalmology
TUTOR: Robin Stanley
Sarah Archard, VIC
Gladys Boo, Singapore
Thawachit Boonyachotmongkol, Thailand
Kriengkrai Chatthakulchai, Thailand
Chris Collins, NSW
Tim Hill, ACT
Andrew Laws, NSW
Nitiwadee Lertitthikul, Thailand
Amy Limbert, SA
Philip Lindsay, New Zealand
Justin McKay, VIC
Sandhya Nair, Singapore
Katerina Papaioannou, Greece
Angela Phillips, NSW
Rosemary Pincini, VIC
Jenny Wilsher, VIC

Sonology
TUTOR: Karon Hoffmann
TUTOR: Cathy Beck
Julian Alexander, VIC
Leah Bonnette, NSW
Heather Breckenridge, NSW
Nicole Brown, New Zealand
Elizabeth Craig, QLD
Elisa De Bont, VIC
Adrienne Easton, QLD
Laticha Engle, QLD
Priscilla Foong, VIC
Fiona Hendrie, VIC
Zane Hsu, NSW
Mila Kasby, NSW
Marlene Kearns, NSW
Jun Jia Koh, Singapore
John Ley, VIC
Amanda Lugsdin, VIC
Christopher Mather, NSW
Cammy O'Rourke, SA
Ildiko Plaganyi, VIC
Stephen Reis, NSW
Michael Small, New Zealand
Pronphan Sukanan, Thailand
Shae Sullivan, NSW
Yuki Totsuka, NSW
Jayde Watling, NSW
Emma Winsor, QLD

Surgery
TUTOR: Wing Tip Wong
TUTOR: Guy Yates
Wesley Allan, VIC
Carla Appelgrein, SA
Erni, Wati Arip, Malaysia
Jocelyn Birch Baker, QLD
Laura Brown, NSW
Chi Lee Chen, Hong Kong
Nicholas Chng, Singapore
Dario Conesa, NSW
Joshua Dearness, SA
Joshua Ekthamasut, Thailand
Jason Evans, VIC
Camilla Forss, VIC
Kylie Grant, VIC
Lauren Harrison, VIC
Olutunbi Idowu, ACT
Terri King, QLD
Nipon Lertsiriladakul, Thailand
Anthony Matthews, Hong Kong
Kelly McDermott, New Zealand
Daniel Mills, WA
Sarah Patullo, NSW
Chia-Kang Peng, Hong Kong
Gundula Rhoades, NSW
Jeerapat Sermwatanakul, Thailand
Jonathan Sirasch, ACT
Tanate Tanapant, Thailand
Francis Tay, Singapore
Leana Watermeyer, NT
Chen Pei Yii, Malaysia
Genevieve Zhang, NSW

Congratulations to our DE Early Bird Winners for 2014

Paying early ensured the following vets not only secured a place in the DE course of their choice for 2014, they also received a hefty discount and were the 3 lucky winners in our Early Bird draw, winning an iPad each. Congratulations to Stephen Fleischer 14IMPS, Daniel Lawrence 14Sono and Hoek Yit Cingy Tan 14IMPS (pictured left to right) and to all our DE Participants who secured a place in our 2014 programme.

Authors' views are not necessarily those of the CVE

Back to TOC page

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

Part 4: Wildlife Flashcard Series

Mammals

C&T No. 5348

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

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)**.

Koala Restraint

Part 4.4

KOALAS

Be aware:-

- Have powerful teeth and claws (both forearms and hind limbs) – can be aggressive particularly after a difficult catch or being attacked by a dog.
- Marsupials – always check for pouch young.
- Koalas have a very specialised diet; they only eat Eucalypt (no supplementary/artificial diets).
- Faecal output is an important assessment parameter.
- Orphaned koalas are highly specialised and if unfurred will require regular feeding (every 2-3 hours), specialist/experienced care is highly recommended.
- You can age a koala by checking the wear on the upper premolar and first molar teeth. Little or no wear indicates that the koala is probably less than 3 years old. If they are worn down to gum level it is probably 10 years +.
- Injuries from dog attacks are often not discernable. A thorough assessment is required if there is any evidence (i.e. saliva on the fur) or possibilities of contact from a dog.
- Marsupial young are born very under-developed and until the stage of development that we are able to successfully hand rear young joeys, they are considered to be 'unviable'. Consult a trained/experienced person for assistance with identification and confirmation on viability.
- Native marsupials don't get tick paralysis.

Handling

- Koalas should be caught using a large blanket or towel and placing in an open top enclosure. To catch, cover the koala's forearms with the blanket and grip the Koala's forearms (between the shoulder and elbow) through the blanket ensuring that the head remains covered. Roll your hands in to create protection with the blanket so it bites that instead of you. Alternatively, you can roll up a small blanket and put that gently against the Koala's chest – it will often grip it and bite into it, distracting it from your hands!
- Never pick up around the ribs.
- Very sick/weak Koalas can be handled by wrapping a blanket around them, and then placing one hand across their thoracic region and the other under their rump.
- Small joeys can be handled by supporting their weight under the rump and placing one hand across their thoracic region. Sometimes they can be aggressive and will require the above adult handling technique.
- Older joeys can be held gently in your hands; keep in a pouch at all times.

Housing the sick or injured koala

- Preferred enclosure temperature for Adults is 28˚ Celsius. Orphaned joeys just furred to furred 28˚-30˚ Celsius and 32˚-34˚ Celsius for unfurred.
- To mimic the natural environment and reduce stress, create a makeshift tree fork. Roll 2-3 towels folded in half lengthways.
- Adults can temporarily be placed in a large open top wire carry cage; soft towels on the bottom and a makeshift tree fork.
- If cold, heat can be given by placing a heat pad under half of the enclosure; this must be monitored with an indoor/outdoor thermometer.
- Adult koalas in a recumbent state can be housed on a soft bed. Still provide Eucalyptus leaves and a makeshift tree fork.
- Orphans can be placed in a cotton pouch. Heat must be given; heat just furred/furred 28˚-30˚ Celsius and unfurred 32˚-34˚ Celsius. Ideally they should be housed in a Vetario® or Humidicrib and monitored with an indoor/outdoor thermometer or alternatively in a wire cage or plastic carry cage with towels underneath and around for support.

Emergency diet

- Offer a variety of fresh Eucalyptus leaves (can be extremely fussy) for koalas just furred or onwards.
- Orphans can be given water and Glucodin initially for first two feeds, then suitable milk replacer (Divetelact®, Biolac® M 100 or Wombaroo Koala Milk Replacer®). This is best given by a glass syringe and appropriate sized teat, due to the high risk of aspiration. Orphaned Koala joeys should be referred to a specialised/trained carer.
- Koalas, when they first emerge from the pouch, nuzzle the cloaca of their mother and consume special unformed maternal faeces called pap. This prepares its digestive tract with bacteria necessary for it to eat a Eucalypt diet. Joeys in care are offered Eucalyptus leaves at approximately 6 months once they start on a course of pap.

Assessment under anaesthetic

Gaseous

Use an anaesthetic mask at 5% induction; can take 3-5 minutes.

Maintain using a mask on Isoflurane at 1.5-2% with an oxygen flow rate of 1 L/min.

Anaesthetic agents (preferred)

Alfaxan® CD RTU 3 mg/kg – (I/M)

Intubation (difficult)

Koalas are difficult to intubate and if available the use of a 4mm diameter rigid endoscope with a 4.5 endotracheal tube slid over the sheath will greatly assist to visualise the larynx and aid intubation. Alternatively, use a laryngoscope with a straight-blade and cuffed endotracheal tube with a stillete (most adult koalas will require a 4-4.5 endotracheal tube). 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. Place in lateral recovery position on soft bedding and extubate only once the patient is swallowing, due to having a long soft palate. With Joeys, Vetario® or Humidicribs are ideal during post-operative recovery.

Preferred routes

- Subcutaneous – administered in loose skin lateral neck/chest, over thigh area.
- Oral – given via a syringe.
- Intramuscular – administered to dorsal lumbar muscles, cranial and caudal thigh.
- Intravenous – cephalic vein or saphenous vein.

Fluid Therapy

It is important to remember to warm the fluids being administered. Administer intravenously with the assistance of an infusion pump. Using 0.9% sodium chloride, dose the patient at 5% of its bodyweight. Fluid therapy can be administered subcutaneously or by using standard I/V infusion rates. Syringe pumps are ideal to use in orphans.

Do not administer fluids too rapidly or overload – use the same caution as would be used in cats.

Euthanasia methods

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

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

Figure 1a. Koalas should be caught using a large blanket (not too thick that you cannot get adequate grip) or a towel.

*Figure 1b. Example of the technique without the towel – **This is a captive koala not a wild Koala***

Authors' views are not necessarily those of the CVE

Back to TOC page

Figure 2. Juvenile koalas can be handled by supporting their weight under the rump and holding their forearms; sometimes they can be aggressive and will require the adult towel technique.

Figure 3a. (above) A towel should always be provided to mimic a tree as koalas naturally feel safe when holding onto something.

Figure 3b. (left) Housing the recumbent patient.

Figure 4. A makeshift 'mother' and something to hold onto reduces the stress on orphans.

Figure 5. Koalas are highly prone to stress; to minimise this keep joeys in a pouch even when weighing.

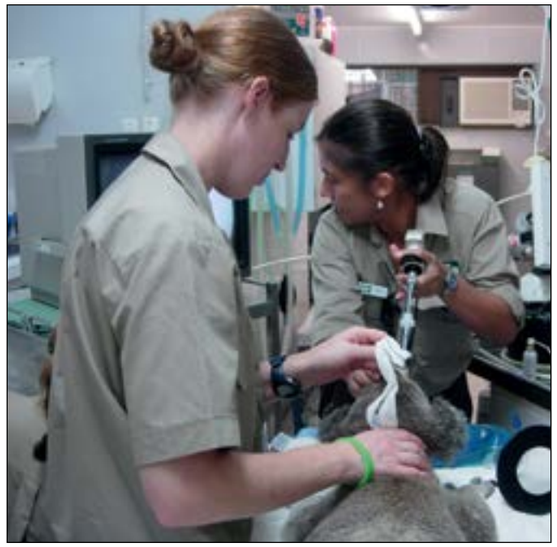


Figure 6. Intubation with the assistance of an endoscope.

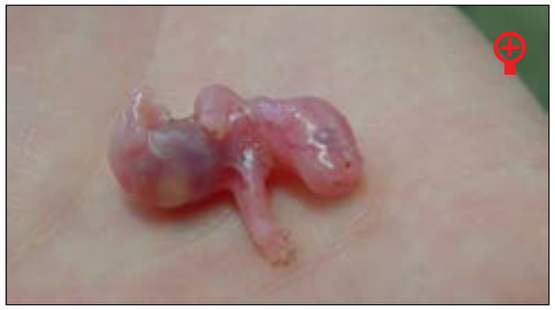


Figure 7. Consult a trained/experienced person for assistance with identification and confirmation on viability.

VALE DR ALAN WARNER

e-book

Read friend and colleague Dr Jim Phelan's obituary here.

Long-term Member and supporter of the CVE/PGF and enthusiastic and generous contributor to the *C&T Series*, sadly Alan died in mid-September after a 6 week struggle with a very aggressive form of cancer. Al, a great believer and participator in continuing veterinary education and particularly the *C&T Series* as a forum, will be well known to *C&T Series* readers from his wonderful array of articles (available in the CV*e-library*), many on Australian wildlife.

True to form, Al submitted (his last) C&T in June 2013 'Lumpy Jaw Success Story', a case about a young white female Red Neck wallaby (*Wallabia rufogriseus*) seen with colleague Dr Jim Phelan. Look out for this article in March 2014 Issue 274.

Vale Alan – a great Australian and a great Vet.

Scooby: A miniature pony in more ways than one!

C&T No. 5349

Heather Shortridge, Sarah Van Dyk & Jason Andrews
New England Veterinary Centres
212 Rusden Street, Armidale NSW 2350
T. (02) 6771 0200
E. heathershortridge@gmail.com

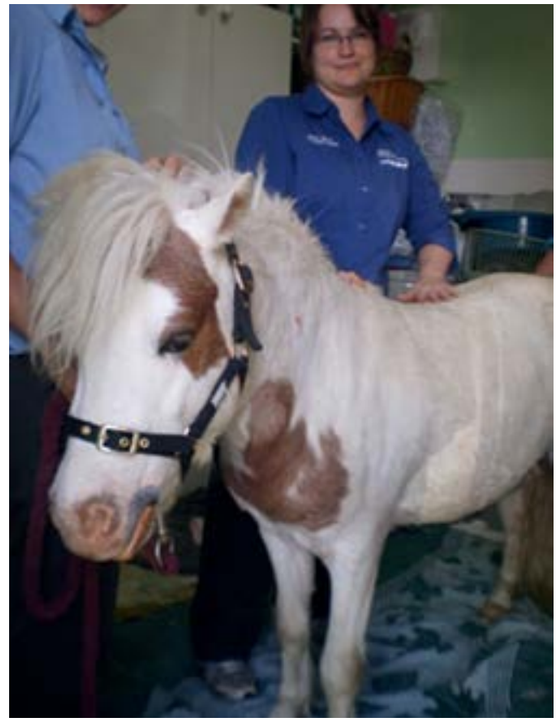


Figure 1. 'Scooby'.

Scooby, a miniature pony, was anaesthetised in the field ahead of his planned gelding. However once 'Scooby' was down, there were no testicles to be found! Scooby was allowed to wake up, and options discussed with his owner. Being a small fellow of only 150kg or so, it was decided that we could anaesthetise him and put him on gas in our small animal facility, to allow more time to hunt for the missing testicles.

A date was set, and a large (size 16) endotracheal tube was ordered for the occasion. The laundry was cleared to allow the surgery to take place, and a crowd of enthusiastic onlookers gathered.

Scooby was led into the clinic, and premedicated with xylazine (110mg) and butorphanol (1mg). A visual inspection was made of Scooby's inguinal area, and there was no sign of any testicular descent (Figure 2). ▶

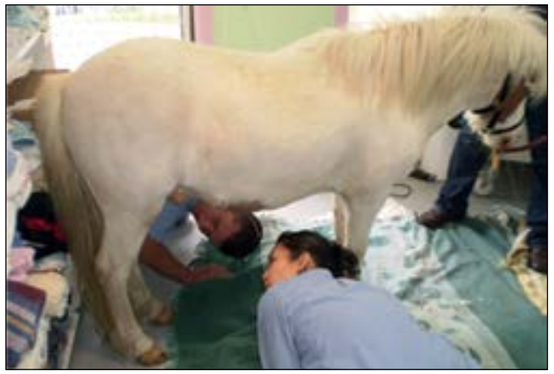


Figure 2.

Scooby was then induced with 260mg ketamine and 1mg diazepam. We induced him onto a pile of towels and blankets (Figure 3).

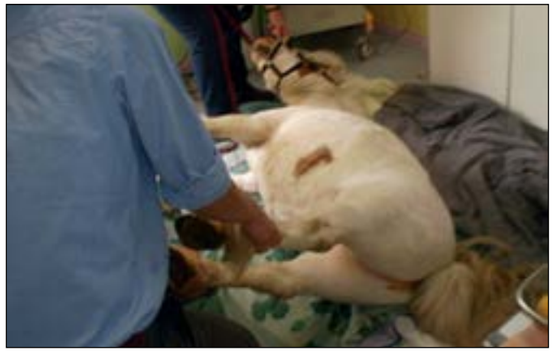


Figure 3.

Scooby was intubated, placed in sternal recumbency, and hooked up to isoflurane. Lubricant was instilled in Scooby's eyes. Hartmann's Solution was given via the jugular vein, at 1.5L per hour during surgery (Figure 4).

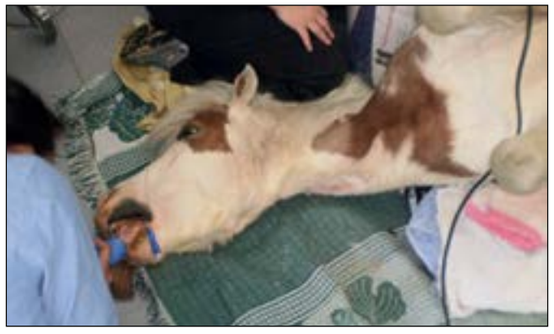


Figure 4.

Our surgeon began his hunt for Scooby's testicles, and it proved to be quite a challenge. An incision was made in the inguinal region. Eventually what seemed like a slightly gonad-like structure was found, but it was really surprisingly small. A text book was called for, and on comparison it was clear that this walnut sized object was, in fact, Scooby's miniature testicle (Figures 5&6).

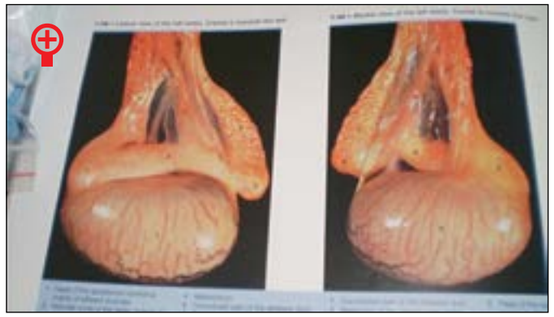


Figure 5.

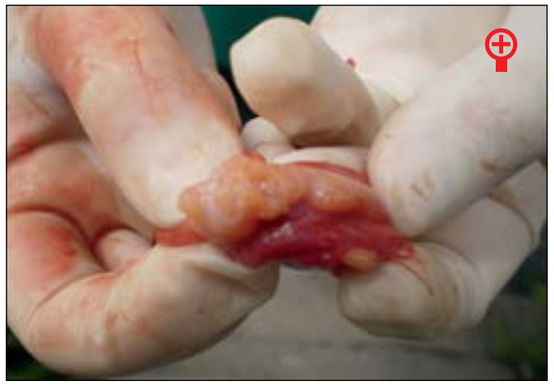


Figure 6.

The contralateral testicle was also removed via an inguinal incision. The incision was closed with both subcutaneous and skin sutures (Figure 7).

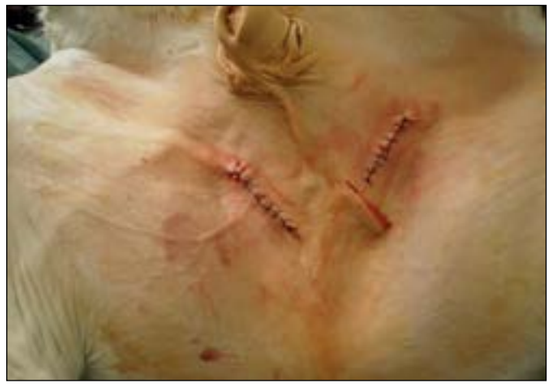


Figure 7.

Scooby was given perioperative flunixin and penicillin and recovery from anaesthetic was uneventful. We had anaesthetised Scooby for 90 minutes in total, so it was good that we had been able to hook him up to gas.

Scooby was sent home with penicillin to continue twice daily; however, a week after surgery the surgical sites became swollen, and so he was given 5 days of intravenous gentamicin as well.

Since surgery Scooby has gone very well, and is a much loved childrens' pony.

The correct way to restrain a large animal on the ground

C&T No. 5350

Nick Scott
Stewart Street Veterinary Hospital
156 Stewart Street
Bathurst NSW 2795
T. (02) 02 6331 1222
E. gainickscott@dodo.com.au

I was shocked to watch the Murdoch University Vet students on the recent ABC show trying to lie on a horse`s head to keep it on the ground and even more shocked when our practice`s own recent graduate from Sydney went to do the same thing.

Clearly our students are not being shown the safest most effective way to do this in a paddock situation and I would assume in University teaching hospitals most animals would be recovering in padded stalls.

In a practice situation we often have to restrain a horse while coming out of anaesthetic, or after having gone down for some other reason, while cows often have to be kept on their sides for intravenous milk fever treatment or sometimes during calvings etc.

Lying on the head is very ineffective as these large animals can generate tremendous power when lifting their head. Once they lift their head the handler is at considerable risk. Before I learnt to always instruct my handlers before starting the surgery, one colt threw his head up and down and broke the handler`s leg in the process.

I believe that the only person that would be strong enough and heavy enough to keep a horse`s head pressed on the ground would be so heavy that they would be very likely unable to get up again when finished!

The attached photos show that with the lead held out in a straight line with the animal`s body and with the nose being held up slightly from the ground, the animal can be held far more effectively with much less risk to the operator. Once you try it you will be surprised at how little weight has to be placed on the lead to keep the animal from getting up.

You will note from the photo that I do not think it essential to wash every last drip of blood off a horse after gelding – too much damp would merely attract mud.

However there is ONE GLARING MISTAKE in the photos. It is a good idea to cover the animal`s eyes after an anaesthetic – hopefully it will prevent sun damage to the retina and the animals are noticeably less inclined to get up too soon.

However, in my rush to get a good picture the eye cover should NEVER BE WRAPPED IN THE HEADSTALL. If the animal did manage to get up while blindfolded the resulting panic could be most unpleasant.

Figures 1-3. Remember, the eye cover should NEVER BE WRAPPED IN THE HEADSTALL.

Authors' views are not necessarily those of the CVE

Back to TOC page

WINNER

Spinosad reaction

C&T No. 5351

Cathy Opie
The Pet Doctors
946 East Coast Road
Browns Bay, Auckland, NZ
T. + 9 478 4349
E. vet@thepetdoctors.co.nz

Thanks to the following authors for allowing us to publish a series of email exchanges.

Dear Richard, I wondered if you have heard of any serious adverse events with the use of Comfortis®? Are you able to forward this email to Mandy Burrows as well please as I don't have an email address for her but I'd be really interested in her opinion. Since attending the Dermatology conference held by the CVE in 2009, we have begun to use Comfortis® in our practice – to date with no serious side effects other than the occasional episode of nausea and vomiting.

However, yesterday, I had a 1.7kg, FS, 13-yr-old Pomeranian present with status epilepticus that had initially begun as excessive salivation and then progressed to weakness overnight and seizures by the time it came to me. The dog had received a single 140 mg Comfortis® tablet about 8 hrs prior to developing salivation.

I should say that the dog had been well up until this point (aside from dreadful dental disease).

The owner had purchased Comfortis® for dogs weighing 2.3 – 4.5 kg approximately 14 months earlier at an annual health check when the dog weighed 2 kg and had given it to the dog on only a couple of occasions since purchase with no untoward effects.

I started supportive therapy consisting of intravenous saline to replace the fluid deficits present, intravenous diazepam and then phenobarbitone while waiting for blood results to investigate potential extra-cranial causes of seizures. The only change in the bloods was a mildly elevated globulin (46g/L) and a high neutrophilia (26 x 10^9/L) and thinking that the dog may have an intra cranial lesion, I referred it to our specialist centre for further work up and intensive overnight care.

The specialist believes it to be a case of an adverse reaction to Spinosad, based on the temporal link, and has apparently seen a few cases (in dogs not receiving any other medications, nor with a history of seizures). I just wondered, given that the drug has been available in Australia for longer than here in NZ, if you are aware of any significant adverse events and whether you have any guidelines for its use, other than those outlined by the manufacturer?

Or is it just a case of too high a dose for the bodyweight?

Reply from Richard Malik, CVE

E. Richard.malik@cve.edu.au

I am not very knowledgeable about this. I thought generally it's a safe drug. There is an issue about potential toxicity if the drug is given at the same time as an avermectin compound. Any chance they gave it ivermectin or moxidectin without you knowing about it?

I don't know dosages by heart – how much should the dog have got on a mg/kg basis. What I'm trying to say is: Was it given a 5 kg dose?

It's important we record these things. Have you contacted the manufacturer, Elanco? They will be interested, and be helpful. Do you have a regulator in NZ?

I think this new class of drugs is a **huge step** forward for flea control – but we always need to know about how safe they are and how safe is relative overdosing. Maybe the periodontal disease is more important than you think.

What about the dog's liver function?

Finally, in my experience hyperA is very common in old Poms – could it have this as a comorbidity?

Let's see what my more knowledgeable colleagues have to say.

P.S. This is off the top of my head. I will now google spinosad and seizures and see what comes up.

Reply from Mandy Burrows BVMS MANZCVS FANZCVS

Veterinary Dermatologist
Registered Specialist in Veterinary Dermatology
Animal Dermatology Clinic-Perth
Murdoch University Veterinary Hospital, Murdoch WA 6150
Perth Veterinary Specialists
T + 61 8 9360 7387 F + 61 8 9360 6482
E. A.Burrows@murdoch.edu.au

Thank you for including me in this discussion, Cathy.

Comfortis® in my experience is a safe and very useful drug in dermatology. In dogs we use it at 30mg/kg once a month (or every 14 days) for therapeutic flea trials. The dose band, however for Comfortis® is 30 to 60mg/kg so this dog has received an overdose but not a significant one. For cats, we use it at 50mg/kg once a month (or every 14 days) for therapeutic flea trials. Please note the drug is not registered for any purpose in the cat in Australia and not at 14 day intervals in the dog. ***(Editor's Note:** It has subsequently been registered for cats in New Zealand.)*

Toxicology and safety studies performed with spinosad (http://www.inchem.org/documents/jmpr/jmpmono/2001pr12.htm#1.0 and http://www.fda.gov/downloads/AnimalVeterinary/Products/ApprovedAnimalDrugProducts/FOIADrugSummaries/ucm062334.pdf, indicate that the product does not appear to be inherently neurotoxic in mammals, including dogs. In one safety study, Beagles were dosed with more than 10x the monthly dose over 10 days and no neurotoxicity was noted, so the relatively minor overdose that the Pom received should not have led to the seizures due to direct spinosad toxicity based on that data. However, in the US field trial in dogs with the product, seizures were noted to have occurred during the trial in 2 dogs with pre-existing epilepsy dosed at the correct label rate, although the cause for these was not determined. But perhaps this is the reason for the precaution on the Australian and NZ labels which recommends that the user 'seek veterinary advice' regarding using Comfortis® in animals with pre-existing epilepsy. Also interesting to note is that the US product label for Comfortis® (http://elms.xh1.lilly.com/comfortis-product-label.pdf) has a section in it on post-approval experiences and within this, seizures are listed as one of the adverse events which has been noted in association with product use.

In addition there is the well documented issue of neurotoxicity associated with spinosad and concurrent dosaging with ivermectin that Richard alludes to, and that contraindication is only with extra label dosages of ivermectin. I have used the drug extensively in both dogs and cats in my dermatology practice and not had seizures reported; I have infrequently seen gastrointestinal adverse events, usually vomiting and especially in cats. I would strongly endorse the suggestion you report this to the APVMA and contact Elanco. The company will be very helpful and thorough. The question, as Richard correctly poses, is whether there are other co-morbidity factors that are contributing to this adverse event in this patient.

Reply from Cathy Opie

Thanks for your help, everyone. I've made an initial report to Elanco. There was no elevation of ALT, AST on the bloods. I wasn't able to get a urine sample from her at presentation, so don't know her USG.

She could have hyperA – her hair coat is thin and there is that neutrophilia present.

She has not received ivermectin and has no prior seizure history. The dose received was 140mg, so she received 82 mg/kg. Unfortunately, after initially appearing to do well at the specialist centre, this little dog died suddenly on the third day. She has been cremated and a necropsy was not performed which is a shame as it may have yielded some very useful information as to the cause of death and any association with spinosad.

Richard, regarding your question about publishing this in C&T, I am happy for you to go ahead. It's always a great forum for what vets out there in practice are seeing.

Reply from Stephen Page BSc(Vet)(Hons), BVSc(Hons), DipVetClinStud, MVetClinStud, MAppSc(EnvTox), MANZCVSc

Advanced Veterinary Therapies
PO Box 905
Newtown 2042 NSW AUSTRALIA
E. swp@advet.com.au

Please find some information attached and below from the adverse event/experience programs in the US and Australia. I would recommend that the case be discussed with the manufacturer (ELANCO) and it would be very beneficial to publish a case report to alert the profession (especially if several cases have been treated).

CVM

FOI – premarketing studies suggest a wide margin of safety.

Adverse Drug Effect cumulative summary: (see pages 580 to 610) Convulsions are listed as the 7th most commonly reported adverse sign with 801 reports (cf most common reported sign of vomiting with 9,024 reports).

APVMA

AERP extract below – only 2 reports presented.
Veterinary Medicines – Summary of Adverse Experience, Reports 2009 for Spinosad - *Canine*

NUMBER OF REPORTS	PROBABLE	POSSIBLE
2	0	2
PRESENTING SIGN	**INCIDENCE**	
Vomiting	1	
Anorexia	1	
Ataxia	1	
Lethargy	1	
Paralysis	1	
Respiratory problems	1	
Tachycardia	1	

WINNER

Malicious freezing of a kitten

C&T No. 5352

Virginia Grice
Inverell Veterinary Clinic
32 Sweaney Street
Inverell NSW 2360
T. (02) 6721 0266
E. vkgrice@hotmail.com

Patient: 'Bones' was aptly named by her current owner who rescued her from a freezer at 4 months of age, having being put in there by a 2-year-old child. The time she had been in the freezer was unknown; however, it had been sufficiently long to cause subsequent necrosis of both ear tips, freeze her eyeballs, cause necrosis of her footpads and cause long-term visual and neurological deficits.

History: Bones was first presented to our clinic for desexing at approximately 7 months of age. Bones' rescuer had persisted with her, despite some significant persistent health issues. Despite her owner's concerted efforts to feed her up, Bones remained extremely thin, with a tiny frame approximately half that of what would be considered normal for a 7-month-old cat. My colleague noted that Bones had an ataxic gait, both ear tips were blunted, particularly the right ear tip, her foot pads were extremely sensitive and she had chronic moderate bilateral conjunctivitis with a purulent discharge but no flourescein uptake. She underwent a routine ovariohysterectomy, and was discharged with Optichlor BID.

I met Bones when she was re-presented to our clinic 15 months later. Her eye problems had continued, and she now appeared cross-eyed in addition to the chronic bilateral conjunctivitis. Her ataxia had lessened, being better described as episodic incoordination. Her frame remained thin; however, she had gained some weight. Her owner had brought her in due to a deterioration in the condition of her forelimb footpads. Closer examination revealed chronic necrosis that would require debridement.

Treatment: A general anaesthetic was administered. Both forelimb footpads had large, deep areas of scabbing and necrosis. The scabs were gently removed, and the necrotic material debrided. Solosite Gel (Smith and Nephew) was applied directly to the freshly debrided footpads, a soothing hydrogel which creates a moist wound environment that aids healing and minimises the risk of scarring. A polyester-covered absorptive cotton sheet was then applied as a primary dressing (Melolin, Smith and Nephew). The secondary layer comprised of 2-3 layers of a synthetic padding bandage to protect the wounded pads and hold the primary dressing in place (Soffban, Smith and Nephew). The tertiary layer comprised of 2 layers; the deeper layer being a conforming self adhesive bandage to compress and hold the padding layer in place (Rip Rap Lite bandaging, Rip Rap), the final layer being a harder-wearing outer self-adhesive layer to keep the bandaged area dry and secure (Askina Elastic Adhesive Bandage). This bandage was left in place for 24 hours, then removed by the owner.

Two weeks later, Bones' feet showed some improvement. Her foot pads were healing, but there were still raw areas, especially on the right forelimb. Both feet were bathed in chlorhexidine. Her owner had sourced some cat shoes which Bones had been wearing without resentment. The cat shoes had a soft lining, and Bones was walking with much less difficulty. The problems to be resolved now were: firstly, what would best resolve the footpad necrosis and, secondly, what would promote healing and skin regeneration without the expense of continual bandage changes? Her owner was currently using a topical soothing cream registered for use in humans. We decided to attempt a therapeutic trial using Rose Hip Oil.

For the next 2 - 3 weeks on a daily basis, Bones' owner placed a few drops of Rose Hip Oil on fresh gauze swabs and placed them inside the cat shoes. Bones' feet improved remarkably quickly, and within 3 weeks, both footpads were completely healed and she no longer required the shoes to walk comfortably. This was extremely satisfying, albeit unexpected, given that footpad discomfort had been part of Bones' life for such a long time. Even more satisfying was that healing was long-term, and at a recent check up 2½ years later, Bones' feet have remained healthy and caused her no pain in the intervening period.

Discussion… and many questions!

Cases of frostbite and associated hypothermia are not unknown in clinical practice. Common causes in companion animals are accidental exposure to a cold environment ('I thought YOU brought the cat in for the night… '- type scenarios) or failure to provide adequate shelter during extreme weather conditions.

When the rectal temperature lowers to 27.8°C or less, dogs and cats lose the ability to return their body temperature to normal, but with treatment they may survive. The extent of the injuries to body tissues varies with the actual temperature of the body and the duration of the hypothermic condition. Animals may survive conditions of mild hypothermia (30-32°C) for 24 to 36 hours. With increasing severity of hypothermia, survival time is considerably shortened. Under conditions of moderate hypothermia (22-25°C) most animals succumb after 4 to 24 hours. The maximum survival time from severe hyperthermia (less than 15 degrees Celcius) is 6 hours.

Frostbite (avascular necrosis) can occur after exposure to a cold environment where body temperature drops below 34°C. Any exposed body part may then become frostbitten due to destruction of superficial tissues secondary to inadequate or absent blood flow through small surface blood vessels. As expected, frostbite occurs more commonly in very young or poorly nourished animals. The most commonly affected body areas in dogs and cats are the pinnae of the ears, the tail, the external genitalia and the footpads[1].

It is unknown how long Bones was subjected to the sub-zero conditions within the freezer. Being a kitten at the time would have rendered her more vulnerable to the effects of frostbite, and certainly her injuries bore that out.

In attempting to treat Bones, it became clear that several veterinary texts contain excellent information on the acute management of hypothermia and frostbite[1], but are largely silent on the management of any of the severe chronic sequelae of these conditions. This was problematic in Bones' case, as her chronic problems were many, namely:-

1. Poor growth, both bone structure and muscle mass
2. Visual deficits, namely bilateral fixed dilated pupils (mydriasis) with greatly reduced pupillary light response (PLR)
3. Chronic conjunctivitis and dry eye
4. Ataxia and episodic incoordination
5. Hypersensitive footpads, with an episode of severe footpad necrosis
6. Loss of tips of ear pinnae

In my opinion, it is entirely reasonable to attribute all of Bones' problems to her unfortunate experience. (Readers may wonder why loss of the tail was not listed amongst Bones' problems. The reason for this was that she already only had 1/3rd of her tail due to a prior 'accident' caused by the same child). With several of her conditions, namely the loss of her ear tips and footpads, causation is easily understood and well documented. The remaining sequelae however, are far less easily understood and notably absent from veterinary discussion.

Bones' visual deficits remain to the present time, 4 years after her experience. A normal PLR requires some normal retina, normal optic nerve, midbrain connections with nuclei of CNIII, intact CNIII efferents and normal papillary sphincter muscle function[2]. It is a matter of conjecture as to the lesional focus, or foci. Presumably deeper structures would have been more protected, making the more superficially placed retina potentially more vulnerable to damage secondary to vascular disturbances. Bones also had chronic dry eye and conjunctivitis, perhaps due to avascular necrosis of the conjunctival blood vessels and those supplying the lacrimal gland. Interestingly, after a 2 year period, Bones' conjunctivitis resolved and her tear film returned, suggesting that regeneration had occurred. Neither visual nor conjunctival disturbances following frostbite appear to be documented, and hence it is difficult to propose a definite pathological mechanism[3].

Bones' general poor growth was a persistent feature, despite adequate caloric intake. As for her ocular problems, unexplained poor growth is not documented as a sequelae to severe hypothermia. A search of human medical literature revealed that growth hormone deficiency can occur secondary to pituitary damage due to ischaemic or haemorrhagic infaction[4]. Given the fact that Bones also exhibited chronic generalized ataxia, I have wondered if she experienced a form of vascular accident due to severe hypotension and/or cerebral microvascular damage. Clearly, such hypotheses are merely conjectural – there was neither evidence of, nor investigation into other pituitary-dependant endocrinopathies. It is, however, fascinating to me that as Bones' ataxia gradually resolved over a 2 year period, so too her growth rate improved. Now, at 4-years-old she weighs a respectable 3kg.

I remain unsure as to what caused the deterioration of her footpads – in mid-summer – some 18 months post-freezing. By this stage, she was starting to grow well, and it is possible that her compromised vasculature could not support new tissue growth adequately. Although I have no definite proof, I am convinced that Bones' recovery was greatly aided by the twice-daily application of Rose Hip Oil. Rose Hip Oil can prove to be a remarkable ally in aiding repair of scarred, burnt or traumatised human skin, even years after the inciting event[5, 6, 7].

The action of Rose Hip Oil in helping to regenerate damaged tissues was originally attributed to its high content of unsaturated essential fatty acids, namely oleic acid (15-20%), linoleic acid (44-50%) and linolenic acid (30-35%)[5, 6, 8]. These acids are important in maintaining healthy skin tissue as they are components of cell membranes, but were found to not be directly responsible for tissue regeneration. In the late 1980s, a team of Chilean researchers identified trans-retinoic acid – a derivative of Vitamin A – as the component responsible for these effects[6]. Trans-retinoic acid is marketed as Tretinoin, and is used to treat a variety of dermatological disorders due to its capacity to induce rapid cellular turnover and increased collagen synthesis[9]. As with many alternative therapies, Rose Hip Oil is variably accepted within the field of human dermatology. Opinions regarding Rose Hip Oil's healing properties range from overwhelmingly effusive to non-committal.

Clearly, usage of Rose Hip Oil in a veterinary setting is an 'off-label' treatment, and is only ever appropriate once the inciting cause of skin damage has been identified and appropriate adjunctive treatment given. From Bones' owners' perspective, with few if any affordable treatment options available, a $19.95 bottle of Rose Hip Oil was an acceptable investment and one that proved to provide long-term benefits.

From my perspective, should a case of frostbite sadly come my way again, I will certainly consider using Rose Hip Oil as an integral part of therapy. ▶

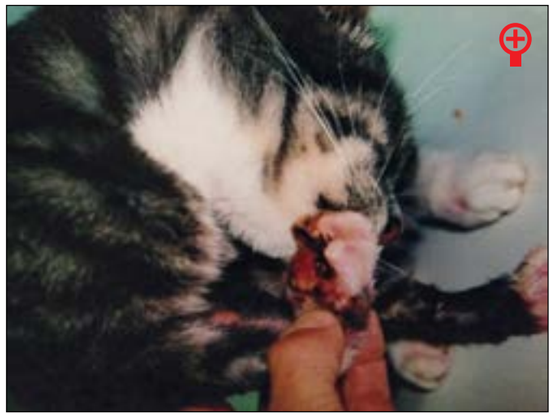


Figure 1. Chronic footpad necrosis at 22-months-of-age, 18 months post-freezing.

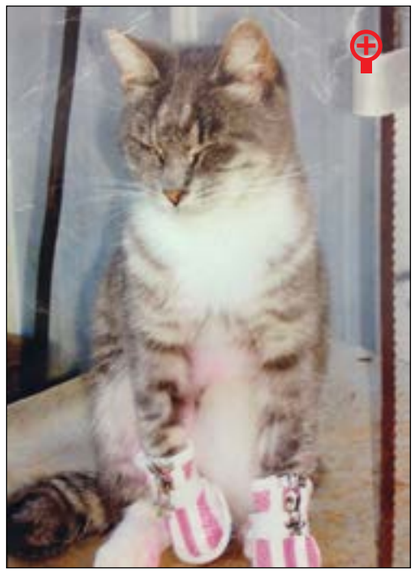


Figure 2.. Bones-in-Boots.

Authors' views are not necessarily those of the CVE
Back to TOC page

Figure 3. Rose Hip Oil – a cost-effective ally.

Figure 4. Footpads healed after less than a month of treatment with Rose Hip Oil.

References

1. *Emergency Procedures for the Small Animal Veterinarian.* 2nd Edition. Plunkett, S.J. WB Sunders 2001. Pp 76-78.
2. Ophthalmic examination. Bayer accelerate online course. www.bayeraccelerate.com
3. Pers comm. Mike Bernays. www.animaleyeservices.com
4. Growth Hormone deficiency. www.wikipedia.com
5. Rose Hip Oil – A Natural Skin Regenerator. www.biodistributors.com
6. Rose Hip Oil – Rosa Affinis Rubiginosa. A review of findings from *Contributions to Identification and Application of Active Components contained in Rosa Aff. Rubiginosa.* Pareja, B. and Kehl, H. 1988. Faculty of Chemistry and Pharmacology at the University of Concepcion (Chile). http://mettamassagetherapy.com
7. Tratamiento de las' ulceras cutaneas con aceite de rosa de mosqeta. Gimenez, J.C.M., Beuno, J., Navas, J and Camacho, *F. Med.Cut.* I.L.A. Vol XVIII/1990.
8. Rosehip oil for Skin Rejuvenation. www.uncleharrys.com
9. How To Use Tretinoin (Retin A). www.dermatology.about.com

'Dug' the dog

Bones is my second patient who has shown a remarkable improvement following treatment with Rose Hip Oil. The first was my own dog 'Dug', a desexed male black Labrador whom I acquired as a 2½-year-old from an animal refuge. Dug had been surrendered 3 weeks previously following a very severe skin reaction possibly to injectable amoxicillin-clavunate.

Dug had been admitted to a local veterinary hospital 2 months earlier with vomiting, diarrhea, recumbency and injected mucous membranes. Following a negative Parvovirus test, he was placed on i/v Hartmann's solution, and an exploratory laparotomy was performed due to a strong history of indiscriminate eating, particularly sticks (as Dug's new owner, this decision was entirely justified). He was premedicated with ACP/methone, induced with Alfaxan and maintained on isoflurane. No foreign body was found, and routine closure performed. He was given 1.5mL Noroclav s/c. Importantly, excessive localised heat caused by a heating pad injury was also categorically ruled out as a possible cause.

The next morning, Dug was an 'unhappy dog.' A patch of hair along his midline had come away, leaving the skin underneath red and moist. Over the course of the day, he developed serous ooze extending from the base of the skull to the tail base and 6cm either side of the midline. Hair epilated easily with epithelial loss, the skin appearing oedematous, erythematous and very painful. Drawing on his considerable experience, the treating veterinarian diagnosed Dug with acute cutaneous vasculitis, most likely triggered by a severe reaction to Noroclav. He had seen such a reaction once before – proven in that particular case by recurrence with repeated exposure to the antibiotic. All antibiotic therapy was ceased, and Dug was discharged on meloxicam. Six weeks later, his owners returned for a check-up. Dug had a 25 x 3cm unhealed area along the caudal midline with pruritic eschars, and pink scars at the cranial extremity. The owners were advised to use Rose Hip Oil, but surrendered Dug to the refuge that day.

When I took Dug on some 2 months after the inciting event, he had a pink scar 1-3cm wide and 30cm long along his caudal midline, with a 10cm unhealed raw strip at the caudal extremity of the scar. Having read his previous treating vet's advice, I began treating the area with Rose Hip Oil BID. The rate of healing was surprising, and the bright pink scar narrowed daily as the wound re-pigmented from the lateral margins. The raw area also contracted, such that after 4½ weeks, the skin was completely healed, just hairless.

For interest, I discussed Dug's condition with Linda Vogelnest, who made the comment that it was a 'spectacularly large area affected.' In her experience, the size of the affected area was less consistent with injectable Noroclav reaction, which is usually more localised, or at least symmetrical around the lesion. She suggested that the sloughing could be due to regional vasculitis, and that given that vasculitis can be secondary to so many things, only definitive re-exposure and recurrence would allow identification of the exact cause.

Causes include:-

- Infectious vasculitis
  - bacterial, fungal, viral, rickettsial, parasitic
- Non-infectious vasculitis
  - exogenous antigens such as food additives or drugs
  - endogenous antigens eg neoplasia or connective tissue diseases
  - excessive localized heat, pressure or contact with caustic agents
- Idiopathic vasculitis
  - most common (See references 1-4 below for a full discussion of pathophysiology, diagnosis and treatment of canine cutaneous vasculitis).

Needless to say, I have not, and do not, intend to re-expose Dug to Noroclav, ACP, methone or Alfaxan if at all possible. He has remained healthy since, and I suspect that he will join the 50% of dogs and cats who experience acute cutaneous vasculitis for whom the exact cause remains elusive.

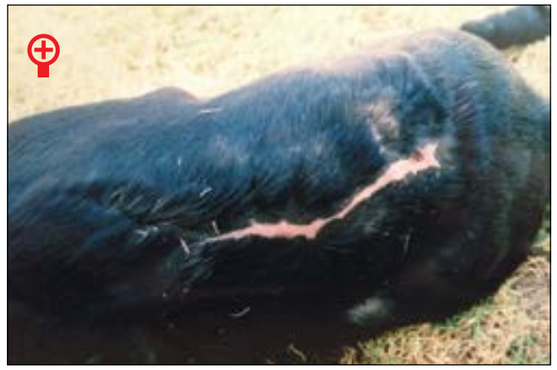


Figure 1. After 1 week of treatment with Rose Hip Oil. The pink scar had halved in width.

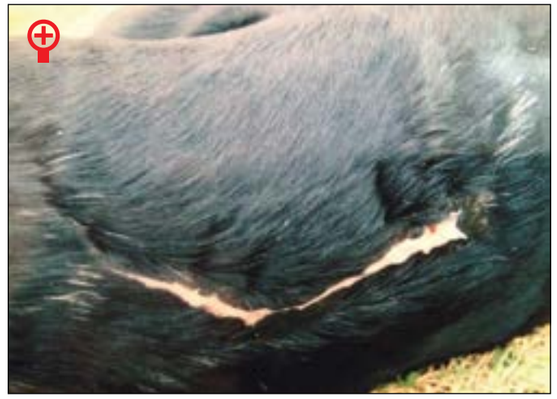


Figure 2. 2 weeks. Going...

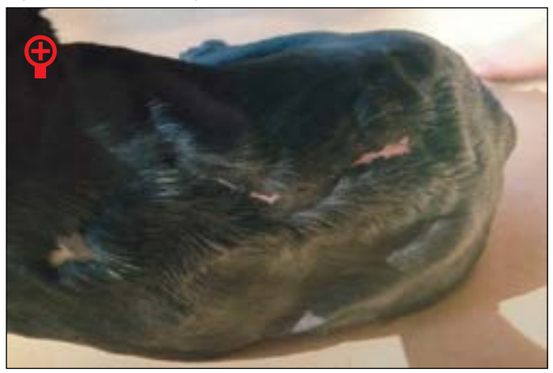


Figure 3. 3 weeks. Going...

Figure 4. 4½ weeks. Gone!

References

1. Cutaneous vasculitis and vasculopathy. Affolter, V.K. www.vin.com
2. *Diagnosis and management of canine cutaneous vasculitis.* Bloom, P.B. 80th Western Veterinary Conference.
3. Vasculitis. www.dermatologyforanimals.com
4. A retrospective study of canine and feline cutaneous vasculitis. Nichols, P.R., Morris, D.O., and Beale, K.M. http://onlinelibrary.wiley.com

Flea-Away spray

C&T No. 5353

Martin Whitehead
Chipping Norton Veterinary Hospital
Albion Street
Chipping Norton, Oxfordshire OX7 5BN, UK
E. martin.whitehead@virgin.net

C&T readers might be amused by the following anecdote...

A lady came in to complain that the 'Flea Away' spray she had bought over the counter did not work – she'd used it several times but the cat still had lots of fleas. Our receptionist explained that we do not sell a product called 'Flea Away' and, after determining that the owner was not using Frontline spray or spraying the cat with an insecticidal house spray, investigated further – to find that she was spraying the cat with Feliway! Presumably the cat was pretty relaxed about its flea problem….

Back to TOC page

MAJOR WINNER

Thoracodorsal axial pattern flap & novel non commercial closed suction drain for the reconstruction of chronic non healing axillary wounds in cats

C&T No. 5354

Matthew Tay BVSc MACVSc (small animal surgery)
Principal Milton Village Vet, NSW
139 Princes Highway
Milton, NSW 2538
T. 02 4454 4949
E. matthewtay@me.com

Matthew Tay graduated from Sydney Uni in 1991, did time at Sydney Animal Hospitals before completing his membership of the Australian College of Veterinary Scientists in small animal surgery in 2003 and 'escaping' to the South Coast in 2005 with wife Carrie Hawthorn and three children in tow. Matt loves all aspects of small animal practice and when not working can either be found in the left hander at South Narrawallee or chasing 'flatties' in his beloved Hobie.

Introduction

Chronic non healing axillary wounds of the cats are usually the result of forelimb collar entrapment with secondary vascular occlusion and necrosis. These wounds often involve the elbow fold and can be extremely difficult to get to heal with conventional surgical closure. This article describes the use of the thoracodorsal axial pattern flap, in combination with a novel non commercial closed suction drainage system, to successfully close chronic non healing axillary wounds in the cat.

The Thoracodorsal Axial Pattern Flap (TAPF)

The thoracodorsal axial pattern flap was first described by Pavletic in 1981 and is based upon the cutaneous branch of the thoracodorsal artery and vein. The thoracodorsal direct cutaneous artery arborises in a dorsal direction behind the scapula. Markers for the flap are: cranial: spine of the scapula, base: shoulder depression and caudal: equidistant from the cranial border to the base (Fig1). Flap type can be peninsular or hockey stick with flap length extending to the contralateral shoulder depression (Fig 3). The flap is elevated below the level of the cutaneous trunci muscle which is the panniculus muscle in this region, beginning at the end of the flap. Division of the opposite cutaneous artery and vein is advised. As with all axial pattern flaps, great care is taken not to damage any section of the vasculature, especially at the base, which is generally easily visible within the flap but transillumination with a good focal light source may assist. A good rule of thumb is to blunt dissect around and outside any vessel within the subcutaneous region if possible or to double ligate and divide otherwise.

Surgical Technique

Visualise and plan flap and bridging incision before making any incisions using a permanent marker. Hanging aseptic technique with large area clipped (bilateral shoulders if expect will need long flap). Drape and wrap foot with sterile hand towel and VetWrap. Minimally debride skin edge and superficial layer of granulation tissue of chronic wound till fresh and viable (bleeding), lavage and control hemostasis and cover with saline soaked swabs. Develop thoracodorsal axial pattern flap of requisite length. Double ligate and separate vessel at end of flap and develop from this point beneath the panniculus muscle as described by Pavletic. Take great care to preserve vessel and branches particularly near the base.

Make bridging incision from caudal incision of TAPF to wound bed. Ensure dermal depth only to avoid damaging any underlying vasculature and rotate flap in a caudal direction into axillary wound bed. Close wound edge with a subdermal/subcutaneous simple continuous (one down each side of flap) using 3/0 or 4/0 absorbable monofilament, combined with dermal closure 2/0-4/0 non absorbable in cruciate mattress. If there are areas of tension place 'far-near-near-far' tension sutures of polyamide or polypropylene in between the cruciate mattress. Do not place any subcutaneous tacking sutures as I presuppose this may accidentally occlude the arborisation of the direct cutaneous vessel. To manage dead space, prior to closing donor site, place a closed suction drain that runs the length of the flap and crosses the donor site dorso-cranially to exit cranial to the shoulder in a stab incision affixed via a Chinese finger trap suture (Fig 2).

Novel Non Commercial Closed Suction Drain

Open standard giving set or extension set tubing (not micro) and 3 way stopcock onto instrument table. Cut a length approximately 50 to 70 cm long. At one end, cut small holes along both sides of tubing for length of flap and leave the other end intact (Fig 4). Place prior to donor site closure by creating 5-10cm access channel with hemostats pushed craniodorsally through subcutaneous fat; tent skin and make small stab incision is just wide enough to push the tip of haemostat through, grasp holed end of catheter and pull it back through. Position the holed section of the tube so that it runs the entire length of the rotated TAPF dead space with a small U-turn at the end of the flap. Ensure all cut holes are buried within the subcutaneous region and close drain exit site with Chinese finger trap suture. Close donor site in 2 layers. After closure, attach 3 way stopcock to end of tubing; if stiffer plastic, expand end with hemostats and apply negative pressure with 10-20 mL syringe and remove accumulated air and fluid and expel and repeat until a 15-20mL plunger pull of negative pressure is achieved in tubing and then close valve on stopcock and remove syringe. This allows ongoing drainage to occur and the tubing extending from the skin will fill with fluid. The drainage can be repeated as required. Remove 3-5 days post operatively when drainage is less than 1mL/kg/day.

Post Operative

An Elizabethan collar is applied until the sutures are taken out which can be staged over 2 to 3 days. Cats must be hospitalised until the drain is out and then small-room-restricted at home until sutures out. Prophylactic IV antibiotics (cephazolin) as well as a course of broad spectrum antibiotics (potentiated amoxil + fluroquinolone) pre (4-5 days) and post operative (7-12 days) are given. Pain relief should include peri-operative opiates then meloxicam and/or temgesic post-operatively. The axillary region of the wound can become quite moist and gentle bathing with 0.05% chlorhex +/- bactroban ointment is advised.

Discussion

This is the first description I could find for the combination of a closed suction drain and TAPF repair which has good results for the repair of chronic non healing axillary wounds in the cat (admittedly n is only 3 to date). The TAPF was first described by Paveletic[1] in 1981 and Remedios[2] did further work on cats specifically in 1989. Remedios described how the flap could reach to the carpus in cats (peninsular extending to contralateral shoulder depression).

Lascelles[3] has described a prospective study combining an omental pedicle flap with a TAPF and claimed it was the first description of a consistently successful 1-step technique for chronic non healing axillary wounds in the cat. Gray[4] described an omental pedicle flap and omocervical axial pattern flap to surgically close chronic axillary wounds in the cat which is helpful when the extent of the axillary wound affects the exit point of the direct cutaneous artery of the TAPF.

Brinkley[5] hypothesises that since collar entrapment injuries result in both an axillary wound as well as disruption of the normal anatomy of the elbow skin fold, that the reconstruction of the elbow fold before closing the wound may increase the chance of a successful first time surgical repair. Hunt *et al*[6] suggested that the elbow fold may indeed be an axial pattern flap supplied by the lateral thoracic artery. As with many regions of the body that fail to heal well (e.g. distal radial fractures in small breed dogs) there is often a blood supply deficit discovered consequently that neatly explains the problem.

I propose that the TAPF combined with closed suction drainage elegantly solves the reconstruction of the elbow fold and blood supply issue without the need for a second concurrent surgical procedure to harvest an omental pedicle flap and the increased surgical experience required, time of surgery and risk of complications this entails.

Commercial and non commercial active/closed suction/vacuum drains have been well described by Pavletic[7]. Previous drainage devices attached to embedded tubing tend to be too bulky for cats (Jackson Pratt 100mL 'grenade' or 10-60mL syringe with lock pin). The novel non-commercial closed suction drain described is simple, cheap and well tolerated by cats. This is partly due to the lightness of the apparatus which the cats seem quite happy to drag around the cage space behind them. Alternatively, it could easily be looped and affixed to the collar.

A 70cm length of standard IV giving set tubing holds approximately 5mL of fluid. With half the length fenestrated and the wound already drained of air/fluid, a standard 20mL syringe with plunger pulled to 15mL mark and the 3-way stopcock closed will cause the tube to fill with approximately 2-3 mL of fluid/air before the negative pressure resolves. The clear tubing containing serosanginous fluid provides a visual alert that the wound requires further drainage which should be performed as regularly as required to maintain negative pressure within the wound. Drainage is a simple, well tolerated, one person procedure.

[1]Pavletic M M: Canine Axial Pattern Flaps, using the omocervical, thoracodorsal and deep circumflex iliac direct cutaneous arteries. *American Journal of Vet Research* 42(3):391-406, 1981
[2]Remedios A M: Thoracodorsal and Caudal Superficial Epigastric Axial pattern Flaps in Cats. *Vet Surg* 18(5):380-385, 1989
[3]Lascelles B D: Combines Omental Pedicle Grafts and Thoracodorsal Axial Pattern Flaps for the Reconstruction of Chronic Nonhealing Axillary Wounds in Cats. *Vet Surg* 30(4):380-385, 2001
[4]Gray M J:Chronic Axillary Wound Repair in a Cat with Omentalisation and Omocervical Skin Flap:*Journal of Small Animal Practice* 46(10):499-503, 2005
[5]Brinkley: Succesful Closure of Feline Axillary Wounds by Reconstruction of the Elbow Skin Fold. *Journal of Small Animal Practice* 48(2):111-115, 2007
[6]Hunt G B et al Skin-Fold Advancement Flaps for Closing Large Proximal Limb and Trunk Defects in Dogs and Cats. *Vet Surg* 30(5):440-448, 2001
[7]Pavletic M M *Atlas of Small Animal Wound Management and Reconstructive Surgery* 3rd p54-58 2010

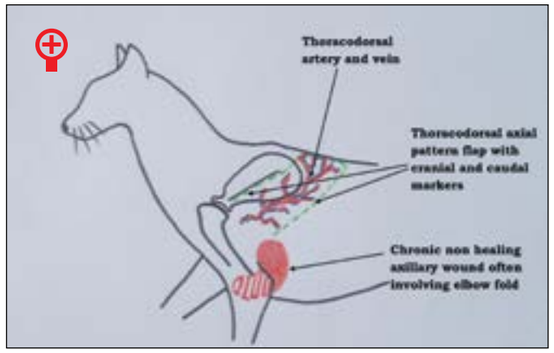


Figure. 1

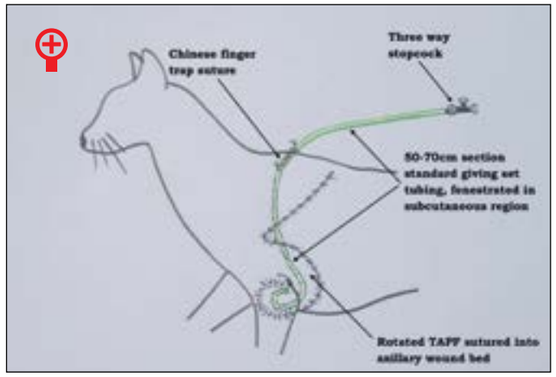


Figure. 2

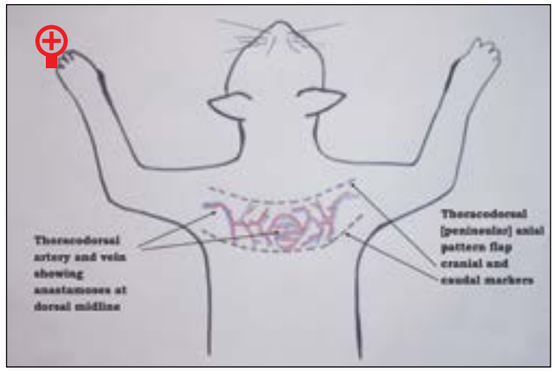


Figure. 3 (above)

Figure. 4 (left)

Fannia spp for feline perineal itch

C&T No. 5355

Aine Seavers
Oak Flats Vet Clinic
58A Central Avenue
Oak Flats NSW 2529
E. reception@oakflatsvet.com.au

Three 9-year-old feral cats live with their long suffering owner. She was 'gifted' them as an orphan litter without knowing what she was given and, being too soft hearted to give them away, they now live in her house, tolerate her presence and do as they please. I see them as infrequently as possible and then from a safe distance whenever possible.

Two began overgrowing in the perineal area; they are indoor cats and there had been no change of food etc.

The owner saw 'creatures' in the faeces so having had her dogs suffer recalcitrant pruritus from an undiagnosed tapeworm infestation years previously, she purchased both an oral tablet and then a topical dewormer-specific product used 14 days apart. There was partial resolution of the problem then the 'creatures' appeared in the urine – which she alerted me to – and as Australia seems to the capital of bladder worm reports I requested a sample.

Macroscopically, the 'creatures' looked like a blob of dried blood. Microscopically, it was unlike anything I had seen before but if a louse and a centipede had a love child then this is what it would look like.

I sent the sample straight off to Jan Slapeta, Associate Professor in Veterinary Parasitology at the University of Sydney, who found it to be:-

> *'larvae of lesser house fly / faecal fly (**Fannia spp**.). They are very attracted to smells and watery faeces, urine etc. Apparently they can be quite a pest in poultry houses. The larvae on their own hardly ever cause problem, but at times have been causing myiases.'*

As the cats are feral and not easily handled, mild myiasis or just fly worry might have been a trigger for the over grooming.

The owner changed their room, supplied new litter and had the house environmentally sprayed and the cats stopped grooming. It's a suburban house with no manure used in the gardens and no chickens but about 2km away there are some big vegetable gardens amongst an estate with many European immigrants who love their veggie patches so perhaps chicken manure is used there? Compost bins might be a more likely source. The flies carry lots of nasty infectious agents so not good to have around generally.

I thought I'd alert colleagues to add *Fannia spp* to the differential diagnosis of perineal/perianal pruritus in the cat.

Figure 1. **Fannia spp.** ***(Image courtesy of Jan Slapeta, Associate Professor in Veterinary Parasitology, University of Sydney)***

ISFM Forum – Cat friendly clinics

Pete Coleshaw MRCVS www.jaffavets.com 'where cool cats chill' ^~^

C&T No. 5356

Jaffa's Health Centre for Cats
52 St Francis Road
Salisbury SP1 3QS, UK
T. 01722 414298

Your biggest challenge may be changing staff attitudes and getting cats granted the importance they deserve. It is very easy to create token gestures which don't really improve kitty's welfare; for example, you can have a dedicated cat waiting area but if the receptionists and nurses pay no attention to barking dogs, or to unruly owners who allow their rottweiler to pull them over to the cat area so they can take a look and a sniff, then your efforts become irrelevant. So it needs education of staff to encourage them to politely ask these clients to respect the cat's privacy. It's not easy to do, but clients do notice when staff ignore the plight of their kitty!

'Cat friendly' also needs space – a small, shared space still means shared noise, smells and frenetic activities so shared facilities are always going to be very much second best to clients interested in cat-only facilities. The only meaningful way around this problem is scheduling cat-only appointment times. Even if this time gets invaded, it is better than nothing, but the practice must be committed to your initiative otherwise you are on a frustrating journey. But where there is a will, there will be a way!

Seeing is also believing. If your efforts become token gestures, any staff who are not fully on board will not see any benefits so it can be a bit of a vicious circle.

In terms of the consulting room, is there scope for a dedicated cat room? If not, the room must be hoovered and cleaned properly after every dog if you are to make any difference in terms of construction. The number 1 priority for me is to make sure that there are no hidey holes and preferably nothing to knock over so that you can allow the cat time to explore in safety.

In a dual-use consulting room, the rest then I think is down to cattitude (and appointment length). Comfy chairs are nice, vetbed mats to sit on the floor with the cat are easily sorted (have you seen the rubber-backed vet-bed? Non-slip – get it off ebay) but it's really all about commitment from the top downwards.

Editor's Note: *For more information, go to the International Cat Care (formerly the Feline Advisory Bureau) website – www.icatcare.org*

Age & anaesthesia

C&T No. 5357

David Moroney
40 Shotton Road
Mount Eliza VIC 3930
E. shottonm@bigpond.net.au

A frequent excuse given for not anaesthetising an animal for a procedure, commonly a dental on a mouth full of rotten teeth, is 'my vet said he's too old'. I've heard this many times over the years from new clients; my surgery nurse, who also does mobile dog washing, frequently hears the same excuse when pointing out to an owner their dog's halitosis and rotten teeth (the cause of their dog's smell, not their coat).

This unfortunately all-too-prevalent attitude is doing a major disservice to these animals and their owners and, potentially, borders on negligence. Clients need to be informed and make their own decision with accurate estimation of the potential risks, as with any procedure.

All vets are familiar with the potential risks of anaesthesia in old animals, but age is not a disease; if an older animal is in good health with no obvious signs of disease on clinical exam and bloods, then there is absolutely no reason not to proceed with general anaesthesia, provided the technique and facilities are adequate:-

1. Premed with acepromazine (low dose) and opioid (methadone or buprenorphine)
2. Rapid knockdown with propofol or alfaxan or similar.
3. Immediate intubation and maintenance with isoflurane.
4. IVF @ 10 mg/kg/hr.
5. Pulse oximetry monitoring.
6. Close observation of the animal during and after the anaesthetic.
7. Post operative analgesia

Humans of all ages are frequently anaesthetised safely for major surgery and with current protocols, deaths are rare. The same holds true for animals: in practice (despite the theoretical risks), old animals rarely die. The rare anaesthetic deaths in practice often occur unexpectedly in a young, healthy animal in for a routine procedure or are due to overdose or poor monitoring.

The risk of anaesthesia always needs to be weighed against the benefit. In the common case of dentals, the benefits are enormous – clients often say 'he's like a new pup, he's happy, new lease on life' etc. Their pain is gone, (severe periodontal disease [POD]is very painful) and also the foul infection associated with severe POD which can have effects on kidney, liver and heart. The risks are extremely small in practice. There are far greater risks for the animal's health by not doing the dental, and humane issues should be considered.

I've noticed that new graduates are uniformly apprehensive about 2 things: anaesthesia and using corticosteroids. It was the same in the dark ages when I graduated. However, with modern drugs and equipment (compared with the early 1970s when we used I/V pentobarbitone and gas anaesthesia was not universal) and some experience, there is no need to lack the confidence to perform general anaesthesia, particularly for experienced vets.

Similarly, medical conditions such as renal or hepatic disease, diabetes, congestive heart failure etc are not contraindications, in practice, of general anaesthesia. If an animal is compensated and stable when awake, chances are it will be the same when under general anaesthetic. (This 'theory' has been confirmed by long experience.) Anaesthetic protocols can be altered to allow safe anaesthesia. How many times do we do anaesthetics on unstable animals as a life-saving measure and they mostly survive?

Once you've done a few anaesthetics on geriatrics and seen the positive results, confidence will increase, and hopefully this outdated attitude will disappear and these animals will get the treatment they deserve.

QUESTION OF THE ISSUE

From the ISFM Forum

Cat pooping in the owner's bed

C&T No. 5358

My friend's housemate's cat started recently to poop on his owner's bed. He did my friend's bed for a period of time a couple of months ago (he couldn't figure out a trigger), so the bedroom door has since been kept shut at all times. After a couple of weeks he had been able to leave my door open without worrying about the kitty pooping in his bed.

The owner went to the US last week and kitty pooped on his bed at least 5 or 6 times (once when his partner was asleep IN it!). They thought maybe it was separation anxiety, but now the owner is back and the cat has done it 3 days in a row.

The cat has 2 kitty litter trays that he also uses, and they keep them clean so it's definitely not the case that he **has** to use the bed.

Qs: Is this simply some stress-related behaviour? And if so, would Feliway / PetRemedy type things help? Or is there some underlying problem that needs to be addressed?

Any advice on what to do would be appreciated!

NOTE: Participating in the ISFM forum is a major ISFM membership benefit.

Authors' views are not necessarily those of the CVE

Back to TOC page

Pimobendan – a double edged sword?

C&T No. 5359

Dr Yuan-pei Lien
Healer Animal Clinic
G/F, 17 Apliu Street, Kowloon
Hong Kong
T. 852-23970800
E. gogovet101@yahoo.com.hk

The following crazy idea has been whirling around in my mind for about 1 year: Since the introduction of pimobendan, patients with mitral valve disease enjoy much better quality of life and life span seems longer.

However, at the same time, it seems that there are more and more pulmonary hypertension cases being found. Could pimobendan be relevant to this – even partially? Few side effects from pimobendan have been reported until now.

Pimobendan increases the strength of the systole and then the cardiac output. Theoretically, at the same time it increases the mitral regurgitation and then rises up pulmonary venous pressure! High pulmonary venous pressure is one of those factors predisposing or contributing to pulmonary hypertension.

Growing knowledge and the popularity of ultrasound equipment, of course, makes it easier to diagnose pulmonary hypertension.

So did we do anything 'harmful' by using pimobendan? Even it is 1 out of 10, or 1 out of hundreds, or one out of thousands of cases!

For general practitioners like me, this 'hunch' about the increasing numbers of pulmonary hypertension cases after patients have been administered pimobendan may not be reliable and could of course be entirely subjective.

So, may I suggest that any referring centres or teaching hospitals (which should use less bias in diagnosing these instances due to their diagnostic skills and state of the art equipment) test patients with both degenerative mitral valve disease and pulmonary hypertension before and after the introduction of pimobendan to see if there is any statistical difference. If the case numbers increase after the introduction of pimobendan, it probably means there is some basis to my 'hunch' about pimobendan and until we determine what the cause is, vets should be more cautious when prescribing pimobendan!

Remember the axiom – 'first, do no harm'.

***Editor's Note** – Lien's C&T was interesting and speculative and – in true C&T spirit – provides an opportunity for members/ readers to learn more about this topic from the CVE's Cardiorespiratory Medicine Distance Education tutors, Niek and Nick, published below.*

Invited Commentary courtesy of:

Niek Beijerink DVM MS PhD Dipl. ECVIM-CA (Cardiology)
Senior Lecturer – Small Animal Cardiology Specialist
Faculty of Veterinary Science, The University of Sydney

Dr Lien raises an interesting topic but I believe the rationale of the article is most likely incorrect. I agree with the statement that pulmonary hypertension is seen more often than let's say 10 years ago, but it is far more likely this is because of better understanding/awareness of the syndrome (thus we suddenly recognise this symptom), and better diagnostic tools. As mentioned by Dr Lien, dogs with mitral valve disease nowadays live longer than previously (thanks to heart failure medication, including pimobendan), so there is more time (or a greater risk) to develop some other symptoms of the disease, in this case secondary pulmonary hypertension (the heart failure medication does not cure the disease) as the disease progresses. Indeed, secondary pulmonary hypertension is relatively common, affecting up to 72% of dogs with end-stage acquired mitral valve disease (Serres et al. 2006). If anything can be said about pimobendan and pulmonary hypertension, it is most likely that this phospodiesterase inhibitor does DECREASE pulmonary vascular resistance, and drops the severity of pulmonary hypertension, as evidenced in recent literature (Atkinson et al., 2009).

References

1 Serres FJ, Chetboul V, Tissier R, Carlos Sampedrano C, Gouni V, Nicolle AP, Pouchelon JL. Doppler echocardiography-derived evidence of pulmonary arterial hypertension in dogs with degenerative mitral valve disease: 86 cases (2001-2005). *JAVMA* 2006;229:1772-8.

ANIMALS AND METHODS: Retrospective case series of 617 dogs examined from 2001 to 2005 with mitral valve disease in for International Small Animal Cardiac Health Council classes I to III.

MAIN RESULTS: 86 (13.9%) dogs with MVD had a diagnosis of pulmonary arterial hypertension. Severity and prevalence of pulmonary hypertension increased with severity of the disease (3.0%, 16.9%, 26.7%, and 72.2% prevalences for ISACHC classes Ia, Ib, II, and III, respectively).

2 Atkinson KJ, Fine DM, Thombs LA, Gorelick JJ, Durham HE. Evaluation of pimobendan and N-terminal probrain natriuretic peptide in the treatment of pulmonary hypertension secondary to degenerative mitral valve disease in dogs. *JVIM* 2009;23:1190-6.

ANIMALS AND METHODS: Prospective short-term, double-blinded, crossover design, with a long-term, open-label component. Short term, the 10 dogs with peak tricuspid regurgitant flow velocity (TRFV) >or=3.5 m/s were randomly allocated to receive either placebo or pimobendan (0.18-0.3 mg/kg PO q12 h) for 14 days. After a 1-week washout, they received the alternative treatment for 14 days, followed by pimobendan open-label for 8 weeks.

MAIN RESULTS: Short-term comparison: peak TRFV decreased in all dogs on pimobendan compared with placebo from a median of 4.40 (range, 3.2-5.6) to 3.75 (range, 2.4-4.8) m/s (P < .0001). In the long-term comparisons, peak TRFV decreased in all dogs from a median of 4.28 (range, 3.5-5.7) to 3.52 (range, 2.4-5.0) m/s (P < .0001).

FUN LINK

http://www.dailymail.co.uk/news/article-2313439/Disabled-fish-swim-right-way-owner-makes-LIFE-JACKET-stop-sinking-tank.html

Invited Commentary courtesy of:

Nick Russell BVSc MVS FANZCVS (Medicine) DACVIM (Cardiology)
Advanced Veterinary Care Center
15926 Hawthorne Blvd
Lawndale CA 90260
USA

Dr Lien's observations have some merit, but there are many factors to consider with pulmonary hypertension (PH).

PH is a condition with many possible underlying causes. The most common causes in small animals depend on the specific breeds and geographic location, and include:-

1. Increased pulmonary venous pressure - usually due to chronic left-sided congestive heart failure (CHF)
2. Chronic bronchopulmonary diseases - especially chronic bronchitis, pulmonary fibrosis, feline lower airway disease.
3. Heartworm disease
4. Left-to-right shunting defects
5. Pulmonary thromboembolism

Cases of PH are therefore not created equally. Similarly, the severity of PH influences the signs and need for treatment. PH is usually moderate-to-severe before animals are symptomatic from the PH; however, they may be clinical from their underlying disease that is causing the PH. Animals with mild PH are rarely symptomatic from the PH. Fortunately, our awareness and ability to detect PH has been significantly improved by the advancement of non-invasive diagnostic ultrasound.

Regarding your concern of pimobendan specifically causing PH in dogs, then I assume that you are using pimobendan for the treatment of CHF from chronic myxomatous valve disease (CMVD) or dilated cardiomyopathy. As well as the positive inotropic effects on the myocardium, pimobendan has a vasodilatory mechanism via inhibition of vascular phophodiesterase (isoenzymes types III & V) and smooth muscle relaxation. At a hemodynamic level, this lowers vascular resistance, and pimobendan has been shown to decrease left heart filling pressures, decrease pulmonary arterial vascular resistance, and increase indices of cardiac output in several species, and models of CHF. Clinically, this transpires in dogs with CHF due to CMVD treated with pimobendan, having longer survival times and improved quality of life scores.

Chronic increased left heart filling and pulmonary venous filling pressures leads to chronic remodeling of the pulmonary arterial vasculature. Some individuals are genetically predisposed to severe pulmonary vascular remodeling, that may result in more severe PH. As many dogs with CHF are living longer with current treatments, some of these dogs may 'switch' from left-sided CHF, to right-sided CHF. Severe PH can be clinically debilitating, but if responsive to vasodilatory (e.g. sildenafil) therapy, many of these dogs can respond favorably for prolonged periods.

To complicate matters further, a moderate-severe degree of PH can actually be beneficial for some case of CMVD with severe mitral regurgitation +/- left-sided CHF. Sometimes the increased pulmonary arterial (pre-capillary) resistance can be high enough to 'limit' the pulmonary flow to the left heart, thereby keeping filling pressures and pulmonary venous pressures lower, reducing the likelihood of developing pulmonary oedema, and reducing diuretic requirement.

The ACVIM consensus statement on CVMD in dogs does NOT recommend the use of pimobendan in dogs prior to the onset of CHF. In these cases it may be prudent to 'first do no harm', until further studies clarify the situation.

Snake Bite

C&T No. 5360

Mark Hynes[1] & Robert Johnson[2]

[1]Kingston Animal Hospital
1 Freeman Street, Kingston TAS 7050
T. (03) 6229 5900
E. hynesmark1@hotmail.com

[2]Robert Johnson BVSc MANZCVS (Feline Medicine)
CertZooMed BA CMAVA
South Penrith Veterinary Clinic
126 Stafford Street, Penrith NSW 2750
T. +612 4721 4796
F. +612 4732 5359
M. + 61 414518188
E. clinic@reptilevet.com.au
www.reptilevet.com.au

The following C&T came about through email exchanges between Mark Hynes, Richard Malik (CVE) and Robert Johnson. We hope this article generates a response from readers, in particular to the question:

Have you seen or heard of cats being presented with increasing (in intensity and duration) intermittent muscular 'convulsions' post snake bite?

Please email your responses to: elisabeth.churchward@sydney.edu.au. Thank you.

Email from Mark Hynes to Richard Malik, CVE

Do you have any CVE resources or have a reference for good information on snake bite in cats and dogs (particularly TAS/VIC snakes)?

I believe there is a lot of variability in the presentation of a snake bitten cat.

I saw a cat that was rushed in because it was suspected of being bitten by a snake. It had acute tachypnoea and convulsions. Its oxygen saturation was normal despite the tachypnoea, it had normal pupillary light reflexes with mild mydriasis, chest sounds were clear, blood glucose 3.4mmol/L. Its major problem was intermittent 'convulsions' of the whole body but mainly the hind legs (the cat was hard to control). These convulsions were escalating and I gave methocarbamol IV which helped the cat a lot (at least from my perspective). Its oxygen saturations remained normal post methocarbamol. We sent the cat to our afterhours vets who ran creatine kinase activity which was through the roof (to be expected with either snake bite or convulsions of other origin?) and it stayed on twice maintenance IV fluids.

A vet down here said that Copperhead venom does not cause an increased activated coagulation time (ACT) in envenomated cats. Have you heard of this?

The most likely explanation for its illness was snake bite as nearly every tremorogenic toxin was not accessible to the patient.

Have you seen or heard of cats being presented with INCREASING (in intensity and duration) intermittent muscular 'convulsions' post snake bite?

Response courtesy of Robert Johnson:

Regarding the email from Dr Mark Hynes, a cursory look at the literature does not highlight epilepsy or seizuring as a sign of snake bite envenomation in cats (Hill and Campbell 1978; Barr, 1984). However, epilepsy has been reported in human victims (Sutherland, 1983). Seizuring was described in a woman bitten by a red bellied black snake (*Pseudechis porphyriacus*) and in a 12-year-old girl bitten by a tiger snake (*Notechis ater accidentalis*). In my experience, I have not seen seizures in envenomated cats. In this particular case the history, initial clinical signs and a high CK are supportive of a diagnosis of snake bite but I suspect some other aetiology due to the reported fitting. By the way, in the case described by Dr Hynes, was a venom detection test carried out (much more useful on urine than blood) and was the cat treated with antivenom?

References:

Barr SC. Clinical features therapy and epidemiology of tiger snake bite in dogs and cats. *Aust Vet J* 1984;61(7):208-212.

Sutherland SK. *Australian Animal Toxins: The creatures, their toxins and care of the poisoned patient.* Oxford University Press, Oxford. 1983.

Hill FWG & Campbell T, Snake bite in cats *Aust Vet J* 1978;54:437-439.

Response from Mark Hynes:

Thanks Robert – interesting information that you bring forward with this case!

My main initial query was whether cats can have a normal ACT while being symptomatic for snake bite (Tasmania having only 3 venomous snakes: Tiger, Copperhead and White-lipped) but also whether a tremor/convulsion presentation can occur compared with flaccid paralysis (and perhaps absolving some concern in my decision to administer methocarbamol to a most likely snake bitten cat!). I had neglected to put into my email to Richard that this cat had a normal in-house ACT. I had not tested the cat with any venom detection kit. A vet at our local afterhours emergency centre stated that Copperhead envenomation may not affect an ACT in a cat (or dog). I have not spoken to this vet about where that information came from.

The cat is going well despite sensory and motor loss to the distal tail (both cutaneous and deep pain were absent on the tail tip up to 3 weeks post suspected envenomation). I assume this may be where the cat was bitten but I did not clip the fur to get a good look.

The day after initial presentation to us (and after spending the night at the afterhours emergency centre) the cat was normal just with mild mydriasis but still normal PLR, very high CK, no more convulsions, eating and drinking well, moving normally but with the above described nervous deficits to the distal tail. No antivenene was administered during the entire treatment, only IV fluids, and methocarbamol.

I have seen many dogs (down here in Tassie) ~ 30 mins post suspected snake bite with ptyalism and tremors that respond very quickly and well to antivenene, but this cat's neuromuscular presentation was quite different to that shivering/tremors that I have seen dogs get with snake envenomation (possibly the shivering tremors are the early stages of neuromuscular paralysis ensuing, causing a weakness?).

As stated previously, this cat's major presenting problems were tachypnoea and episodic convulsions (in retrospect- I think convulsions rather than tremors is the best description) but distinctly localised to the caudal half of the body (I think the cat was bitten on the tail as previously mentioned so I do not know whether this has any bearing?). I am not sure if the 'convulsions' were 'panic' or 'distress'. It was certainly not a generalised seizure but could have been a focal seizure (to the hind legs) as there appeared to be no loss of consciousness.

The 'convulsions' seemed to be elicited by handling of the caudal half of the body e.g. abdominal palpation (this was noted also by the afterhours care) but were also occurring spontaneously. The severity/duration was increasing with time up to the methocarbamol administration. The cat seemed better (as one would expect post methocarbamol), the severity of the 'convulsions' decreased and the propensity for a 'convulsion' to occur with handling decreased (but was still present for ~12 hours after slowly regressing). I wish I filmed it, but it was 'all hands on deck' as you would expect.

Note: Thanks to Glenn Shea, Senior Lecturer Vet. Anatomy & Pathology, The University of Sydney and Scott Eipper, Nature for You, for providing these great images.

Figures 1 & 2. **Australaps superbus** ***known as the lowland copperhead (courtesy of Scott Eipper)***

Figures 3 & 4. **Notechis scutatus** ***known as the Tasmanian tiger snake (courtesy of Scott Eipper)***

Figure 5. **Drysdalia coronoides** ***known as the white-lipped snake (courtesy of Glenn Shea)***

Back to TOC page

Beware the Bullrout: Small fish, big sting, excruciating pain

C&T No. 5361

R B Cope BVSc BSc.(Hon 1) PhD cGLPCP DABT ERT FACTRA
Toxicology Excellence in Risk Assessment
Cincinnati, Ohio USA
www.tera.org/
T. +1-513-542-7475
E. rhian_b_cope@yahoo.com

Introduction

The bullrout (freshwater stone fish, kroki, rock cod; *Notesthes robusta*) is a small member of the wasp fish family (tetraogidae) that are found in tidal estuaries and slow-flowing fresh waters of NSW and Queensland. Bullrouts are amongst the most common causes of fish stings in humans in these areas and are commonly found at popular swimming spots such as river crossings and causeways. They are a potential menace to pets (particularly dogs) that are allowed to play in the water in these areas.

Identification

Bullrouts (Figure 1) are relatively small fish (up to 30 cm long; but more commonly about 20 cm). Its head, body and fins are mottled dark brown, olive-brown to yellowish-tan. There are spiny ridges on the head and the dorsal fin has 15 strong spines.

Habitat and Range

Bullrouts can be found in coastal streams, rivers and creeks from the Annan River (Walker bay, just south of Cooktown) in north-eastern Queensland to the Clyde River (Bateman's bay) in NSW.

Bullrouts are tolerant of salt water, tidal esturine water and freshwater (although there is limited evidence that they require exposure to salt water in order to breed). They are commonly found in bays, estuaries and especially the lower freshwater reaches of rivers and creeks.

Bullrouts particularly seem to like living at the foot of dams and weirs, close to the limit of brackish water or tidal influence. They prefer mixed, sheltered bottom conditions with water-weeds, rocks, submerged tree roots and sunken logs.

Bullrouts have also become a popular aquarium tank species amongst enthusiasts because they are unusual and relatively easy to keep in captivity. This potentially dangerous species is available in the Australian aquarium trade from time to time. It is reputedly relatively easy to breed in salt/brackish water aquarium systems.

Venom Apparatus, Toxins and Circumstances of Poisoning

Bullrouts are well-camouflaged, sluggish and will 'stand their ground' and erect their spines when disturbed. This lack of retreat behavior is one of the reasons why dogs (with their typical exploratory and hunting/alarm behaviors) are at significant risk of being stung. Based on limited anecdotal information, bullrout stings appear to be more common in the summer. Once stepped on or bitten, the dorsal, anal or pelvic spines and associated venom glands of the bullrout detach and lodge in the contact tissues. The venom is then injected, primarily subcutaneously.

Bullrout venom is poorly characterized. The limited available information demonstrates that the active components are proteins. The venom is mildly proteolytic and mildly hemolytic. However, it does not appear to contain hyaluronidase or phospholipase A2. **Notably, the venom does not antigenically cross-react with stonefish antivenom.**

The major effect of bullrout venom is to trigger severe pain, apparently via a novel algesic protein that acts on polymodal nociceptors. While objective information from dogs (the animal most likely to be affected) is limited, human reports indicate that bullrout stings are excruciatingly painful and that without treatment, the pain is unremitting.

Toxinodrome and Diagnosis

The clinical history is most often the most useful piece of diagnostic information. A typical history generally includes at least some of the following features:

- The animal affected is usually a dog (although people swimming at the same location may also be affected).
- The dog was swimming or playing in a river and suddenly started screaming and showing other signs of extreme pain.
- There were stands of aquatic plants, submerged tree roots, submerged logs or other mixed habitat in the creek or stream-bed; or the activity was carried out near a river crossing, dam or weir wall.
- The owner may have removed the bullrout spines from the animal's feet and or mouth by the time of presentation for treatment. Removal of the spines made no difference to the level of apparent pain.

Careful clinical examination will typically demonstrate a series of one or more small 2mm long slits in the affected tissue. The slits are generally spaced about 1-1.5 cm apart. If the foot is affected, the puncture wounds are often on the plantar or palmar surfaces, often on the outer edge of the pad or on the toes. Typically there is little edema (insufficient to pit on pressure), erythema or other evidence of local inflammation. The level of apparent pain is massively disproportionate to the actual extent of physical injury and palpation **usually does not make the pain any worse.** Usually there is no evidence or regional lymph node swelling or pain. Some degree of pain-induced shock may be present. Body temperature remains normal.

Diagnosis is classically based on the history, clinical signs and possible identification of bullrout spines if they can be recovered.

Treatment

Fortunately, treatment is generally very effective and relatively simple. If the feet are involved, some immediate relief of the pain can be accomplished by placing the affected area(s) into hot water (as hot as the animal can stand without producing thermal burns).

Stonefish antivenom is not effective because it does not cross-react with the nociceptive toxin(s) of the bullrout.

Local anesthesia with injectable lignocaine* is commonly the most effective method of controlling the pain associated with bullrout stings. Volumes and doses are 'to effect' – there are no fixed rules except not to use so much that you start getting systemic effects (i.e. cardiac effects of lignocaine etc).

When the affected area involves an appendage it is particularly important not to use preparations containing adrenalin or other vasoconstrictors because of the risk of distal ischemia. Local anaesthesia can be combined with warm compresses and other supportive treatment as needed. Systemic analgesia is rarely required, but may be needed if injectable local anesthesia is insufficient. Bullrout stings in the mouth can be problematic in terms of providing adequate injectable local anesthesia and analgesia. Thus systemic analgesia may be required under these circumstances.

Although never recorded, the proteinaceous nature of the venom carries with it the possibility of anaphylaxis which must be dealt with quickly and effectively by intramuscular injection of adrenalin, possibly with follow up treatment with H1-antihistamines and management of circulatory shock.

Given the nature of the wounds associated with bullrout stings, tetanus prophylaxis should be considered in affected dogs. General supportive care may be required in some cases.

Figure 1: **Notesthes robusta*, the bullrout; an aquarium-kept example.***

* It has been suggested that in some cases it may be best to provide general anesthetic or sedation first to facilitate administration of the local anaesthetic and that bupivicane could be a better option than lignocaine because it lasts longer. However, the duration of action of lignocaine is usually sufficient since the toxin(s) effects are fairly short-lived (actual duration of action has never been studied). You can also argue for mepivicaine because of the lower tissue inflammatory properties. However, on the limited available data, lignocaine seems to be sufficient and effective; plus it is cheap and commonly available.

My understanding is that the maximum upper limit for bupivicaine is 2-3 mg/kg body weight for dogs. For lignocaine, it is 8 mg/kg body weight. For this reason, lignocaine offers an advantage: you can use more of it.

Tips from ISFM forum - Our feline DE partners

Oesophagostomy tubes

DE Feline Tutors:-

Sarah Caney, Richard Malik, Carolyn O'Brien, Andy Sparkes, Andrea Harvey, Wayne Mizon, Elise Robertson, Sheila Wills & Samantha Taylor

www.cve.edu.au/defelinemedicine

C&T No.5362

Q. I would be interested to hear ideas on what people are finding to be the most comfortable oesophagostomy tube for cats, and any information they care to add on source, size, material and supplier. (Caroline, The Oxford Cat Clinic)

A. MILA silicone ones! (Andrea Harvey, DE Feline Tutor)

A. Surgivet tubes are also very good. See: www.surgivet.com/catalog/critical-care/gastroenterology/feline-esophagostomy-tubes-silicone.html. We ordered ours from Smiths Medical. (Stephanie)

A. Our clinic LOOOOOVES the Surgivet ones. They are very soft and the kitty-collars are great. We do use a 3mm biopsy punch and core a hole in the end of the tube to make them an open-ended tube – allows easier and faster food delivery and easier to grab onto to pull through the wound. We did find some blocked when using thicker diets as they only have 2 small side ports.

The MILA ones have very sharp edges and we found they were slightly more traumatic in placing them, plus the large port on them is cumbersome. (They were our preferred O-tube for years.)

For those so inclined – the Surgivet tubes are 're-usable' if you autoclave them...? (Richard Gowan, The Cat Clinic Melbourne)

WINNER

My favourite tool!

From the DE Feline Medicine Listserve (May 2013)

C&T No. 5363

Donald Wiggins
E. donaldwiggins@hotmail.co.uk

My old boss had a technique he used to use. He would get a long piece of suture material, and tie about a 1 cm piece of swab in the middle of the suture material, leaving 'blob' of swab with 2 long ends either side. He would then feed suture material down the nostril into the pharynx, grab the end and drag the knotted blob of swab through the nasal cavity.

This often produced material for biopsy, cytology or difficult to remove grass blades.

I have been rather scared to do this, but did remove grass once that snapped when I pulled it from pharynx. I don't know if he came up with this himself!

Elise Robertson BS BVetMed MACVSc (Feline) DipABVP (Feline) MRCVS
DE Feline Tutor
E. e.robertson@felinevet.net

Sometimes you can feed a urinary catheter (widen the side holes so slightly abrasive) and advance into nasopharynx in anterograde direction (like a feeding tube). I tend to have my ▶

Back to TOC page

patients in ventral recumbency with nose pointing down. I've been able to shave enough bits of tissue with this kind of debridement (you can also suction/aspirate and flush as you debride. Look carefully on your pharyngeal pack as lymphoma pieces blend in with the white gauze pack! I find fine needle aspiration (FNA) via (or through) soft palate relatively unrewarding unless it's a crypto granuloma!

Also when doing endoscopy, take care when using spring loaded mouth gags. I will dig out the 2 recent reports suggesting an association with post general anaesthetic cortical blindness and use of these gags during dental and endoscopic procedures. The maxillary artery is the main blood supply to the feline brain and it is hypothesised that compromised blood flow/ischemia to this vessel may contribute to this event. I instead use a chopped down needle cap or dental wedge (I think sold by Kruuse) and relax the jaw every 3-5 minutes to be on safe side. The spring loaded ones tend to slowly and gradually over-extend the mouth...

Mary Jean Thomson
E. 4prism@magma.ca

We recently had a cat with a lymphoma in the naso-pharynx and attempted FNA only to have it come back as lymphoid hyperplasia. We sent samples after euthanasia and got the final diagnosis of lymphoma. Without an endoscope, and nasal flushing was unrewarding, what is the best approach for a definitive biopsy?

Elise Robertson BS BVetMed MACVSc (Feline) DipABVP (Feline) MRCVS
American Board Certified Specialist Feline Practice
Feline Vet Referrals www.felinevet.net
E. e.robertson@felinevet.net
Endoscopy Vet Referrals www.endoscopyvet.net
E. e.robertson@endoscopyvet.net
T. 01273 931 139 efax. 01273 376 932

I thought I'd take this opportunity to share my 'second' most favourite, practical, cost effective, and useful tool in feline medicine! (My favourite...clearly my endoscope(s))!

For your sneezing, retching, gagging cats... this is perfect thing for in practice use. It's a dental mirror with integrated light source = less tools in mouth and no need for enhanced dexterity! This, and a spay hook – perfect combo! It simply attaches to your otoscope/ophthalmoscope light source.

Figure 1. Straight laryngeal mirror with integrated light source.

Figure 2. With the light turned on.

Those Frustrating Vomiting Cats

C&T No. 5364

Gary D. Norsworthy DVM DABVP (Feline)
Alamo Feline Health Center
San Antonio, Texas USA

I practice in Texas, many kilometers from Australia. However, I bet we see chronic vomiting in cats with equal frequency – at least once per week and often every day in a busy small animal or feline practice. We hear owners state that their cats vomit from twice per month to twice per day, have done so for many years, and they are otherwise normal. Owners (and us alike) have 4 typical excuses that allow us to accept chronic vomiting: 1) He eats too fast, 2) She has a sensitive stomach, 3) It's just hairballs, and 4) 'He's just a puker,' to quote one of my clients.

Many years ago I tried to understand chronic vomiting with endoscopic biopsies of the stomach. Although the pathologist would describe some abnormal findings, it was rare to get a clear diagnosis that would respond to treatment. Therefore, I changed to ultrasound only to find that the stomach was consistently normal in appearance. However, ultrasound allowed me to move to the small bowel, which is where I found wall thickness to be consistent abnormal. *This led me to the understanding that chronic vomiting in the cat is a sign of small bowel disease and typically not a gastric disorder.*

The clinical signs and ultrasound findings took me to surgery for full thickness biopsies of the small bowel. I would run the bowel to determine the locations for the biopsies. This exam agreed with the ultrasound findings: *small bowel disease in cats is usually segmental.* Most cats have normal and abnormal areas in the small bowel so visualizing the bowel in a surgical setting permitted me to take biopsy samples in the correct places.

Four of my colleagues and I published a paper recently detailing the results from biopsies of 100 cats.[1] Only one was reported to be normal; 99 were abnormal. Forty nine had chronic inflammatory disease, mostly inflammatory bowel disease. Forty six had lymphoma, 2 had mast cell disease, and 1 had adenocarcinoma.

These 100 cases and about another 150 cases that followed have convinced us that chronic small bowel disease is extremely common in cats with manifestation of chronic vomiting, chronic small bowel diarrhea, and/or weight loss. Many cats have no weight loss, but most have a combination of weight loss and vomiting or weight loss and diarrhea. On the other hand, some have weight loss without vomiting or diarrhea. The early event in the progression is hypomotility of the bowel keeping food and hair from moving downstream (aboral) properly and resulting in vomiting. Ultimately, small bowel disease disrupts absorption of nutrients resulting in weight loss.

While in the abdomen we also biopsied the liver and the pancreas trying to understand the incidence of feline triad disease. To our surprise, concurrent inflammation in all 3 organs only occurred in 5% of the cats. We are also pleased to report that, contrary to widely held beliefs, pancreatic biopsies did not induce pancreatitis in our patients.

During the course of this study we redefined the normal ultrasonographic measurements for the cat's small bowel. The study clearly showed that 0.28 cm or greater justifies surgical biopsies. Some references state that up to 0.32 cm is normal. *The study shows that cats with measurements of 0.28 cm or more have a 99% chance of having chronic small bowel disease.*

Although our patients were often 12 years or older, had low body condition scores, and had a 50% chance of having neoplasia, we have only lost 2 cats of the 250 taken to surgery. The cause of death of those 2 and the greatest complication of surgery is hypothermia so several steps should be taken to maintain body temperature. These include administering warmed IV fluids, use of a warming mat on the surgery table, use of booties (for infant children) to prevent heat loss through the foot pads, continuous temperature monitoring during surgery, and placement in a recovery cage with a warming mat. Surgical time is also very important. We average going 'skin-to-skin' in 22 minutes.

One of the motivations for being aggressive diagnostically is that there is mounting evidence that some cases of IBD will transform to lymphoma. If we can diagnose cats at the IBD stage, we have a therapeutic chance of preventing progression to lymphoma, saving the owners hundreds, or even thousands, of dollars and prolonging the cats' lives.

We continue to be amazed at the amount of vomiting cat owners will tolerate. Many of our patients vomited 10 times per week or more, and owners just accepted them as 'normal.' I want to say to these people, 'If your grandchild vomited this much, what would you say to your son or daughter?'

In the study, 25% of the cats taken to surgery were presented for an annual examination. Careful history taking, including proactively asking about vomiting and a comparison of prior and present weights, is an opportunity to identify cats that have not caused alarm in their owners. Failure to motivate our clients to have annual examinations on their cats is a tragic loss to the cat, the owner, and your practice.

Reference

1. Norsworthy GD, Estep JS, Kiupel M, Olson JC, Gassler LN. Diagnosis of chronic small bowel disease in cats: 100 cases (2008–2012). 2013; *JAVMA*. 243(10):1455-1461.

Footnote: The September 2013 isssue of the C&T Series has an article by Dr Norsworthy on his treatment protocol for lymphoma (Perspective No. 100, My Experience with Lomustine for Lymphoma and Inflammatory Bowel Disease. Issue 272, pgs 62-63). This was developed for the cats that came from this study and for the cats that followed.

Dr Norsworthy is Chief of Staff and owner of Alamo Feline Health Center, an adjunct professor at 2 veterinary colleges, an author, a lecturer, a husband, a father, a grandfather, and called 'Papa Nors' by many of his employees. He has practiced for 40 years and looks forward to the next 20.

Re C&T No. 5280 Has anyone seen anything like this before? (March 2013, Issue 270)

C&T No. 5365

Ludmila Mudrinic
E. sneakydroplets@gmail.com

A very long time ago, one of my cat's kittens developed very similar wounds a couple of weeks after birth (only 1 of 4 kittens). The therapy that helped was oral Vitamin B complex + topical ethacridine lactate (Rivanol) + a gauze wrap. I can't remember how long it took to heal, but it did heal.

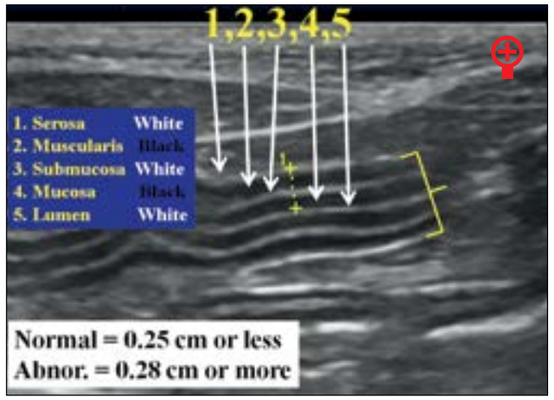


Figure 1: The wall thickness of the small bowel is measured from the outer surface of the serosa to the inner surface of the mucosa (the mucosa-lumen junction) (dotted line). At least 5 (and usually 7-8) measurements of the small bowel wall should be made. We define normal wall thickness as ≤ 0.25 cm, abnormal as ≥ 0.28 cm, and 0.26-0.27 cm as the grey zone.

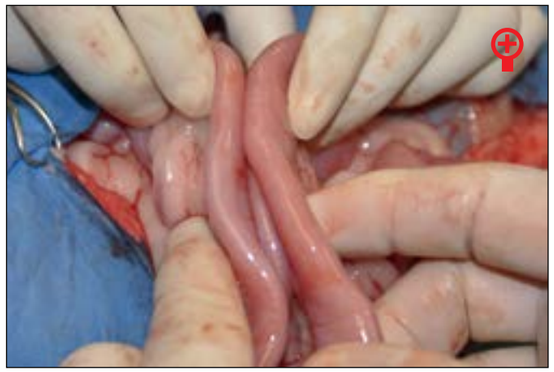


Figure 2: For cats with 2 or more abnormal measurements of the small bowel, laparotomy and full thickness intestinal biopsies are recommended. During surgery, the bowel is run, palpated, and examined visually. Thickened or abnormal areas are selected for biopsy. At least 3 small bowel biopsies should be collected. Note the difference in thickness of these 2 bowel loops.

Figure 3: A 6 mm punch biopsy is used to collect full thickness biopsy samples. The intestine is depressed with the side of the punch biopsy so the anti-mesenteric surface is flat, making the biopsy easier to perform.

Authors' views are not necessarily those of the CVE

Back to TOC page

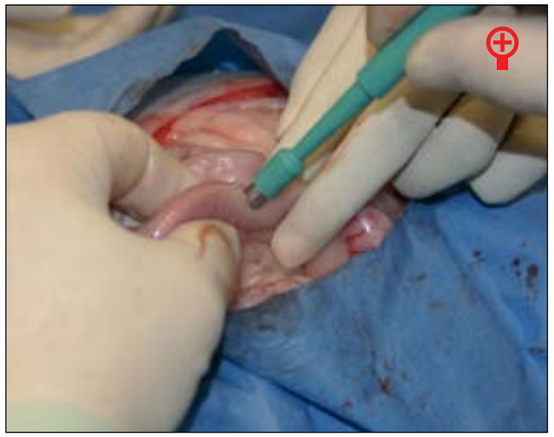


Figure 4: The 6 mm punch biopsy is used to collect a full thickness sample of the small bowel wall. Just as in the skin, the punch is gently spun in place until it cuts through the tissue. Care should be taken not to damage the tissue on the mesenteric side of the intestine.

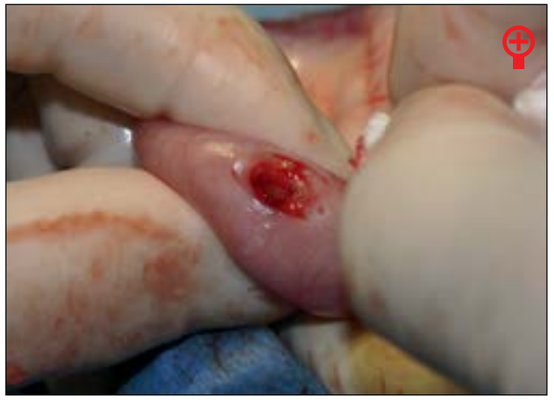


Figure 5: The biopsy site has a smooth margined circular defect which makes closure easy and uniform. Note that occasionally you may need to trim away excess mucosa (although not for this patient).

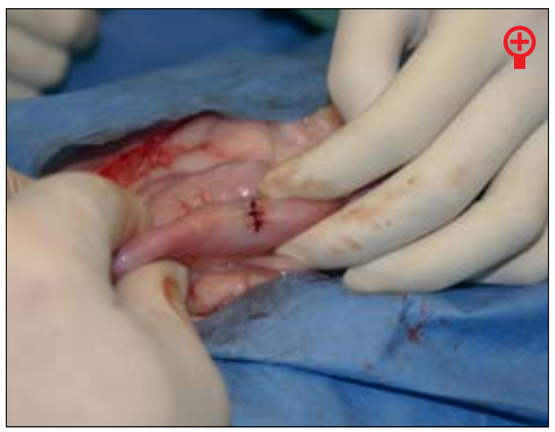


Figure 6: Three simple interrupted sutures (4-0 PDS) are placed to close the biopsy site. The area is leak tested by injecting sterile saline into the lumen of the small intestine with either side of the biopsy site pinched off.

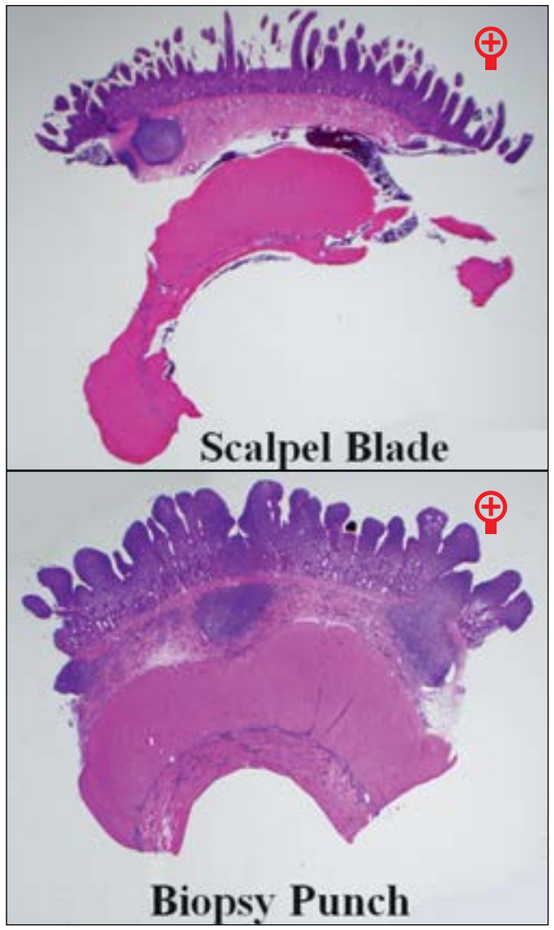


Figure 7: Our pathologist commented with delight when he saw our first biopsy sample using the biopsy punch. These sections are a nice visual that 'Good data in will lead to good data out.' For our patients, this means high quality biopsy samples can lead to clear cut proper diagnoses.

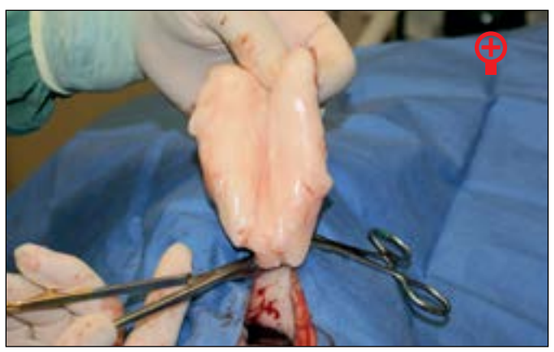


Figure 8: This image shows removal of the falciform fat pad to help in visualization and biopsy of the liver. To screen for concurrent disease in the liver and pancreas (and therefore triaditis), biopsies are taken of both the liver and pancreas during surgery. The liver and pancreatic biopsies are performed first, and then small intestinal biopsies are taken. The liver and pancreas are re-inspected for hemorrhage before the abdomen is closed.

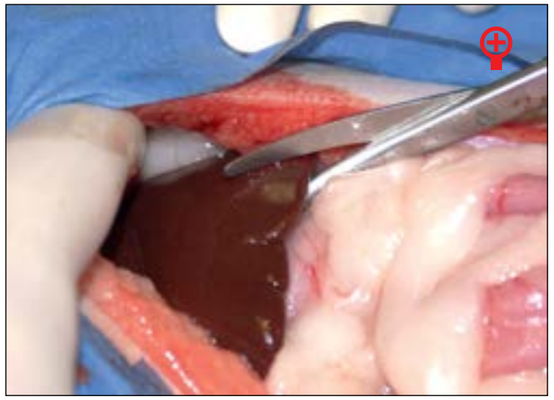


Figure 9: An abnormal area of the liver is preferentially biopsied, as shown in this image. However, if the liver is homogenous a wedge biopsy is cut from an easily accessible edge of tissue using Metzenbaum scissors.

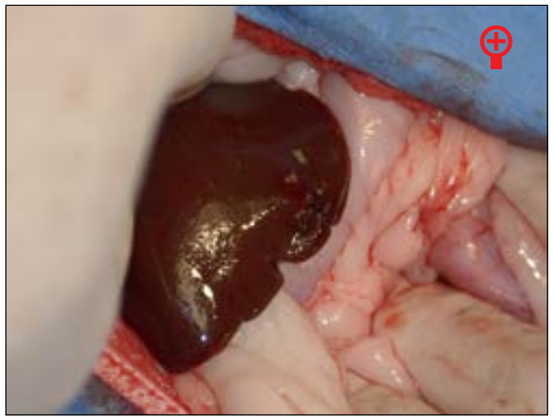


Figure 10: The liver biopsy site predictably hemorrhages, but a single simple interrupted or mattress suture is used to achieve hemostasis. If this is not effective, sterile hemostatic powder (Bleed-X) is also applied topically.*

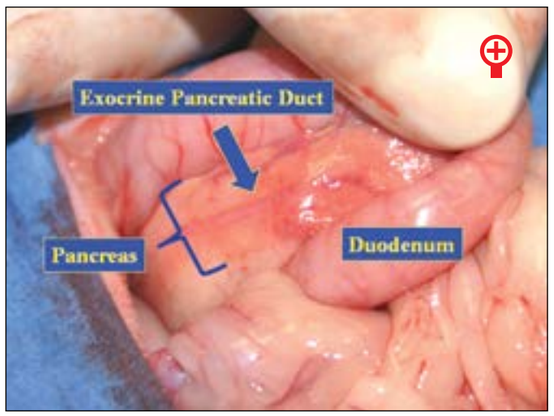


Figure 11: Pancreatic biopsy is routinely performed using a 6 mm punch biopsy. Understanding pancreatic anatomy is important so that the pancreatic duct is avoided. This can be achieved by sampling the edge of the pancreatic tissue.

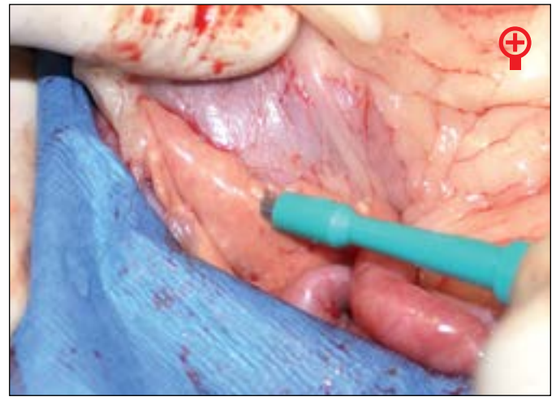


Figure 12: To collect the pancreatic biopsy, a finger is placed gently behind the pancreas. The pancreatic tissue is thin and typically cuts through easily, so delicate tissue handling with the biopsy punch easily yields a sample. Hemorrhage is uncommon, and digital pressure usually achieves hemostasis. Rarely, hemostatic powder (Bleed-X) is used.*

**Bleed-X, LLC; www.bleed-x.com.*

Nebuiliser Cage

C&T No. 5366

Richard Malik
CVE
E. Richard.malik@cve.edu.au

Figure 1. Image courtesy of Mikael Malmberg.

This is a nebulisation unit for a cat with chronic snuffler syndrome. The cat benefits from nebulisation with saline once or twice daily when mucus builds up in the nasal cavity. Pictured is the soft `oxygen cage' (powered by the nebuliser) which lets the cat rest comfortably while receiving nebulisation therapy.

WINNER

Unilateral Thrombus in a Cat

C&T No. 5367

Heather Shortridge
New England Veterinary Centres
212 Rusden Street, Armidale NSW 2350
T. (02) 6771 0200
E. heathershortridge@gmail.com

Figure 1. Manwell

'Manwell', an 11-year-old domestic short hair (4.5 kg) presented after hours to one of my colleagues on a Sunday evening. He had been missing for 24 hours and had returned dehydrated and intermittently open mouth breathing. A heart murmur, mild pyrexia (39.1˚C) a palpable thyroid flick, and sluggish papillary response in the right eye were noted. Bloods were taken, to be sent to the laboratory on Monday, and fluids started at twice maintenance (6mL/kg/hr). Manwell was also given an injection of amoxyclav.

I took over Manwell's case on Monday morning. He had ripped his drip out overnight and was now showing weakness in the hindlegs, including knuckling over in the back right. Remembering a trick I learnt from VIN, I took blood glucose from Manwell's neck (8.3) and this was high compared with that from his back right foot (4.3) suggestive of compromised vascular circulation to the leg. I assumed there was a clot secondary to heart disease and sent away Manwell's blood for a thyroid profile. We began hot and cold compressing Manwell's leg.

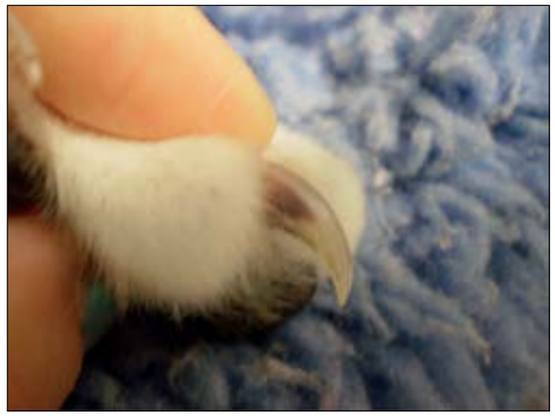


Figure 2.

The blue tinge to Manwell's nails was a cause for concern (Figure 2).

Manwell didn't react to toe pinching, but seemed to resent the whole leg being pulled. I was a bit worried when cutting the toenails very short on the affected leg did not result in much bleeding. We started Manwell on 780iu of heparin BID (initially given IV, and later switched to SQ), as well as starting aspirin using Cardiprim 100mg tablets (50mg sid). I also presumptively started Manwell on Fortekor (2.5mg SID) and Acepromazine (0.5mg IV BID).

Manwell's bloods showed elevated white blood cells (WBC 35.4), mild non-regenerative anaemia (PCV 24%), and a very high creatine kinase (CK) activity (83,865). Manwell's T4 was normal.

I wrote up Manwell's case on VIN, and chest radiographs were suggested, as a major differential for limb thromboembolism is thoracic neoplasia. (Finances were somewhat restrictive and I was trying to work-up the case as stepwise as possible!) Radiographs were taken and there was no obvious tumour present.

The Vinners suggested that the prognosis for Manwell's limb was guardedly optimistic due to collateral circulation of the limbs. Once Manwell was stabilised we sent him home for massage and compressing of the limb and repeated rechecks.

Figure 3.

No positive signs of circulation ten days after first presentation (Figure 3)

Unfortunately the condition of Manwell's foot declined, and by 2 weeks after his initial presentation we had decided we needed to amputate Manwell's distal limb.

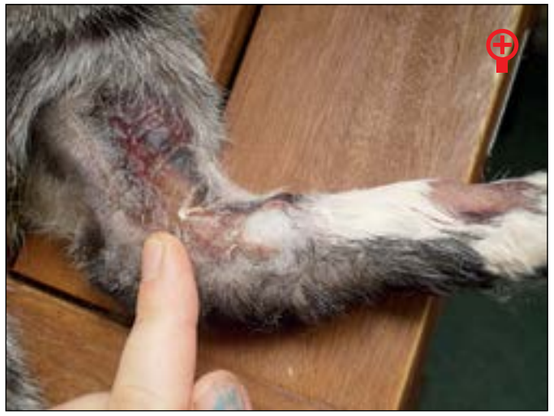


Figure 4.

Two weeks after presentation- non-viable distal limb (Figure 4)

I was fairly concerned about the anaesthetic risks and the risk of Manwell throwing a clot during surgery. We dosed Manwell with aspirin pre and post operatively and kept him on fluids for an extended period. A mid femoral amputation was performed, in the hope of having a good margin of tissue above the clot. I was very worried about Manwell developing further clots but thankfully the surgery, anaesthetic and recovery all went smoothly.

Manwell went home on continued daily Fortekor, as well as 1/8 of a Cardiprim aspirin tablet (12.5mg) every second day. His owners reported that Manwell quickly became accustomed to his 3 legged status.

Over a year down the track, Manwell is 'king of the neighbourhood', and I was inspired to finally write up his case, when his owners brought me a lovely Christmas photo of Manwell relaxing. It was quite stressful to perform surgery on Manwell, but the wonderful year of health he has had since the amputation makes his case a very satisfying one – Figure 5.

Figure 5.

Revolutionise telemedicine by combining your smartphone & telescope

SkyLight- the iPhone Microscope Camera Adapter

Thanks to Mark Krockenberger, Associate Professor, Veterinary Pathology Faculty of Veterinary Science University of Sydney for alerting C&T Readers to this handy device.

The SkyLight camera adapter (basically a plastic frame which allows you to best position the camera on your smartphone to the slide) allows you to attach your smartphone to your microscope – instantly upgrading available microscopes of any age. The captured images and videos – comparable to those obtained with digital microscope-mounted cameras – can be transmitted and shared with anyone in the world. Remember to get the best out of your microscope by keeping it clean and setting it up correctly for the purpose.

For more info, go to: www.skylightscope.com/

(Images sourced from www.skylightscope.com)

SHARE TIPS WITH COLLEAGUES

Email to: elisabeth.churchward@sydney.edu.au

Authors' views are not necessarily those of the CVE

Back to TOC page

Two different clinical presentations of feline pulmonary carcinoma

C&T No. 5368

Yaiza Gomez Mejias LdaVet GPCertFelP – DE Feline DE Participant 2012
Norcross Vets
FY5 3FT Thornton Cleveleys, Lancashire, UK
T. 00 44 (0)7443844085
E. yalpispa@yahoo.es

Thanks to DE Feline Tutor Wayne Mizon for sending us this article for publication in the C&T Series. Due to space constraints, we are publishing an abridged version here but Members/Readers may go to the ebook to read the full version. Download here:

Abstract: Two different clinical presentations of similar pulmonary problems are described. An adenocarcinoma was diagnosed on a 12-year-old neutered female domestic short hair cat that presented with weight loss, appetite loss and respiratory signs. A bronchial carcinoma was diagnosed in a 16-year-old neutered female domestic short hair cat, which was presented with acute onset of hindlimbs paresis and pain. Both diagnoses were done post mortem.

Introduction: Primary lung neoplasms are uncommon in the cat. Older animals are most affected and some authors suggest a predisposition of female old cats (Withrow 2007). Persian cats with pulmonary carcinoma seem to be overrepresented in the data of some studies, although this predisposition should be further validated on the basis of investigations using large sample sizes (D´Costa et al 2012).

With regard to the lung tumours presented in these case reports, the proportion of bronchial adenocarcinomas is high in cats (66%). Bronchioalveolar carcinoma seems to be less common (10%). Other primary lung neoplasms are anaplastic carcinomas, bronchial adenosquamous carcinoma, malignant fibrous histicytoma, sarcoma and bronchial adenoma. (Goldfinch & Argyle 2012)...

Case report 1

A 12-year-old neutered female domestic short hair cat presented with weight loss, anorexia and intermittent coughing. Its body score was 3/5 (4 Kg). Abnormal findings detected on the physical examination included slight tachypnea (RR 70bpm), reduced heart sound on the right side of the chest and an elevated heart rate (200 beats per minute) was observed on the thoracic auscultation.

A right lateral chest radiograph revealed an increased opacity of the caudal lung lobes compared to the cranial lobe compatible with consolidation, unusual focal pleural effusion and a rounded density superimposed to the liver, which showed a slight enlargement (liver tip caudal to last ribs). No ventrodorsal view of the thorax was taken. The differential diagnosis at this point was lung lobe torsion, neoplasia, cardiogenic oedema and pleural effusion, focal diaphragmatic hernia, feline infectious peritonitis (FIP) and tissue inflammation.

Haematology revealed mild anisocytosis. Biochemistry showed a low T4 level (6 nmol/l; reference range 19-65) and this reduction was thought to be associated with a concurrent disease as no signs of hypothyroidism were present.

Fifteen millilitres of pleural effusion was drained and identified as a modified transudate after measuring the protein contain (39 g/l) and the total nucleated cells (0.55 10^9/l) (reference values on appendix 1.A.3). Some of the mesothelial cells showed mild vacuolation of the cytoplasm due to activation. Cells showing criteria of malignancy or microorganisms were not observed.

A fine needle aspiration from the liver revealed suppurative inflammation (appendix 1.A.4).

A congestive heart failure was considered unlikely, given the absence of any other clinical finding consistent with cardiac disease and the uncommon distribution pattern of the fluid within the thoracic cavity. Lung torsion was considered unlikely given that it is a rare condition in cats and affects most commonly cranial lobes (Cohn 2010). Although commonly presented on the right side of the thorax, a diaphragmatic hernia was considered unlikely too, as no abdominal organs were observed within the chest on the radiograph. FIP was ruled out due to the lack of consistent laboratory findings (no anaemia, neutrophilia, lymphopaenia or low alb:glob ratio). A primary liver inflammation and secondary pleural effusion – 'sympathetic pleural effusion' appears to be a common finding in human medicine – was considered unlikely as liver enzymes were within the reference range.

The main diagnosis considered was neoplasia of the caudal lung and secondary inflammation of the liver and therefore an ultrasound guided fine needle aspiration of the consolidated lung was attempted. Unfortunately the procedure did not succeed due to poor cell recovery and the difficulties locating the tumour on the ultrasound (US).

A referral for US guided biopsy of the consolidated lung and suspected mass, computerized tomography for accurate location and delimitation of the tumour and possible lobectomy was advised, but refused by the owner.

The cat was treated with amoxicillin – clavulanic acid (Synulox; Pfizer; 12.5 mg/kg, PO, q 12 h) to prevent an iatrogenic infection and treating a possible pneumonia and meloxicam (Metacam; Boehringer Ingelheim; 0.1 mg/kg, PO, q 12h) as pain relief. A close monitoring of the progress of the pleural effusion was not possible, as the owner was not keen on hospitalization. Therefore it was advised to bring the cat daily. As the client was not able to come that often, the case was monitored by phone. The cat recovered some appetite after draining the pleural effusion. Seven days after presentation the cat was anorexic again. At that moment, the physical examination revealed a restrictive respiratory pattern. A second thoracic draining attempt was suggested but the owner refused any further treatment given the poor prognosis, so the cat was euthanased.

A necropsy examination of the chest revealed the presence of a mass between the heart and the diaphragm, surrounded by fibrosis. There was also atelectasis and consolidation of the cranial right lobe. The abdomen was examined thoroughly and no macroscopic clue of abdominal neoplasia was found.

The histopathology report revealed foci of pleomorphic large epithelial cells occasionally lining cavities containing necrotic debris. The epithelial cells had large secular nuclei, prominent nucleoli and high mitotic rate and variable amount of cytoplasm. Diagnosis was carcinoma of the accessory lung lobe with chronic pneumonia and focal infarction of one diaphragmatic lobe, locally invasive growth with adjacent fibrosis and invasion of the pleural surface (appendix 1.A.4 and 1.B Figures 4-7).

Case report 2

A 16- year-old neutered female domestic short hair cat was presented with acute onset of hindlimbs paresis and pain. Physical examination revealed pale mucous membranes, hypothermia (36.6ºC), paresis, poor anal tone and severe pain on the rear limbs on movement. Proprioceptive deficits were observed on both hindlimbs and withdrawal reflex was absent. Other findings were reduced mentation, anxiety, panting, severe thoracolumbar pain, absence of femoral pulses both sides (but nails vessels pink). Body condition score was 2/5 (3.25 kg). The thoracic auscultation was unremarkable: the heart rate 160 bpm, rhythm was regular and no heart murmur was detected.

The list of differential diagnosis at this point included spinal cord disease, trauma, peripheral neuropathies and arterial thrombo-embolism (ATE).

Haematologic abnormalities included neutrophilia (13.33 X 10^6 cells/mL; reference range, 2.5 to 12.5 x 10^6 cells/mL), mild lymphopenia (0.84 X 10^6 cells/mL; reference range, 1 to 6 x 10^6 cells/mL) and thrombocytopenia (41 X 10^6 cells/mL; reference range, 170 to 650 x 10^6 cells/mL), although the latter was not taken into account as there were clumps present on the film.

Serum biochemical abnormalities included slightly increased creatinine (189 umol/l; reference range, 27 to 186 umol/l) and mild hyperglycaemia (Gluc 10.5 mmol/L; reference range 3.9-8.3 mmol/L).

A right lateral thoracic radiograph did not reveal any enlargement of the cardiac silhouette (Buchanan vertebral heart score was 7.2) or congestive heart failure. However a focal calcification in the dorsocaudal lung fields and opacity of the caudal lung lobes was seen. A ventrodorsal radiograph confirmed the opacity on both caudal lung lobes. Echocardiography was not available. Thromboembolic disease secondary to pulmonary neoplasia was suspected.

The cat was treated with lactate Ringer´s solution (8 mL/kg/h IV), 10 mg/mL methadone (Comfortan; Eurovet Animal Health BV Netherlands) at a dose of 0.3 mg/kg/4h IV, a 12 micrograms/h transdermic fentanyl patch (Mezolar Matrix; Sandoz UK) and heparin sodium 5000u/mL (An initial dose of 200 IU/kg followed every 8h by 50-100 IU/kg SC).

Referral for US guided FNA of the masses, CT and further investigation of the pulmonary mass was suggested but not accepted by the owner. After 2 days on treatment the withdrawal reflex appeared on the right hind limb, but was still completely absent on the left one. Given the poor prognosis, the cat was euthanased at owner´s request.

At necropsy, 2 masses involving the dorsal side of both lateral lung lobes were observed (Appendix 2 Figure 3). A thorough examination of the abdomen did not reveal the presence of any macroscopic signs of abdominal neoplasm.

The histopathology examination showed areas of oedema, mineralised exudate within a distended bronchus, cells formed acini containing mucoid material and chronic pneumonia. The tumour was extending into the tissue around a large bronchus and a section of a pulmonary vein was partially occluded by organising thrombus in which there were foci of solid cords of epithelial cells. Several vessels contained fibrin thrombi.

Diagnosis was bronchial carcinoma with vascular invasion and disseminated intravascular coagulation.

Discussion

Pulmonary carcinomas present with a great variety of clinical signs, which may make diagnosis difficult. In order of common occurrence, clinical signs present in pulmonary neoplasias are: weight loss, lethargy, dyspnoea, anorexia, tachypnoea, wheeze, non-productive cough, ataxia, vomiting, lameness, productive cough, diarrhoea, polyuria/polydipsia and haemoptysis. Pyrexia is infrequently observed (Goldfinch & Argyle 2012). In the cases reported above, inappetence seemed to be caused by the pleural effusion in the first case and by pain in the second one and no obvious appetite loss was noticed by the owners before presentation.

Pneumonia appears to be common and was found, often with suppuration, in about three-fourths of the lungs involved with tumours in one study (Moulton 1981).

Respiratory signs often occur late in the course of the illness. Pleural effusion appears to be one of the most important causes for the dyspnoea. Cats can have concurrent diseases, such as hyperthyroidism and/or cardiomyopathy (Fox & King 2002) because they, as well as neoplasia, also tend to be present at later ages too.

In order of common occurrence, metastatic sites for feline primary pulmonary neoplasms are pleural cavity, bronchial lymph nodes, digits, skeletal muscle, skin, liver, spleen, heart, brain, kidney, eye, intestine, bone, omentum/mesentery and adrenal gland (Goldfinch & Argyle 2012).

Feline 'lung-digit syndrome' is an unusual pattern of metastasis that is seen particularly in feline bronchial and bronchioalveolar adenocarcinoma. The weight bearing digits are most frequently affected, and multiple-digit and multiple-limb involvement is common (Goldfinch & Argyle 2012).

No radiographs were done in these cases to rule out the presence of digits metastasis, but no swelling, reddening of the digit or purulent discharge or dysplasia of any nail was noticed on the physical examination. No signs of local lymph nodes or distant organ metastasis were observed in either necropsy. In the first case, there was invasion of the pleural surface. Liver inflammation revealed on the cytology result might be associated with the organ's location, adjacent to the diaphragm, which was in contact with the tumour. No histology of the liver or diaphragm was performed so it remains unknown whether there was any metastasis involving these organs. However, the liver cytology did not reveal anything but inflammation.

Paresia reported in the second case was suspected to be related to a neoplastic arterial thrombus, which may appear as a part of metastatic process. Ischemia resulting from tumour embolus as a cause of arterial occlusion is indistinguishable clinically from thromboembolus (Smith 2006).

When fluid is present in the thoracic cavity or when lung consolidation, atelectasis or mass lesions are present, ultrasonography may be useful. A portion of the caudal mediastinum, including the accessory lung lobe, can be visualized through the abdomen using a trans-hepatic window created by the liver and diaphragm. (Lora-Michiels *et al* 2003). Ultrasound can distinguish between pulmonary and pleural origin of the mass by observing if it moves with respiratory motion (pulmonary origin) or is stationary and originating from the parietal pleural surface. Because of its resemblance to hepatic parenchyma, consolidated lung may be incorrectly identified as herniated liver (Mattoon 2007). That may make difficult the procedure of taking a fine needle aspiration sample from the consolidated lung, as liver tissue can be aspirated instead.

A recent study showed a greater sensitivity of CT in comparison to radiographic views in detection of pulmonary nodules in dogs (Armbrust et al 2012). Even if a single pulmonary nodule is seen radiographically, it is not uncommon to find multiple small ▶

Authors' views are not necessarily those of the CVE
Back to TOC page

Appendix 1: Case 1 Appendix 1.B: Images

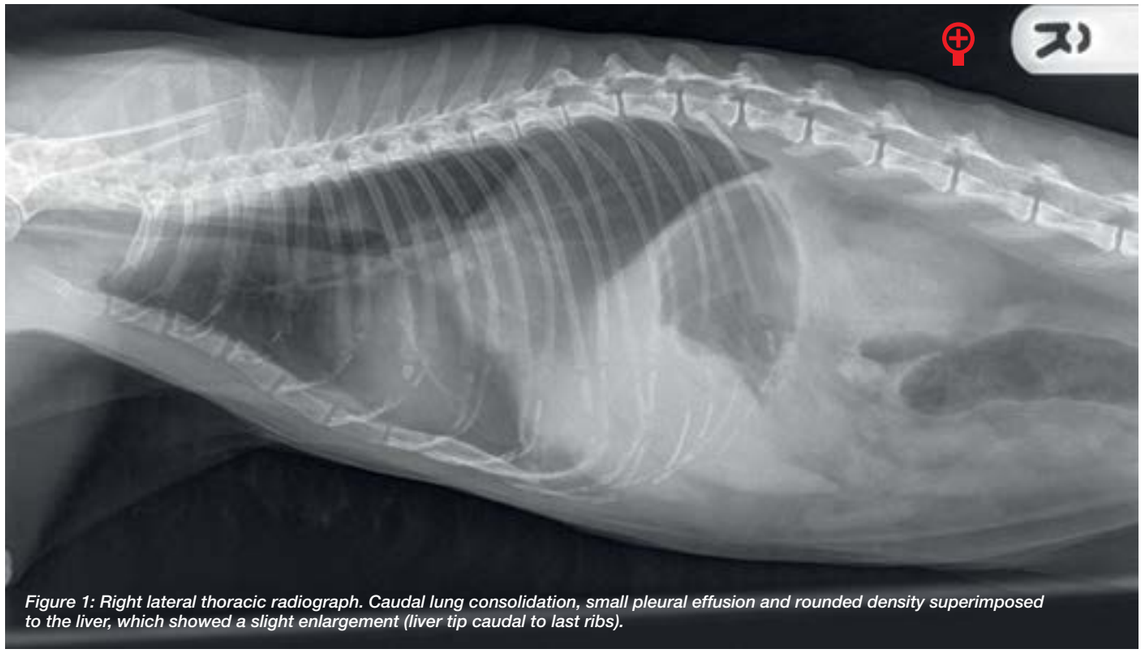


Figure 1: Right lateral thoracic radiograph. Caudal lung consolidation, small pleural effusion and rounded density superimposed to the liver, which showed a slight enlargement (liver tip caudal to last ribs).

lesions at surgery, suggesting the importance of advanced diagnostic imaging before surgery. (Rissetto et al 2008)

Transthoracic fine-needle aspirates can be quite rewarding for larger lesions in a peripheral location; the correlation between cytopathology from FNA done with small-gauge (25 to 27) needles and histopathology of the lung seems to be high (82%) and the procedure has been shown to be safe (DeBerry et al 2002). However, these tumours often have a necrotic centre that may confuse interpretation (Withrow 2007).

Other methods of obtaining lung tissue for microscopic examination are percutaneous cutting needle biopsy, transbronchoscopic biopsy and thoracotomy and lung biopsy (Fox and King 2002). Despite a higher cellular yield, the complication rate using 18- to 21-ga needles is reported to be 46%, with a fatality rate of 15% (DeBerry et al 2002).

Metastatic cancer to the lung is much more common than primary lung cancer in the dog and the cat (Withrow 2007). Primary and metastatic carcinomas are similar and they cannot be definitively differentiated by cytologic evaluation alone (Burkhard et al 2001). The fact that cats are more likely than dogs to have atypical radiographic patterns of pulmonary metastasis does not help either, so staging can become difficult. However most primary neoplasias are mammary and pulmonary carcinomas (Forrest and Graybush 1998), so clinical data may provide useful information.

Lung cancer presents a great mortality. The median survival time is related to the histologic type: undifferentiated tumours present a shorter life expectancy that the differentiated carcinomas (D´Costa 2011). The treatment of choice is lobectomy. Small masses away from the hilus may be removed by thorascopic techniques. Malignant pleural effusions have a very poor prognosis and may limit the value of the surgical intervention. The use of mitoxantrone as adjuvant therapy after pneumonectomy in cats has been reported (Clements et al 2004).

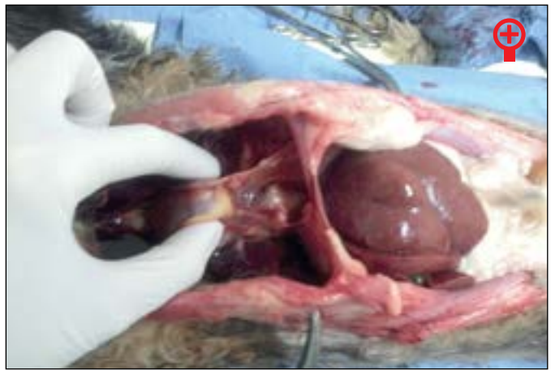


Figure 2. (above) Necropsy. Accessory lung-lobe tumour with adjacent fibrosis and invasion of the pleural surface.

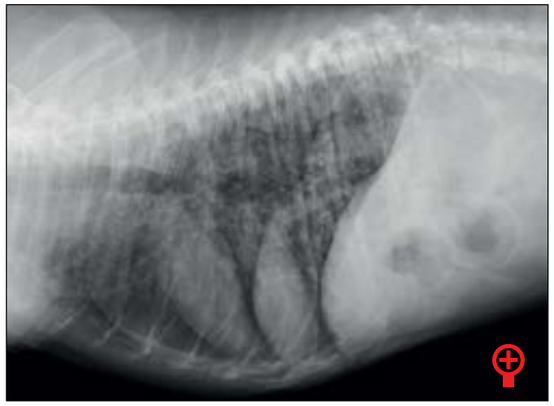
Figure 3: (left) Macroscopic view of the lung after fixation. Neoplastic proliferation visible on accessory lung lobe.

Answer to C&T No. 5344

C&T No. 5369

Suzanne Pears
Maroubra Veterinary Hospital
88 Bunnerong Road
Pagewood NSW 2035
T. (02) 9344 8722
E. suzannepears@hotmail.com

e-book Rollover to view enlarged image



Laboratory: USYD results BAL:

Wet prep: no parasites or fungi. Direct smears reveal mucous strands that entrap large numbers of WBCs. The majority of WBCs are small, to more generally medium, lymphoid cells. Nuclei are round or lobulated and the cytoplasm scant and deep blue-grey. Other cell Types include approx. 10% neutrophils, 7% macrophages. Orophangeal squames are common. Aggregates of well-differentiated plump cuboidal/columnar epithelial cells are present. An infectious agent was not detected.

Lymph nodes aspirates: The smears of the L & R prescapular lymph nodes are similar. Cell recovery is very good. The majority (90%) of cells belong to a population of medium lymphoid cells with a slightly eccentric, round or indented nucleus and a modest volume of blue-grey cytoplasm. A juxtanuclear pale zone is often apparent. Lymphoglandular bodies and ruptures cytoplasm fill the background. Mitotic figures and degenerate cells are scattered amongst the lymphoid cells. Most of the remaining cells are small lymphocytes, although larger lymphoid cells and macrophages ingesting cell debris are scattered throughout the smears. The smears of L&R mandibular nodes are much less cellular but the findings are similar to that of the prescapular nodes.

Comment: Marked lymphoid infiltrates are evident in the respiratory wash. Taken in conjunction with the results of the lymph node aspirates, the findings suggest a round cell/ lymphoid malignancy involving both the lungs and peripheral lymph nodes - lymphosarcoma of a medium lymphoid cell.

22/07/11: Culture results – light mixed bacterial growth suggestive of oropharyngeal contamination. No fungi isolated after 3 days incubation. Final report if culture remains negative.

25/07/11: History – Immunoperoxidase staining showed lymphoma is B-cell.

Answer to C&T No. 5345

C&T No. 5370

George Reppas
Registered Specialist Veterinary Pathologist
Vetnostics
60 Waterloo Road, North Ryde NSW 2113
T. (02) 9005 7012
F. (02)9005 7950
E. george.reppas@vetnostics.com.au

George is a Registered Specialist Veterinary Pathologist working at Vetnostics in North Ryde NSW and is a Fellow in Veterinary Clinical Pathology of the Australian and New Zealand College of Veterinary Scientists as well as a Diplomate of the European College of Veterinary Pathologists. He has written and co-authored articles in many scientific journals and veterinary texts and has served as an examiner of the ANZCVS Fellowship examination in Veterinary Clinical Pathology. Recently he has been collaboratively involved in developing a selective range of advanced diagnostic techniques (Immunocytochemistry & Infectious Diseases PCRs) specifically adapted to veterinary cytology which are available through Vetnostics.

Q. A 14yo FN DSH Cat presented with multiple firm subcutaneous swellings about the head and swollen/thickened distal limbs and feet. A smear made from the FNA of one of the skin lumps on the cat was stained with Diff-Quik (Figures 1-3 below).

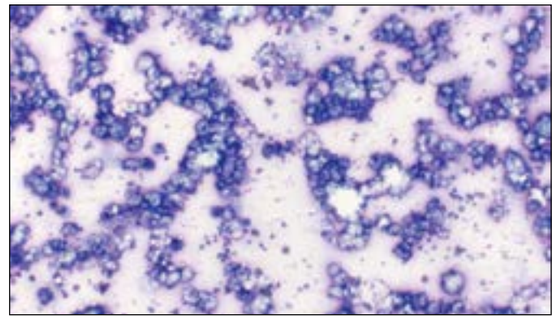


Figure 1. Diff-Quik photomicrograph of FNA smear - low power (x 20)

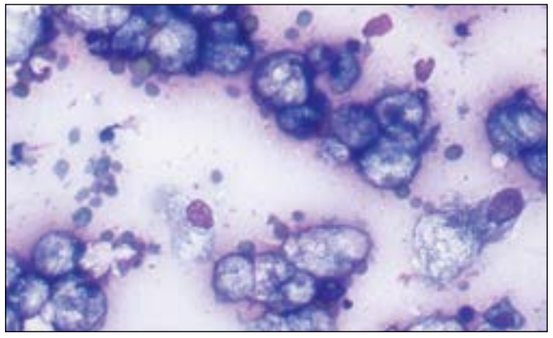


Figure 2. Diff-Quik photomicrograph of FNA smear - low power (x 40)

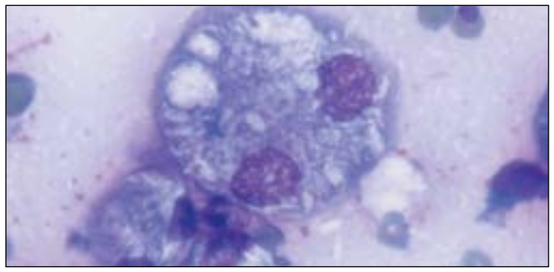


Figure 3. Diff-Quik photomicrograph of FNA smear - high power under oil immersion (x 100).

A. This is a case of Feline leprosy syndrome.

Previous figures 1-3 (inclusive) reveal negatively staining bacilli located both intracellularly and occasionally extracellularly.

Feline Leprosy Syndrome (FLS)

FLS may present with single or multiple cutaneous nodules ranging from 2-40 mm in diameter typically accompanied by peripheral lymphadenomegally. In cats with focal disease, lesions are often found on the face, head, limbs or trunk – areas subject to penetrating injury. The location of lesions on cats suggests inoculation of organisms through insect bites, rodent bites or (most likely) fight wounds. Fight wounds are normally ascribed to cats, but may include injuries from prey species such as rats or possums.

Mycobacterial species associated with feline leprosy include *M. lepraemurium, M. visibile, Mycobacterium* sp. strain Tarwin and a novel species found in New Zealand and the East coast of Australia. Some organisms (such as *M*. sp. strain Tarwin) are associated with localised disease in an immunocompetent host, while others (such as the novel East-coast species) are associated with haematogenously disseminated disease (usually limited to the skin) in an immune-deficient host.

Performing fine needle aspiration (FNA) cytology would be, in the first instance, the easiest and fastest way of establishing a definitive diagnosis or raising the index of suspicion for the possibility of infection as well as helping dictate the next course of action in the further investigation of this mycobacterial infection which would usually include qPCR (see below).

Cytologically, numerous negatively staining bacilli (NSB), either within macrophages and giant cells or extracellularly, are present on Diff-Quik stained smears (**Figure1-3**). Acid fast bacilli are easily seen in Ziehl-Neelsen (ZN)-stained smears from needle aspirates of these lesions as well (**Figure 4**) which reveal numerous pink/red mycobacterial bacilli located both intra-cellularly and extra-cellularly.

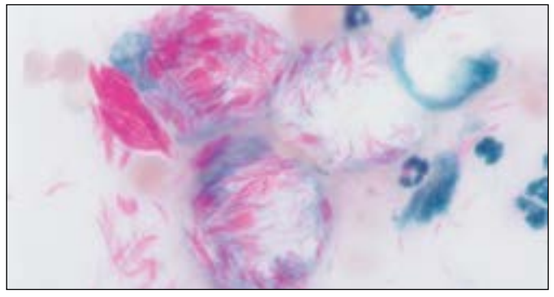


*Figure 4. **ZN stain** high power under oil immersion x 100*

Once a cytological diagnosis has been established, and as most organisms associated with FLS cannot be cultured, PCR testing is recommended for mycobacterial species identification. PCR testing utilises primers that amplify regions of the 16S rRNA gene and ITS region followed by sequence analysis of the resulting amplicon (**Figure 5**). PCR testing is very sensitive even when used on paraffin embedded formalin fixed (PEFF) tissues, as long as the contact time of the tissue specimen with formalin is less than 48 hours.

TGAGTAACACGTGGGTAATCTGCCCTGCACTTCAGGGATAAGCCTGGGAAA
CCGGGTCTAATACCGAATAGGACCTTAAGGCGCATGCCTTGTGGTGGAAA
GCTTTTGCGGTGTGGGATGGGCCCGCGGCCTATCAGCTTGTTGGTGGGGT
GACGGCCTACCAAGGCGACGACGGGTAGCCGGCCTGAGAGGGTGTCCGGC
CACACTGGGACTGAGATACGGCCCAGACTCCTACGGGAGGCAGCAGTGGG
GAATATTGCACAATGGGCGCAAGCCTGATGCAGCGACGCCGCGTGAGGG
ATGACGGCCTTCGGGTTGTAAACCTCTTTCACCATCGACGAAGGTTCGGGT
TTTCTCGGGTTGACGGTAGGTGGAGAAGAAGCACCGGCCAA

Figure 5. PCR amplicon amplified from a fresh tissue specimen obtained from the cat with cutaneous mycobacterial infection.

Recently with the advent of qPCR testing, as for Canine Leproid Granuloma (CLG), we have been able to utilise methanol-fixed and Diff-Quik stained smears of fine needle aspirates which have been diagnosed cytologically with mycobacterial infection, to amplify DNA in cases where NSB or AFB are abundant; this involves aseptically scraping material from the slides using a scalpel blade into a sterile tube, followed by appropriate DNA extraction procedures. Indeed, this represents the most expedient and least invasive way to obtain a definitive diagnosis, where appropriate laboratory support is available.

Histologically, FLS is not distinguishable from potentially zoonotic tuberculous infections. In cases due to *M. lepraemurium* and *M. avium* complex (MAC), bacilli are not visible in haematoxylin and eosin (H&E) stained sections. In contrast, *M. visibile* (hence the name) and the novel East coast species can be seen in H&E sections because they weakly take up the haematoxylin. Despite this distinguishing feature, histological features should not be relied on to predict aetiology, the specific organism involved or the prognosis. Indeed, it is desirable to instruct the veterinary laboratory to forward tissue specimens to a mycobacteria reference laboratory for PCR and sequence analysis (to determine aetiology).

In contrast to canine leproid granuloma (GLG), FLS syndromes generally have an unrelenting, progressive clinical course. Furthermore, lesions can recur following surgical excision, especially when inadequate margins are obtained and appropriate anti-mycobacterial agents are not given post-operatively. For this reason, medical treatment should be instituted immediately after diagnosis, as a delay may permit lesions to spread to contiguous skin, lymph nodes and even internal organs.

Reference

Malik R., Smits B., Reppas G., Laprie C., O'Brien C., Fyfe J., (2013) Ulcerated and nonulcerated nontuberculous cutaneous mycobacterial granulomas in cats and dogs. *Veterinary Dermatology* 24, 146-e33 *Vetnostics Winter 2012 Newsletter*

Winner

Congratulations to Mark Hynes from Kingston Animal Hospital, who answered this Q and therefore won a CVE proceedings of his choice. ***www.vetbookshop.com***
Answer: Mycobacterium aka feline leprosy

Invited Comment on C&T No. 5332 Canine behaviour – have we got it right?

(David Bligh, Sept 2013, Issue 272, pg 29)

Courtesy of:

Kersti Seksel (Behavioural Medicine DE program Tutor)
Registered Veterinary Specialist, Behavioural Medicine
BVSc (Hons) MRCVS MA (Hons) FACVSc DACVB DECAWBM
Sydney Animal Behaviour Service (SABS)
55 Ethel Street, Seaforth NSW 2092
T. +61 2 9949 8511
E. sabs@sabs.com.au

Places still available in our DE Behavioural Medicine course led by Kersti. For full details, visit: www.cve.edu.au/debehaviouralmedicine

The behaviour of all species, including dogs, is a complex yet elegant interplay between genetics, learning and the environment. The speciality of veterinary behavioural medicine (VBM) is multifaceted and follows scientific principles which are constantly being adjusted and refined, as we learn more about the brain at the neuro-molecular level, neuronal pathways and the intricacies of how neurons communicate and change in response to learning. It involves the study of the normal behaviour of a species as well as the study of mental health disorders (or what may be termed as abnormal behaviours). VBM is much more than just training or using the reinforcement/redirection/distraction/ignore unwanted behaviour techniques. It involves the diagnosis and then management of animals with mental health disorders in a scientific and humane way that focusses on the welfare of the animal as well as the people that live with it.

In the middle of last century it became popular to classify dogs as pack animals that lived in fixed dominance hierarchies and from this, constructs, like dominance in dogs arose. However, further research has shown that this was based on misinterpretation of findings. About 10-15 years ago the dominance model was replaced by the anxiety model to help explain many behaviour problems in pets. This is similar to the models that are used in human psychiatric medicine and it is recognised that there are many, many similarities across species, particularly in regard to fear and anxiety processing in the brain. Anxiety is a medical condition just like diabetes and thyroid disease are medical conditions.

It is well recognised that an animal's ability to control and predict its environment can significantly reduce anxiety. People like predictability too. We like to know what time to turn up to work or social events and most people prefer to interact with stable, predictable people and find dealing with unpredictable people stressful. The same holds true for dogs.

Programs that include 'taking charge', or being 'authoritative', help create predictability which can significantly reduce anxiety. However, predictability can be created without being 'authoritative'. Being 'authoritative' or using 'reminders' also risks reducing the bond the dog has with the owner and risks increasing aggression. This is particularly significant for animals with anxiety disorders as they are very sensitive to 'authoritative' techniques which can damage the human animal bond and their behaviour often deteriorates with such techniques. And a good owner-pet bond is integral to successful treatment.

Recognition of the abnormality of the animal's behaviour is important. If the behaviour is considered normal then referral to a dog trainer to help teach the dog manners is appropriate. But if teaching the dog to come sit or stay will not resolve the underlying problem then it is not a training problem. However, dogs that exhibit aggression in contexts when aggression is not warranted are not normal and need referral to a veterinary behaviourist to diagnose and treat their mental health disorder. Aggression is often anxiety based so treatment relies on creating a consistent and portable framework for how interactions with humans work, so the dog's anxiety can be reduced by increased reliability.

The treatment of dogs with anxiety disorders such as aggression always includes environmental management, behaviour modification and sometimes medication (at appropriate dose rates). This combination used by veterinary behaviourists creates predictability and stability in the dog's life without being 'authoritative' but rather being consistent and predictable in all interactions with the dog.

There is no doubt that 'authoritative' training methods work. It is because they are predictable when done correctly and the dog can work out how to avoid the correction or reminder. However, there are better and more humane ways to accomplish the same outcome.

Good trainers who use non aversive techniques are a great asset to veterinary behaviourists. They provide lots of hands-on help to assist clients to implement the treatment plans recommended by the veterinary behaviourist.

Anxiety is a medical condition and only veterinarians can diagnose and treat medical conditions. It is unfair on the trainers, clients and their pets to put this responsibility on trainers. After all, would veterinarians send their patients to a dog groomer to treat and diagnose atopy?

VBM is an enormous field and we still have much to learn about the brain, and our approach is likely to change further as we learn more. However, as with any specialty in veterinary medicine, diagnosis and treatment should be evidence based and focus on the humane treatment of the animals in our care.

I commend all readers to www.dogwelfarecampaign.org

Further reading:

Berteselli GV, Servidaq F, DallAra P, *et al.* Evaluation of the immunological, stress and behavioural parameters in dogs (Canis familiaris) with anxiety-related Disorders. In: Mills D et al (eds). *Current Issues and Research in Veterinary Behavioral Medicine, Purdue Press*, (2005), 18-22
Bradshaw,JWS. Blackwell,EJ, Casey RA Dominance in domestic dogs: useful construct or bad habit? *Journal of Veterinary Behavior*(2009) 4, 135-144
Dreschel NA. Anxiety, fear, disease and lifespan in domestic dogs. *Journal of Veterinary Behavior* (2009): 4: 249-50
Herron ME, Shofer FS, Reisner IR. Survey of the use and outcome of confrontational and non-confrontational training methods in client-owned dogs showing undesired behaviors *J App Anim Behav Sci* (2009); 117, 47–54
Riva J, Bondiolotti G, Micelazzi M, *et al.* Anxiety related behavioural disorders and neurotransmitters in dogs. *J Appl Anim Behavr Sci* (2008); 114, 168-181

WINNER OF BEST PICTURE

Share and share alike – Casper with bandicoot cobber *(cover image)*

Nicole Laing – DE Feline Medicine 2011 Participant
VetWest Animal Practice CANNING VALE WA 6155

Congratulations to Nicole for her award-winning pic, entitling her to a free DVD.

Do YOU have a great pic?

Email it to: elisabeth.churchward@sydney.edu.au for your chance to win a DVD of your choice www.vetbookshop.com

WINNER OF BEST FILM CLIP

Dental mystery in a Boston Terrier

GO TO THE ‹e-book› TO VIEW THIS FILM CLIP

C&T No. 5371

Mark Hynes
Kingston Animal Hospital
1 Freeman Street
KINGSTON TAS 7000
T. (03) 6229 5900
E. hynesmark1@hotmail.com

Today I performed dentistry on a 4-year-old French Bulldog male castrate and an incidental finding was a gelatinous mucoid string emanating from a gingival puncta above the upper left PM4/M1. You could keep pulling this tenacious material like children's toy slime. I showed the video to the owner who reported that they have had to regularly pull this jelly-like drool from that side of his face with a chux on a regular basis for most of his life!

It is obviously a normal salivary gland duct but I had never seen such tenacious material exude from it like this one, so thought I'd share the film clip with C&T readers.

Invited commentary courtesy of:

Dr Christine Hawke BSc (Vet) (Hons) BVSc (Hons) PhD MACVSc (Veterinary Dentistry)
Sydney Pet Dentistry (Mon to Wed)
T. 0408 782 611 OR
Animal Referral Hospital (Thurs & Fri)
T. (02) 9758 8666
E. christine@sydneypetdentistry.com.au

What a great video! This saliva is obviously very thick and mucoid with little or no serous component. Xerostomia, or 'dry mouth' due to a lack of serous saliva production, can be seen with dehydration, in certain diseases such as diabetes mellitus and Sjogren's syndrome. It is also a common side effect of some drugs such as antihistamines, calcium channel blockers and of course atropine (was this used as a premedicant prior to anaesthesia?).

In most cases, as long as the other salivary glands are producing enough watery saliva to do the job, this is not an issue for the dog. If not, the dog will be predisposed to periodontal disease, as normal saliva has a flushing effect, and contains enzymes and immunoglobulins that help control plaque accumulation. Being a French Bulldog, periodontal disease will be an issue either way!

Further comment on: Economic validation for stocking uncommonly used antidotes and antiemetics on the drug shelf (C&T No. 5329, Sept 2013)

Reply to C&T No. 5323

(Sep 2013, Issue 272)
Stabilisation of a fractured mandible in an Eastern Grey Kangaroo

C&T No. 5372

Gary Wilson BVSc MVSc MACVSc DICEVO
Advanced Animal Dentistry Pty Ltd
PO Box 2095
Wellington Point Qld 4160
T. (07) 3824 8895
E. gwvet2thdoc@gmail.com

The principle of oral fracture repair is to place an orthopaedic device at the tension side of the fracture. In all oral fractures this is the occlusal surface of the teeth and in this particular case would be the dorsal surface of the mandible.

Stabilisation at one end of a fracture is not suitable. In a joey of this age and on the diet stated, no repair would be needed in most cases. If the fracture site was unstable, it would have remained so after this type of repair as the muscles of mastication would have distracted the fracture site. The fact that it healed is an indication that no stabilisation was required.

If the joey had an unstable symphysis (they normally do move independently to a degree) and it was thought necessary to 'repair', then drilling holes through teeth is never an option. This has the potential to introduce bacteria into the pulp either directly if the hole goes through the pulp chamber or indirectly via the exposed dentinal tubules. Secondarily, the resultant exposure of the dentine will have irritated the odontoblastic process in the dentinal tubules. This is very painful. Will the animal show this? - No. It is a prey species and will just put up with this pain.

If repair of the holes was deemed necessary, then they should not have been drilled in the first place. If they were thought to not have caused iatrogenic damage to the sensitive tissues then why repair them at all? Small defects in the teeth would be irrelevant. Putting a restoration over the holes adds no more inherent strength to the tooth, it just covers the hole.

If the teeth actually needed to be stabilised, the dental materials mentioned could have been used to bond the teeth together without drilling holes in the teeth. If these materials were used correctly, undercuts (undermining) are not made in the dentine of the tooth. In fact undercuts are not recommended except if one is using amalgam. Acid etching and correct bonding techniques allow these materials to be placed without removing further dental tissue.

C&T 5332 & Comment by Prof Paul McGreevy

C&T No. 5373

Mario Viscardi DVM
Ballantrae Drive Veterinary Clinic
Shop 10B, 91 Ballantrae Drive
St Andrews NSW 2566
T. (02) 9820 2711
E. drmario@optusnet.com.au

I thought that the initial article was sensible, and found myself largely agreeing with the writer. However, I did not find the invited comment helpful for a number of reasons.

It opens with 'I strongly encourage veterinarians who care about dogs to read it in full...'

To me this is patronising because it implies either than some of us do not care about dogs, or if we do care, we need to read the article. Following that is the abstract of his journal article which I found difficult to understand, to say the least. I tried to locate the article on the internet, but the only site that gave me some hope wanted $35 to access it. Maybe there is an easier way to retrieve it, in which case it would have been helpful to have that link at the bottom of the comment. So I remain a frustrated reader.

Perspective 101

A Toxicologist's Perspective

Rhian B Cope & Rosalind Dalefield

Practical Practitioners – Prepare to be Enlightened!
By Aine Seavers

This might be a very academic text in its perfection and attention to detail, but boy are there total gems of practical knowledge in here that will set you back on your heels questioning what we do and why.

I was amazed to find the curve balls in some day-to-day beliefs we have about toxins. For instance; My Rule – taught to me 30 years ago – was never to induce emesis in a poison case unless within 20 minutes ingestion, a known non-irritant and no seizures present, which means I have rarely induced emesis other than in a toad fish ingestion or a methiocarb case after atropine had controlled the fasciculations.

But – I would have induced emesis for rat bait if one had ever been promptly presented post ingestion. Luckily for me and my patients, rat bait ingestions tend to get presented many hours to days later because I would have induced emesis...

For why I was lucky and more – read this perspective and be enthralled.

Rhian B Cope BVSc BSc(Hon 1) PhD cGLPCP DABT ERT FACTRA
Toxicology Excellence in Risk Assessment
Cincinnati, Ohio USA
E. rhian_b_cope@yahoo.com
www.tera.org/
T. +1-513-542-7475

Dr Cope received her veterinary degree from the University of Queensland and went on to obtain her PhD in phototoxicology from the University of Sydney. She has held teaching positions in toxicology at the University of Illinois and Oregon State University and from 2006-2013 owned and operated a private toxicology consultancy company. Currently she is a toxicologist at Toxicology Excellence in Risk Assessment in Cincinnati USA.

Dr Cope holds the Diplomate of the American Board of Toxicology certification and is a European Registered Toxicologist as well as being a Fellow of the Australasian College of Toxicology and Risk Assessment. She is a US National Institutes of Health research grant awardee and has published more than 40 peer-reviewed papers. She is also the author of a number of book chapters in relevant veterinary toxicology texts.

Rosalind Dalefield BVSc PhD DABT DABVT
E. rosalind@dalefield.com
www.toxi.co.nz
M. +6427 543 6668

Rosalind Dalefield graduated from Massey University with a BVSc in 1984 and with a PhD in veterinary pathology in 1992. After completing a postdoctoral residency in toxicology at the Comparative Toxicology Laboratories at Kansas State University she passed the examinations to become a Diplomate of the American Board of Veterinary Toxicology in 1999 and a Diplomate of the American Board of Toxicology later in the same year. Her experience in toxicology includes preclinical pharmaceutical toxicology, environmental toxicology, military and industrial toxicology, food safety toxicology and risk assessment, analytical toxicology, performance drug testing, veterinary and wildlife toxicology, and toxicological pathology.

1. SINGLE DOSE ACTIVATED CHARCOAL AS A TREATMENT FOR POISONING IN DOGS AND CATS

The purpose of this short commentary is to review the basic principles, types of activated charcoal (AC) modes of action, efficacy, risks, contraindications and recommendations regarding the use of single dose oral AC (SDAC; synonym activated carbon) typically administered at a dose rate of 1 g/kg body mass as a treatment for acute oral poisoning in dogs and cats. Recently, a dose rate of 10 times the ingested toxin/toxicant mass as been suggested (i.e. a 10:1 AC to drug ratio) and there is limited meta-analysis data to support the concept that the higher this ratio the greater the efficacy of SDAC. However, a ratio AC to drug ratio of 10:1 is generally accepted as 'reasonable'.[1] SDAC is commonly performed using either oral dosing or via a nasogastric or orogastric tube. We have specifically excluded any discussion on the use of AC in ruminants and its use to disrupt enterohepatic or enteral cycling of toxicants.

Basic Principles

The basic objective of SDAC is to reduce the enteral bioavailability (F) of a putative toxin/toxicant, thus reducing its area under the curve (i.e. systemic exposure; AUC) and/or its plasma C_{max}. Small-scale studies on the efficacy of SDAC in healthy human volunteers have generally supported this concept. However, for a fixed dose, the AUC is dependent on both bioavailability (F) and clearance (CL):

$$AUC = \frac{Dose \; x \; F}{CL}$$

Hence SDAC may theoretically also act by increasing clearance. And in fact, there is evidence that SDAC administered to healthy humans dosed with supra-therapeutic doses of paracetamol increased drug clearance. This effect is probably due to reduction of enterohepatic cycling.

The fundamental underlying basic principles of SDAC are as follows: (a) the toxicant must come in direct physical contact with the administered AC in the stomach; (b) following on from this, the toxicant must still be in the stomach when the SDAC procedure is performed; (c) the toxicant must bind to the AC; and (d) the toxicant must remain bound to the AC under the variable physicochemical conditions of the gastrointestinal tract until it is passed in the faeces.[2]

Points (a) and (b) have a clear implication: unless the toxin/toxicant forms gastric concretions or delays gastric emptying, **SDAC treatment must be performed soon as possible following ingestion. Current thinking is that the likely effective treatment window for SDAC treatment is 0-60 minutes post-ingestion.**

Types of Activated Charcoal

AC is not the same as ordinary charcoal. AC is carbon that has undergone 'activation' by either physical reactivation or chemical activation. Physical reactivation means exposure to hot ▶

gases either by carbonization/pyrolyzation or oxidation at high temperatures. Chemical activation consists of adding a strong acid or base to the material followed by carbonization.

AC products are a diverse group of materials with differing physicochemical behaviors. The broad groups of ACs that may be encountered in veterinary medicine include:-

- Powdered activated carbon (PAC). This is the usual and traditional form that consists of particles of < 1 mm diameter (and typically 0.15-0.25 mm in diameter). PAC has a very large surface to volume ratio and a small diffusion distance.
- Granular activated carbon (GAC). GAC has a relatively larger particle size, lower surface to volume ratio and a greater diffusion distance. For these reasons, it is generally **less suited** for SDAC treatment.
- Extruded activated carbon (EAC). EAC consists of powdered AC plus a binder that are fused together and extruded into cylindrical pieces with diameters of 0.8-130 mm. These materials **are not suitable** for SDAC treatment.
- Bead activated carbon is made from petroleum pitch and supplied in diameters from approximately 0.35 to 0.80 mm. This material is primarily used in filters or other industrial applications.
- Impregnated carbon (IC). IC is a porous carbon that is impregnated with various metal cations or catalysts. It is primarily used in control of air pollution, water purification and hemoperfusion.
- **AC biscuits, capsules or tablets. These over the counter products are intended as aids for the control of diarrhoea, indigestion and flatulence. Related products are used to prepare the bowel for radiography by reducing the amount of intestinal gas. These products are generally not *useful* for SDAC.**

Because of its porosity and large surface area to volume ratio, 1g of pharmaceutical grade AC will have an effective absorptive surface area of 500 – 1500 m^2.

Iodine number (mg of iodine absorbed per gram of AC) is the usual parameter used to measure the binding capacity of AC. It is a measure of the number of micropores (up to 2 nm diameter) present per unit mass. Iodine number is also proportional to surface area per unit mass. Pharmaceutical-grade ACs will generally have an iodine number of 600-1100 mg/g.

Molasses number and methylene blue number are measures of mesopore (greater than 2 nm diameter) content and is a less useful indicator of effectiveness under most toxicological circumstances.

The author's personal preference is to use pre-mixed AC preparations. Some of these preparations will also contain kaolin, which is claimed to increase the spectrum of materials absorbed by the AC. Pre-mixed AC preparations that are available for veterinary use include:

- Liquid formulations: Liqui-Char®, Toxiban®, Actidose-Aqua®, Actidose® with Sorbitol, CharcoAid®, CharcoAid 2000®;
- Gel formulations: Liqui-Char®. Please note that this gel preparation is not suitable for use with a nasogastric tube. It is specifically designed for oral administration and comes in a oral dosing syringe (similar to the syringes used for horse worming pastes).

Modes of Action

The primary mode of action of AC is the binding of toxicants via van der Waals forces. Given the very large surface area per unit mass, the amount of toxicant binding that can occur is considerable.

AC is known to bind poorly to alcohols, sugar alcohols (e.g. xylitol), glycols, strong acids, strong bases, metals, lithium, sodium, iron, lead, arsenic, fluorine, boric acid and many other inorganic materials.

Figure 1. The authors stress that AC and SDAC are unlikely to be effective with xylitol poisoning cases (courtesy of Anne Fawcett)

The surface of AC is capable of oxidative/reactive chemical reactions. However, these reactions require the presence of an aerobic environment, and are likely to be insignificant within the SDAC context.

Efficacy

To produce toxicologically significant effects, SDAC should be administered as soon as possible following ingestion; ideally within 30 minutes. Administration after 60-120 minutes is less likely to result in clinically significant effects, except in the cases where gastric concretions are formed (e.g. delayed release preparations), when there is delayed gastric emptying or possibly with extremely acutely toxic materials (assuming survival is a possibility at 1 hour post-ingestion). Studies in normal human volunteers using therapeutic and supratherapeutic doses have demonstrated that SDAC is capable of reducing the AUC by about 90% if administered within < 5 minutes following dosing.[1, 2] However, the individual variation is extremely large, ranging from about a 5% reduction in the AUC to a 100% reduction.[1, 2] **By 30 minutes post dosing, the average reduction in AUC is 50% and by 60-120 minutes, the reduction is about 20-30%. Again, the individual variation at the different time points is large and for the most part, unexplained.**

Rapid orodispersible tablets (available for paracetamol and aspirin) or other rapid release formulations are likely to decrease the therapeutic window for SDAC effectiveness.

Despite having a sound theoretical underpinning and supporting data from studies in normal humans, there is surprisingly little data from actual poisoning events. Actual clinical cases typically involve oral exposures many times higher than those used in the available human volunteer studies and there are potentially large differences in the toxicokinetics of these different situations (notably the predominance of saturation/0 order kinetics in cases of poisoning as well as the potential for progressive decay of the excretory mechanisms due to toxic effects).

Significant recent data from actual poisoning cases indicate important features: (a) different toxins/toxicants are affected by SDAC in different ways; (b) a reduction in AUC does not necessarily translate into clinical benefit; and (c) SDAC is only likely to increase the clearance of toxins/toxicants that have long half-lives under poisoning conditions.

Two well-designed randomized human clinical trials that compared SDAC to supportive care alone are available. Neither of these trials demonstrated any benefit of SDAC treatment. The difficulty with these trials is that one excluded severe poisoning cases and only included cases where significant toxicity was unlikely; and the second study mostly examined cases of pesticide and oleander poisoning. It is thus difficult to apply the findings of these trials to the broad range of toxins/toxicants that are important in veterinary medicine.

A third group of clinical studies primarily focus on the effects of SDAC on paracetamol poisoning. SDAC does appear to significantly reduce the absorption of paracetamol if the procedure is performed within 2 hours of ingestion of a substantially toxic dose.

A fourth group of small clinical studies involving the tricyclic antidepressants failed to find any significant benefit of SDAC, and formal toxicokinetic analysis was not performed.

Risks

Overall, SDAC is a low-risk procedure. Despite being an unpleasant tasting material, AC is not a cause of increased risk of emesis.

The single greatest risk of SDAC treatment is aspiration pneumonia. Assuming no clinical error (i.e. AC is administered into the stomach and not into the lungs by iatrogenic error), there are two basic scenarios in this situation: (a) the risk that SDAC increases the risk of aspiration; and (b) whether or not AC aspiration in addition to gastric contents increases the severity of the pneumonitis.

Human clinical trial data demonstrates that SDAC is not a risk factor for aspiration pneumonia. Anecdotal veterinary clinical data supports this position in dogs and cats provided that the gag and swallowing reflexes remain intact.

The bad news is that aspiration of AC is substantially worse than aspiration of gastric contents alone. Aspiration of AC will result in significant pulmonary injury.

Gastrointestinal obstruction, significant serum ion disturbances, a ruptured viscus plus peritonitis and rectal ulceration are often listed as potential complications of SDAC, but appear to be statistically rare occurrences.

Desorption of the toxin/toxicant is often cited as a potential risk of SDAC to the point that this effect has reached almost mythological status. Surprisingly there is effectively no *in vivo* evidence that AC desorption has any clinically or toxicologically significant impact.

The justifications for addition of a small dose of sorbitol to AC are commonly cited as: (a) improving the taste and thus ability to ingest the material (irrelevant if administered by nasogastric tube); (b) to reduce the risk of GI obstruction (which is extremely low to begin with); and (c) to reduce the risk of toxin/toxicant desorption from AC (and there is little actual *in vivo* evidence to support that this a significant actual real-world issue with SDAC). *Administration of sorbitol based on the principle of catharsis is completely discredited in clinical toxicology.*[3]

Contraindications

Altered consciousness and lack of airway security are the two most significant contraindications for SDAC. In both of these situations, endotracheal intubation will reduce the risk provided the ingested material does not have an inherently high aspiration risk. **SDAC should not be used if the ingested material has an inherently high aspiration risk such as light petroleum distillates or with materials that have corrosive potential.**

The other group of contraindications involves altered integrity of the GI wall and propensity for hemorrhage.

SDAC has the disadvantage of potentially obscuring endoscopic visualization.

A Suggested Protocol For SDAC

- SDAC is always a lower priority than stabilization (airway, breathing, circulation) and administration of effective antidotes.
- Carefully weigh the risk:benefit ratio of the procedure for the individual case. Do not perform SDAC if any contraindicating factor is present.
- Although recent evidence demonstrates that activated charcoal *per se* does not induce vomiting, some practitioners may prefer to administer a parenteral antiemetic agent prior to SDAC (e.g. maropitant citrate 1 mg/kg SC).
- Depending on the brand used, the activated charcoal may come as a powder, granules or as a pre-prepared aqueous slurry (with or without sorbitol; the author's personal preference) or gel. Powder or granulated activated charcoal should be prepared as an aqueous slurry/suspension by mixing 1g of the activated charcoal with 6-7 mL of water. When mixing activated charcoal powders, a dust mask should be used to prevent inhalation of the material. Eye protection is also warranted due to the risk of AC abrasions to the corneas.
- If dosing by mouth is possible, administer 1-3 g of activated charcoal USP. /kg body weight (same dose irrespective of species) as an aqueous slurry using a syringe (with or without a mouth gag). If dosing by mouth is not possible, then the slurry can be administered using either an orogastric or nasogastric tube.

The author's personal preference (provided that the gag reflexes are intact) is to use a fine bore nasogastric/nasoesophageal tube since this can be left in place in case repeated activated charcoal administration is desired. Furthermore, samples of gastric contents for diagnostic testing can be collected via nasogastric aspiration *before* the activated charcoal is administered. Using a correctly placed nasogastric tube also helps ensure getting the activated charcoal into the correct place and is often less messy than other methods. Typically the author will use a 3.5 or 5 Fr soft feeding tube (red rubber, polyurethane or silicone – avoid polypropylene as they kink). Larger nasogastric tubes are generally not needed unless very thick suspensions are used (they are also not needed for nasogastric aspiration since the objective is to obtain samples of gastric fluids, rather than solids). A couple of drops of ophthalmic examination topical examination local anesthetic (e.g. proparicaine 0.5%) in the nostril as well as liberal use of an aqueous lubricant will facilitate tube placement. Beware of using large amounts of topical anesthetics as the agent may run back into the pharynx and anesthetize the arytenoid cartilages, disrupting the gag reflexes. The tube is passed through the ventral nasal meatus. Great care should be taken to avoid traumatic passage of the tube. After the activated charcoal has been administered through the nasogastric tube, a small volume of water should be used to flush the tube. The tube can be fixed in lace with tape/sutures as required. An Elizabethan collar is recommended.

It should be emphasized that provided that the activated charcoal ends up in the stomach and no aspiration occurs, there is no perfectly correct method for administration for SDAC. The authors strongly suggest that practitioners should develop a technique with which they are personally comfortable and regularly practice the technique.

- Commercial activated charcoal preparations (e.g. ToxiBan®, Liqui-Char®, UAA-Gel®) have product-specific dose rates – ▶

check the package before using. Some of these preparations are relatively viscous gels (e.g. UAA Gel®) and are thus less amenable for use with a fine-bore nasogastric tube.

- If at all possible, SDAC should be administered without sedation in order to preserve the gag reflexes.
- Care should be taken not to get activated charcoal in the eyes of either the patient or the treatment team because of the risk of corneal abrasions.

Some Recommendations

To have at least the potential for clinical benefit, SDAC should be: (a) administered as soon as possible following ingestion of a toxin/toxicant that is known to bind effectively to AC *in vivo*, and preferably within 30 minutes (although at least some benefit after 60 minutes cannot be categorically excluded); (b) a sufficient dose of AC of at least 10 times that of the ingested dose of toxicant or at least 1g/kg body mass is used; (c) administered in cases where life-threatening toxicity is not easily treated by supportive care and/or a specific antidote; and (d) where relevant risk factors are not present.

Within the treatment context, SDAC does not take precedence over stabilization (i.e. airway, breathing and circulation in that order) or administration of known effective antidotes. SDAC is not a panacea, a 'universal antidote to poisoning' nor is SDAC a substitute for effective supportive and antidotal treatment.

Remember; treat the patient and not the poison.

References

[1] Isbister GK, Kumar VV. Indications for single-dose activated charcoal administration in acute overdose. *Curr Opin Crit Care.* 2011 Aug; 17(4):351-7.

[2] Chyka PA, Seger D, Krenzelok EP, Vale JA; American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position paper: Single-dose activated charcoal. *Clin Toxicol (Phila).* 2005;43(2):61-87.

[3] Anon. Position paper: cathartics. *J Toxicol Clin Toxicol.* 2004;42(3):243-53. Review. Erratum in: *J Toxicol Clin Toxicol.* 2004;42(7):1000.

2. THE USE OF MULTIPLE DOSE ACTIVATED CHARCOAL FOR TREATMENT OF POISONING IN SMALL ANIMALS

Introduction

Multiple dose activated charcoal therapy (MDAC) involves the repeated (i.e. more than 2 doses) oral (or intra-gastric) administration of activated charcoal with the objective of increasing the whole body elimination of a toxicant that undergoes entero-systemic, enterohepatic, entero-gastric or entero-enteric cycling. An additional rationale is that MDAC should 'mop up' any non-absorbed xenobiotic still remaining in the gut. MDAC has also been referred to as 'gastrointestinal dialysis'.

Indications for MDAC treatment

MDAC is commonly promoted for all instances where the systemic exposure of a xenobiotic is extended by enterohepatic cycling (or other forms of enteric cycling). However, the currently available data indicates that MDAC performed the best with agents that:

- Have a prolonged elimination half-life following overdose; AND
- Have a small volume of distribution (i.e. < 1L/kg body weight, indicating that sequestration is not occurring).

Currently available human data have demonstrated some possible benefit for MDAC treatment based on the evaluation of clinical outcomes, in cases of poisoning by the following agents:

- Cabamazepine
- Dapsone
- Phenobarbital
- Theophyllin
- Possibly yellow oleander (*Thevetia peruviana*) poisoning. MDAC is attractive under these circumstances because it is possibly a cheaper alternative when digoxin F'ab antibody therapy cannot be afforded or as a supplement to antibody therapy. However, the available data regarding clinical effectiveness is mixed. Based on the available data, MDAC alone is not a suitable replacement for digoxin F'ab antibody therapy.

  It should be clearly noted that the effectiveness (or lack thereof) of MDAC for treatment of yellow oleander cardenolides cannot be readily translated to other toxic cardenolides. This is because of the significant toxicokinetic differences between different members of the cardenolide toxin family.

However, the above information should be treated with caution: there is little available, overwhelmingly convincing, data that indicates that MDAC substantially reduces morbidity and mortality in poisoned patients.

In addition to the above, MDAC has been demonstrated to reduce the total body clearance of; however the clinical benefit(s) of this effect remain unproven:-

- Paracetamol
- Digoxin
- Digitoxin
- Disopyramide
- Nadolol
- Phenylbutazone
- Piroxicam
- Quinine
- Sotalol
- Possibly valproic acid (conflicting data and small data sets)
- Tricyclic antidepressants (Note: although the apparent half-life may be reduced, there are sound pharmacological reasons that indicate that such changes are unlikely to be clinically beneficial during an overdose situation)
- Possibly salicylate (conflicting data and small treatment effect)

Indications for MDAC Treatment

The indications for MDAC treatment are:-

- Good clinical judgment regarding the presence of contraindications to MDAC within a risk:benefit paradigm.
- The effectiveness of alternative methods of treatment (e.g. the prompt use of n-acetyl cysteine for paracetamol poisoning).
- The likelihood of significant clinical benefit. This is more probable with overdoses of carbamazepine, dapsone, phenobarbital, quinine or theophylline that would result in death without clinical intervention. The case for yellow oleander poisoning is less well established. However, MDAC is possibly one of the few economically practical forms of treatment where large numbers of grazing animals have been affected. In valuable animals and in small companion animals there is insufficient data regarding the clinical effectiveness of MDAC versus digoxin F'ab antibody fragments or combined MDAC/antibody therapy. Perhaps the best that can be said is that MDAC is relatively cheap, has a low rate of serious side effects and ***might*** have some positive effect.
- ***Possibly*** in the case of drugs that undergo substantial enterohepatic (or other forms of enteric cycling) ***provided*** that the volume of distribution is < 1 L/kg. Again, perhaps the best that can be said is that MDAC is relatively cheap, has a low rate of serious side effects and ***might*** have some positive effect.

Contraindications and Risks for MDAC Treatment

Contraindications for MDAC include:

- Any clinical situation where airway security may be compromised. Repeated endotracheal intubation, while reducing the risk, may not be practical for MDAC treatment. Xenobiotic-induced vomiting may increase the risk of aspiration following MDAC.
- Any clinical situation where altered consciousness is present.
- The risk of aspiration associated with MDAC may (unproven) be higher in young animals.
- **MDAC should not be used if the ingested material has an inherently high aspiration risk such as light petroleum distillates.**
- **AC is known to bind poorly to alcohols, sugar alcohols (e.g. xylitol), glycols, strong acids, strong bases, metals, lithium, sodium, iron, lead, arsenic, fluorine, boric acid and many other inorganic materials.**
- **Any clinical situation where there may be altered integrity of the gastrointestinal wall or a propensity for gastrointestinal hemorrhage.** MDAC has been associated with rectal ulceration and subsequent massive hemorrhage.
- Any clinical situation that increases the risk of substantial constipation, obstipation or bowel obstruction. **Although rare, the formation of charcoal bezoars is a potentially serious complication.**
- MDAC has the disadvantage of potentially obscuring endoscopic visualization.
- Very rarely, corneal abrasions have been reported following activated charcoal dosing. This complication is preventable by the use of good technique.
- There is a small risk of exacerbating hypernatremia and inducing hypermagnesemia.
- Appendicitis is a rare complication of MDAC in humans.

The MDAC Procedure

- The optimum dose of activated charcoal is currently unknown. However, a dose rate of 10 times the ingested toxin/toxicant mass as been suggested. A dose of 0.7 – 1.4 g/kg BW administered at a rate of greater than 0.17 g/kg BW/hour has been suggested. This dose and rate of administration may need to be reduced in smaller animals.
- While activated charcoal by itself does not increase the risk of vomiting, it is currently recommended practice to administer an intravenous antiemetic concurrently with MDAC.
- MDAC is often best performed via a small-bore nasogastric tube. However, repeated bolus dosing can be used (although this may [unproven] increase the risk of aspiration).
- The co-administration of a **mild** cathartic is currently not recommended, particularly in smaller animals. This is due to the risk of serious fluid and electrolyte imbalances associated with multiple administration of cathartics.
- If a cathartic is going to be administered, it should be limited to the first dose of activated charcoal. **Repeated dosing with cathartics (even sorbitol) should be avoided.**

Editor's Note: *Practitioners are urged to report their experiences with multiple dose activated charcoal (MDAC) to AVPMA. While each case is just an observational study, a growing collection might raise some research hypotheses for further exploration.*

3. CATHARSIS AND ENEMAS FOR THE TREATMENT OF ORAL POISONING IN DOGS AND CATS

The purpose of this short commentary is to review the rationale for the use of catharsis (oral cathartics, enemas and colonic irrigation) following oral poisoning and to present the current generally accepted case against their usage within this context. The contraindications and potential adverse effects of these techniques are also reviewed.

We have specifically excluded techniques like whole bowel irrigation (administration of a large volume of isotonic with the purpose of flushing out the entire gastrointestinal tract) because the technique is not the same as catharsis and the technique is rarely available or used in veterinary medicine.

Rationale for the use of cathartics

The usual basis for the use of cathartics (including the use of enemas) is to accelerate the expulsion of a toxicant from the gut with the hope of reducing absorption. The expected end result is a reduction in systemic exposure (i.e. area under the curve or AUC).

An unavoidable fundamental concept that underlies the rationale for the use of cathartics is that the method is mostly likely to benefit patients who have absorbed acutely toxic materials that are absorbed very slowly or that undergo very slow, but substantial enterohepatic cycling or that undergo slow reabsorption in the lower bowel. The vast majority of toxicants commonly encountered in veterinary medicine do not have such toxicokinetic characteristics; most are rapidly absorbed in the upper GI tract and their toxicokinesis is unlikely to be affected by catharsis.

A second common pattern of use involves the combination of a low dose, mild cathartic plus single dose activated charcoal (SDAC) treatment. The justifications for addition of a small dose of sorbitol to AC are commonly cited as: (a) improving the taste and thus ability to ingest the material (irrelevant if administered by gastric or nasogastric tube); (b) to reduce the risk of GI obstruction; and (c) to reduce the risk of toxicant desorption from AC.

Given that very few known toxicants undergo significant absorption in the large intestine, there is no clear basis for the use of enemas and/or colonic irrigation to reduce systemic exposure to poisons (except in some very rare and specific circumstances).

Common techniques

Oral dosing using osmotic cathartics, colonic irrigation and enemas have been commonly employed in veterinary medicine. The osmotic cathartics used typically have fallen into two main groups: (a) saccharide cathartics such as sorbitol and (b) saline cathartics such as magnesium citrate, magnesium sulfate and sodium sulfate. Occasionally, these techniques have been supplemented by the use of various laxative preparations. Various enema preparations have been used, with the exception of phosphate enemas that are known to be toxic in cats. Colonic irrigation is often performed with warm water.

The case against the routine use of catharsis for the treatment of oral poisoning

We cannot say it any better than the joint statement of the American Academy of Clinical Toxicology and the European Association of Poisons Centers and Clinical Toxicologists:

'The administration of a cathartic alone has no role in the management of the poisoned patient and is not recommended as a method of gut decontamination. Experimental data are conflicting regarding the use of cathartics in combination with activated charcoal. No clinical studies have been published to ▶

investigate the ability of a cathartic, with or without activated charcoal, to reduce the bioavailability of drugs or to improve the outcome of poisoned patients. Based on available data, the routine use of a cathartic in combination with activated charcoal is not endorsed. If a cathartic is used [in combination with activated charcoal], it should be limited to a single dose in order to minimize adverse effects of the cathartic.'[1]

Currently available data for cathartics-alone treatment in humans indicate:

- Magnesium sulfate catharsis does not significantly affect the serum concentrations of lithium and salicylate when administered 30 minutes following oral dosing.
- Sodium sulfate catharsis does not significantly affect the total urinary recovery (a way of indirectly measuring the fraction of the dose absorbed) of paracetamol and its metabolites and did not affect the total urine recovery of salicylate.
- Sorbitol catharsis results in an increased plasma C_{Max} and shorter plasma T_{Max} for theophylline, suggesting that the catharsis actually *increased the speed of absorption from the GI.*

There are currently no clinical or other studies that demonstrate that catharsis alone reduces toxicant bioavailability and improves clinical outcomes.

The claim that catharsis alone produces 'benefit' for the treatment of oral poisoning has no scientific basis.

The currently available in vivo data for the use of a single low dose of a cathartic in combination with SDAC is mixed. The available studies have demonstrated either a relatively small beneficial effect or no significant effects at all.

It should be noted that there is some evidence that oral dosing with sorbitol plus SDAC may actually *increase* systemic exposure following oral paracetamol poisoning in dogs. When sorbitol + SDAC was compared with administration of activated charcoal alone, the paracetamol AUC was *75% higher and the plasma C_{Max} was 80.4% higher in dogs treated with sorbitol + SDAC when compared with dogs that were only treated with SDAC.[2] In this study, the various GI decontamination procedures were performed at 1 minute (!) following oral dosing – an optimal situation that is never really going to occur in real-life veterinary practice.* The same study demonstrated that SDAC alone and without a concurrent cathartic reduced the paracetamol AUC by 93% whereas the administration of cathartics (sorbitol or castor oil) at 1 minute (!) post exposure reduced the AUC by 15-30%. Thus, even under optimal conditions that are never going to occur in veterinary practice, catharsis alone had minor to negligible (in terms of clinical outcomes) benefit. *It should be noted that as the exposure to treatment interval increases from 1 minute (as in this study) to 30 minutes, the effects of cathartics alone decreases to zero.*

Veterinarians should be aware that combining sorbitol with SDAC for the treatment of paracetamol poisoning in dogs *may actually make the situation worse* compared with the use of SDAC without a cathartic.

There is simply no scientific evidence that enemas and/or colonic irrigation produce any clinical or therapeutic benefit in the treatment of oral poisoning. The colonic irrigation/enema paradigm dates back to the Egyptian and Greek 'auto-intoxication' hypothesis that has been progressively disproved since the original scientific challenge to this theory in 1919. *Colonic irrigation has no known medical value and risks damage to the rectum or bowel.*

The techniques of colonic irrigation and/or the use of enemas for the treatment of oral poisoning, quite literally, stink.

The same conclusions apply to the use of these techniques in veterinary medicine. In point of fact, their use in the treatment of any condition that reduces the efficacy of the clotting system is clearly contraindicated because of the risk of hemorrhage (particularly with the anticoagulant vitamin K antagonist rodenticides). Other significant risks include significant electrolyte imbalances, bowel perforation, bowel infections and rectal prolapse. These techniques produce no benefit and have significant risks.

Contraindications and risks

The contraindications for the use of catharsis or a single low-dose cathartic in combination with SDAC are:

- Absent or diminished bowel sounds.
- Recent abdominal trauma.
- Recent bowel surgery.
- Intestinal obstruction.
- Risk of intussusception or prolapses.
- Intestinal perforation or risk of intestinal perforation.
- Ingestion of a corrosive substance.
- Volume depletion.
- Hypotension.
- Significant electrolyte imbalances.
- Magnesium-containing cathartics should not be administered to patients with evidence of renal failure, renal insufficiency or heart block.
- Cathartics should not be used in very young animals nor should they be used in very old animals.

The potential risks associated with the use of cathartics (with or without SDAC) are:

- Deyhydration.
- Hypernatremia, particularly with sodium-containing cathartics.
- Hypermagnesemia.
- Hypotension.
- Vomiting.
- Abdominal pain.
- GI hemorrhage.

Some recommendations

- Treat the patient, not the poison.
- GI decontamination is almost always a lower clinical priority than stabilization (airway, breathing and circulation in that order) and the administration of known effective antidotes.
- In general, catharsis, colonic irrigation and enemas have no place in the routine management of oral poisonings. Catharsis alone, colonic irrigation and enemas have no proven benefits and thus have poor risk:benefit ratios.
- If a mild, low-dose cathartic is going to be used in combination with SDAC, then its use should be limited to a single dose.
- Repeat dosing with combination preparations containing sorbitol and activated charcoal is unwise.
- The use of sorbitol should be avoided in cases of oral paracetamol poisoning in dogs.

Reference

[1] Anon. Position paper: cathartics. *J Toxicol Clin Toxicol.* 2004;42(3):243-53. Review. Erratum in: *J Toxicol Clin Toxicol.* 2004;42(7):1000.

[2] Van de Graaff WB, Thompson WL, Sunshine I, Fretthold D, Leickly F, Dayton H. Adsorbent and cathartic inhibition of enteral drug absorption. *J Pharmacol Exp Ther.* 1982 Jun;221(3):656-63.

Cat with Permethrin film clip (courtesy of ISFM)

4. GASTRIC LAVAGE AS A TREATMENT FOR POISONING IN DOGS AND CATS: THE GOOD, THE BAD AND THE DOWNRIGHT UGLY

Recent controversy has occurred in Australia and New Zealand regarding the routine use of gastric lavage in cases of dog and cat poisoning. Thus a short review of the principles underlying this procedure, contraindications for the procedure and its risk:benefit ratio are timely.

In this short commentary, the term gastric lavage is defined as: 'passage of a large bore orogastric tube and the sequential administration and aspiration of small volumes of liquid with the intent of removing toxic materials present in the stomach.'[1] This technique has been in use for well over 180 years.

We have very specifically excluded techniques that involve 'the use of a small bore nasogastric tube when used only to aspirate stomach contents or to administer activated charcoal.'[1] We have also specifically excluded techniques that involve focused endoscopy-assisted aspiration and/or lavage. There is also a deliberate focus upon the use of gastric lavage in dogs and cats and we have specifically excluded discussion on the use of the technique in standing horses and ruminants.

The fundamental objective of gastric lavage in cases of oral poisoning is to remove as much of the ingested material (toxicologists call this 'oral exposure' rather than dose because in toxicology the concept of 'dose' specifically refers to the concentration of the agent and its toxicological site(s) of action) from the stomach, thus reducing the amount of material available for absorption into the systemic circulation. Thus, it follows, that the material must be still present in the stomach in order for gastric lavage to actually work.

The Good (the benefit side of the ratio):-

Studies in dogs and in humans with a variety of ingested materials (paracetamol, ibuprofen, benzodiazepines, sodium salicylate, barium sulfate, aspirin, ampicillin, cyanocobalamin, temazepam, verapamil, moclobemide, technicium 99-sulfur colloid, and thiamine), human multicenter clinical trials, clinical studies and epidemiological studies of oral poisonings over the last 50 years have consistently demonstrated the following:-

- Under the very best experimental conditions, the area under the curve (AUC, a measure of total systemic exposure) can be reduced by 30-50% and the plasma maximum concentration (C_{max}) can be reduced by 0 – 50%, *provided that effective gastric lavage is performed within about 10-30 minutes following oral dosing.* The term 'effective gastric lavage' means that the technique is performed correctly. In other words: (a) a sufficient volume of an appropriate lavage fluid is used; (b) a sufficiently large and appropriately fenestrated lavage tube is used; (c) the ingested material is either sufficiently soluble in the lavage fluid or is finely enough divided to pass through the lavage tube; and (d) the procedure is performed by an experienced practitioner with good technique.
- *Longer exposure to treatment intervals usually results in a rapid decline in the efficacy of gastric lavage. By 60 to 120 minutes post-ingestion, the efficacy of the technique is generally poor unless the material results in delayed gastric emptying, undergoes slow dissolution (time release) or forms gastric concretions (e.g. chocolate). The implications of this are that unless the ingested material is highly toxic (e.g. lily ingestion in cats or some other situation where small reductions in AUC and/or Cmax are likely to result in substantial clinical differences – a relatively rare situation in clinical toxicology), little to no practical therapeutic benefit is likely after about 60 minutes post-exposure.*
- Contrary to early reports, gastric lavage does not propel the stomach contents into the small bowel (which was claimed to increase toxicant absorption).
- Gastric lavage may yield useful samples for toxicological testing.
- Unfortunately, high quality, well-controlled studies on common canine poisons such as metaldehyde, carbamates and the vitamin K antagonist rodenticides are not available.

And Now for the Bad and the Downright Ugly (the risk side of the ratio):-

- *It is critical that the treating veterinarian and nearby staff wear appropriate personal protective equipment when performing the procedure. This means gloves that are resistant to the suspected toxicant, a protective apron, a long sleeve shirt, gown or coat, suitable footwear, eye protection and in a number of cases, adequate respiratory protection (including a full-face gas mask with appropriate cartridges in the case of phosphine gas sources or cyanide).* Gastric lavage potentially exposes the treating veterinarian and any nearby staff to significant levels of toxicants. The classical example of this is the ingestion of phosphine gas sources (such as zinc or aluminum phosphide) by dogs and cats. Acute phosphine poisoning of veterinarians (and human emergency room staff) under these circumstances has been repeatedly reported.

Editor's Note: *Go to the ebook to rollover to read C&T No. 5144. RODENTICIDE TOXICITY ALERT! Reports of zinc phosphide poisoning and potential new management strategies. Sept 2011.* C&T Series*, Issue 264, pgs 15-16.*

- Patients that present for treatment within optimal treatment window of 0-30 minutes (possibly extending to 60 minutes with some toxicants and circumstances) following ingestion of a poison are the *exception rather than the rule*.
- The effectiveness of gastric lavage (i.e. its ability to remove material from the stomach), even under the best of circumstances is highly variable and operator-dependent. Thus there is substantial case-to-case variability and variability between practitioners.
- Under the best of conditions, there is substantial toxicant-to-toxicant variability in the effectiveness of gastric lavage, even when the procedure is performed within the optimal treatment time window.
- Even though modest reductions in AUC and C_{max} can be accomplished under ideal circumstances and when the technique is used within the optimal time window, these reductions have generally been shown to be of minimal to no clinical toxicological benefit in the majority of cases (the exception being for extremely toxic materials combined with short exposure to treatment intervals). As a general rule of thumb, at least a 50% decrease in AUC and/or C_{max} is required for clear clinical benefit to occur. Such large decreases are only likely to occur if gastric lavage occurs within 15 to 30 minutes following ingestion and then only in some individuals (given the wide inter-individual variability).
- Gastric lavage is of absolutely no use for treatment of poisonings where significant amounts of the toxicant are rapidly absorbed through the gastric mucosa (short chain alcohols such as ethanol or propanol/isopropanol; short chain glycols such as ethylene glycol; weak acids such as aspirin because they will be present mainly in their non-ionic, more lipophilic, state in the low pH of the stomach).

- Likewise, gastric lavage is of unlikely benefit for treatment of poisonings due to toxicants that rapidly trigger gastric emptying.
- In cases where the toxicant triggers emesis, gastric lavage is very unlikely to provide any significant additional benefit.
- Human clinical studies and clinical trials have demonstrated that under most circumstances gastric lavage results in at best no benefit and often results in worse clinical outcomes (i.e. higher morbidity). We acknowledge that there is a limited amount of anecdotal evidence suggesting that gastric lavage might be less damaging in dogs/cats compared with humans.
- Human clinical studies and clinical trials have demonstrated that omission of gastric lavage is not associated with poorer patient outcomes. Despite the decline in use of gastric lavage in cases of deliberate self-poisoning in humans over the last 2 decades (its use has declined from above 50% of cases to less than 0.7% of cases), there has been no worsening in clinical outcomes.
- Human data demonstrates that gastric lavage is associated with worse outcomes (particularly a higher occurrence of aspiration pneumonia) and increased admissions into intensive care facilities.
- **Provided that the putative toxicant binds effectively to activated charcoal, the reductions in AUC and C_{max} seen with gastric lavage are, from a practical clinical toxicology perspective, essentially the same as those observed with single oral dose activated charcoal administration at the same exposure to treatment interval. *The big difference is that oral single-dose activated charcoal therapy is significantly less invasive, has substantially fewer contraindications and is significantly less likely to result in iatrogenic injury compared with gastric lavage.* Single dose activated charcoal treatment is also likely to be cheaper than gastric lavage.**
- There are common and important contraindications for the use of gastric lavage: (a) the single greatest problem is airway security. Even the presence of a cuffed endotracheal tube is *not a guarantee of 100% airway security!* (b) gastric lavage, even with a cuffed endotracheal tube in place, is contraindicated in cases of low viscosity hydrocarbon distillate ingestion (the cutoff for viscosity in terms of aspiration risk is about that of Johnson's Baby Oil, although some aspiration may still occur with thicker petroleum jellies); (c) gastric lavage should always be performed with a cuffed endotracheal tube in place. This means that the poisoned patient must be in a suitable physiological state that safely allows for a level of sedation/anesthesia consistent with intubation. This may not be possible in significantly debilitated animals; (d) gastric lavage is contraindicated in cases where gastric hemorrhage is present because it may worsen the bleeding; (e) gastric lavage is contraindicated where there is the potential for gastrointestinal perforation (e.g. ingestion of corrosive materials).
- There are important complications that are associated with the use of gastric lavage: (a) aspiration pneumonia is always an associated risk, particularly if low viscosity hydrocarbons have been ingested, or if the airway protective reflexes are depressed or if the patient has not been intubated. Aspiration may still occur even if the patient has been intubated with a cuffed endotracheal tube; (b) significant laryngospasm and associated hypoxia may occur, particularly in semi-conscious patients; (c) the technique can produce tension pneumothorax; (d) the technique results in increased heart rate, atrial and ventricular ectopic beats and transient ST elevation. Thus there is a small risk of triggering or exacerbating cardiac arrhythmias in susceptible patients; (e) esophageal and gastric perforations can occur; (f) there is a significant risk of hypernatremia if saline or intravenous infusion preparations are used as the lavage fluid; (g) water intoxication can occur, particularly in smaller patients; and (h) the technique can result in conjunctival hemorrhages.
- *It is critical to note that failure to detect ingested material in lavage fluid ('negative or poor lavage return') does not mean that significant ingestion of a toxicant has not occurred. In other words, negative results may be meaningless!*

The Conclusions:-

We can't say it any better than the American Academy of Clinical Toxicology and the European Association of Poisons Centers and Clinical Toxicologists[2]:

'At present there is no evidence showing that gastric lavage should be used routinely in the management of poisonings. Further, the evidence supporting gastric lavage as a beneficial treatment in special situations[3] is weak, as is the evidence to exclude benefit in all cases. *Gastric lavage should not be performed routinely, if at all, for the treatment of poisoned patients.* In the rare instances in which gastric lavage is indicated, it should only be performed by individuals with proper training and expertise.'

We would like to emphasise the following points:-

- Gastric lavage for the sole purpose of collecting toxicological samples is usually unjustified. Most cases of poisoning are given a presumptive diagnosis on the basis of the clinical history. The clinical history *always includes the original toxicant container (and most critically its label) and an attempt to clarify the exposure to treatment interval.*
- Always protect yourself and your staff first: *there is no excuse for inadequate personal protective equipment.* If necessary, get adequate training in its use.
- *In all cases, treat the animal and NOT the poison.* By this, we mean that in the majority of circumstances clinical stabilisation (i.e. airway, breathing, circulation in that order) and effective initial nursing/allied treatment generally takes priority over decontamination of the upper digestive tract.
- The administration of known effective antidotes will usually take priority over decontamination of the upper digestive tract.
- In our view, there is no substitute for good clinical judgment in relation to specific cases. *This includes clear, objective, rational and documented decisions that take into account the risk:benefit ratio in individual cases. Knowledge and good clinical judgment generally takes precedence over 'one-size fits all' standard operational procedures.*
- Gastric lavage is really only clearly indicated in a fairly uncommon set of circumstances: (a) a highly toxic material has been ingested (e.g. lilies in cats or paracetamol); and (b) the patient presents within the optimal treatment window (which may be extended if the toxicant forms gastric concretions or if it delays gastric emptying or if it is extremely toxic and small decreases in systemic absorption are likely to make big clinical differences); and (c) other less invasive and less risky techniques (e.g. single dose activated charcoal treatment, nasogastric aspiration) cannot be used for some reason; and (d) gastric lavage is not contraindicated; and (e) clinical circumstance and best clinical judgment indicate that the significant potential risks of the procedure are clearly outweighed by the potential benefits.
- Gastric lavage is *not a clinically innocuous procedure.* It is an invasive and potentially high risk procedure, particularly: (a) in significantly debilitated patients; (b) in patients that lack airway security; (c) in cases of light hydrocarbon ingestion; (d) in patients that are poor candidates for anaesthesia/sedation; (e) in patients that have ingested corrosive materials; and (f) in patients who may have reduced esophageal and gastric mucosal integrity.
- Negative findings following gastric lavage *does not categorically rule out poisoning*.
- Positive findings (i.e. detection or dyes, formulation remnants or remnants of carrier materials) following gastric lavage *does not mean that the procedure has been effective.* Most product formulations are designed to release the active ingredient relatively quickly in the gastrointestinal tract. The warm, acid environment of the stomach provides an efficient extraction system for most toxicants. Just because you detect the presence of a dye or even remnants of a carrier (e.g. the stained grain pellets) does not mean that gastric lavage has actually reduced systemic absorption – the active ingredient may already have been extracted and absorbed.
- To be effective, gastric lavage *requires significant clinical skill and regular practice.*
- If the toxicant is known to bind to activated charcoal and the patient presents within the optimal treatment window of 30-60 minutes[4] (assuming no delay in gastric emptying, no time release formulations and no gastric concretions), single dose activated charcoal treatment without prior gastric emptying is at least as effective as performing gastric lavage and is (in almost all cases) a lower risk procedure. In other words, single dose activated charcoal treatment will often have a better risk:benefit ratio than gastric lavage.
- There is very little evidence to support the routine use of gastric lavage in the treatment of oral poisoning in dogs and cats. It is rare for these patients to present for treatment within the optimal treatment window and usually the risk:benefit ratio will be poor (particularly if activated charcoal is effective and available).

References

[1] Benson BE, Hoppu K, Troutman WG, Bedry R, Erdman A, Höjer J, Mégarbane B, Thanacoody R, Caravati EM; American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position paper update: gastric lavage for gastrointestinal decontamination. *Clin Toxicol* (Phila). 2013 Mar;51(3):140-6.
Vale JA, Kulig K; American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position paper: gastric lavage. *J Toxicol Clin Toxicol.* 2004; 42(7):933-43.

[2] Benson BE, Hoppu K, Troutman WG, Bedry R, Erdman A, Höjer J, Mégarbane B, Thanacoody R, Caravati EM; American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position paper update: gastric lavage for gastrointestinal decontamination. *Clin Toxicol* (Phila). 2013 Mar;51(3):140-6.

[3] i.e. in cases of ingestion of highly toxic material and the patient presents for treatment within the optimal treatment window of less than 30-60 minutes or if gastric emptying is delayed or if the substance forms gastric concretions.

[4] For single dose activated charcoal treatment to work, it must come in direct physical contact with the ingested material in the stomach or it must substantially disrupt enterohepatic cycling.

5. CURRENT SUGGESTED PRACTICES FOR SKIN DECONTAMINATION AND THE CASE AGAINST SOLVENTS FOR DERMAL DECONTAMINATION

Decontamination of the skin remains a common procedure in small animal veterinary practice. To be effective and to have an acceptable risk:benefit ratio, proper technique is required. The currently generally accepted recommendations for dermal decontamination are as follows:-

- Always treat the patient and not the poison.
- The original product container and an intact label are often the single most useful pieces of diagnostic information in cases of poisoning. *Most cases of poisoning are diagnosed on the basis of the clinical history rather than other techniques.*
- Dermal decontamination is almost always a lower clinical priority compared with stabilisation/resuscitation (airway, breathing and circulation in that order) and the administration of known effective antidotes. Stabilisation and antidote administration should not be delayed by dermal decontamination. **It may be possible for all of these procedures to be performed concurrently, and often ideal.**

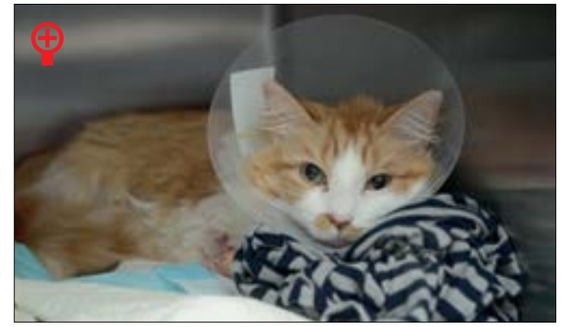


Figure 2. Elizabethan collar stops further dermal exposure (courtesy of Anne Fawcett)

- Dermal decontamination can expose the treatment team to significant levels of toxicants. Appropriate personal protective equipment (PPE) including chemical resistant gloves, a long sleeve coat and/or shirt, long pants, eye protection and closed footwear are the minimum PPE requirements. Some toxicants will require the use of a full-face gas mask with appropriate canisters. If in doubt, get adequate training in the use of PPE.

  Ill-advised and/or ill-prepared attempts at rescue and decontamination remain a significant cause of human toxicological casualties.
- Flushing of the skin surface with large volumes of water as soon as possible following exposure is recommended. *Particular care must be taken to prevent hypothermia during this treatment, especially with small animals. Veterinarians should take particular note that reducing body temperature actually increases the effects of pyrethroids and pyrethrins on the nervous system of mammals.* Part of the selective toxicity of these pesticides is based on the lower body temperature of insects compared with mammals. On the other hand, *it is also important to avoid flushing with water that is too warm, as this will produce dermal vasodilation, increased dermal blood flow and greater dermal absorption of the toxicants.*
- Several wash-rinse cycles of the affected area using a *mild* hand dishwashing detergent should be applied as soon as possible following exposure. Again, it is particularly important to avoid both hypothermia and washing/rinsing with materials that are too warm. *A particular emphasis is applied to the term 'mild' for a reason: strong detergents, strong surfactants, laundry detergents and machine dishwashing detergents must not be used because of their corrosive potential. It is also important not to go to extremes with this procedure. Even mild detergents cause some defatting injury to the skin in their own right.*
- Attempts to chemically neutralise acids and bases on the skin should be avoided because of the risk of thermal injury. The answer to this issue is dilution.
- There is some evidence that products such as Diphoterine may improve the outcomes of skin contact with corrosive materials in humans. However, large-scale trials are lacking and the safety properties of these products in domestic animals have not been assessed. ▶

- Specialised skin decontamination may be required for particular materials (e.g. the use of calcium gluconate gels for hydrofluoric acid exposures). The best source of information regarding this is your local poisons information service. Alternatively the US ASPCA Animal Poison Control Center: www.aspca.org/ should be contacted or the UK Veterinary Information Poisons Service: www.vpisuk.co.uk
- Substances that are adherent to the skin or hair (e.g. adhesives, polymers, bitumen, tar) or produce skin/hair stains should be left in place (and an Elizabethan collar used to prevent self-mutilation) unless they interfere with biological functions e.g. materials adhered to the eye-lashes or that interfere with breathing/eating/drinking/defecation/urination. If the materials interfere with biological functions, an attempt to remove the material can be made using very gentle treatment with a non-irritant, simple cosmetic emulsion. Substances such as Tween 80 and PEG 500 have also been suggested for this purpose. Vegetable oil and peanut butter have also been suggested for this use. However, care must be taken with all of these materials as they are not sterile and are good media for bacterial growth. Great care must be taken if these materials are to be used around the eyes. If this is not successful, more aggressive surgical interventions may be required. Again, it should be emphasized, particularly in the case of bitumen, that it is often better to leave adherent materials in place if possible.
- Eye exposures commonly co-occur with skin contamination. Eye exposures are often genuine ophthalmic emergencies: Seek specialist help as soon as possible.
- In general, the assessment and management of chemical skin burns is the same as for thermal burns.
- Dermal exposures may produce significant systemic toxicity (e.g. phenol, hydrofluoric acid, pyrethrins/pyrethroids) that requires additional treatment and support.
- Solvents e.g. turpentine have no place in dermal decontamination.

Light petroleum distillates and other strong solvents have no place in skin decontamination procedures. Veterinarians often fall into the trap of using these materials when confronted with materials that adhere to the skin/hair coat or that produce skin staining (such as paints, wood stains, glues/adhesives, tar/bitumen). *While the presence of adherent material or stains may be upsetting to an owner, this situation is far better than the risk of inducing chemical skin burns and possible systemic toxicity that are associated with light petroleum distillates or other strong solvents.*

The case against the use of light petroleum distillates, such as mineral turpentine, in dermal decontamination can be summarised as follows:-

- A complete lack of evidence of any clinical efficacy (except in very specialised and rare circumstances).
- The very real risk of combustion and associated thermal injury. These materials, particularly Stoddard solvents, will spontaneously combust when in direct contact with the cotton fibers commonly used in skin dressings. This spontaneous combustion is an oxidation reaction that needs no external ignition source.
- The very real risk of skin irritancy and defatting injury.
- The risk of causing a freeze injury when volatile hydrocarbons are used.
- The very real and substantial risk of producing a chemical skin burn, particularly with sustained or repeated contact.
- The destruction of the barrier function of the skin that may actually increase skin absorption.
- The combination of washing with a detergent and wiping with a light petroleum distillate may exacerbate the skin damage.
- The very real risk of aspiration associated with grooming behaviours.
- The risk of systemic absorption of the solvent and the possibility of systemic effects.

If animals have been exposed either dermally or via other routes, veterinarians should be aware of the key features of light petroleum distillate intoxciation.

The light petroleum distillate toxidrome has at least 4 components:-

- The single most critical hazard is hydrocarbon aspiration pneumonia. In dogs and cats, this can occur either by ingestion (or malicious oral dosing) or by the animal grooming the contaminated hair coat. If these petroleum distillates are present at ≥ 10% and overall the product has a kinematic viscosity of less than ≤ 20.5 mm^2/s at 40°C (the viscosity cutoff is approximately the viscosity of 'Johnson's Baby Oil') there is a significant risk of aspiration. Veterinarians should note that while clinical signs such as petroleum smelling breath, coughing, gagging etc are indicators of petroleum distillate aspiration, the majority of cases will display few or no clinical signs before the onset of respiratory distress at several hours to several days following exposure (typically 24-48 hours post-exposure). Radiological changes are also typically delayed and slow to develop. For the most part, petroleum aspiration pneumonia is a quiet, insidious process involving progressive respiratory distress, often with fatal respiratory failure at 24-48 hours post-exposure (depending on the aspirated volume).

  Veterinarians should take note that any medication or procedure that suppresses the gag or swallowing reflexes will increase the risk of hydrocarbon aspiration pneumonia.
- Narcosis and other central nervous system (CNS) effects are the second of the classical tetrad of effects associated with light petroleum distillates. Evidence of CNS effects may include restlessness, head pressing, episodic stargazing, pupillary dilation, ataxia, generalised cognitive depression and so forth. The severity of these effects does show some patterns in relation to composition: as a 'general rule of thumb', the higher the aromatic content, the more potent the narcotic properties and the more severe the CNS effects. It is the CNS and narcotic properties of the light petroleum distillates (combined with their low cost) that make them attractive drugs of addiction.
- Skin, digestive mucosae and respiratory mucosae irritancy is the third feature. The modes of action for these effects are: (a) defatting injury to the skin and other membranes and (b) solvent action on cell membranes. As 'general rules of thumb' the longer the contact with the skin and the greater the aromatic content (i.e. the greater the product's solvent action), the more severe the skin/mucous membrane damage is likely to be. Some very light petroleum distillates that evaporate very rapidly from the skin surface (e.g. the C3-C6 distillates in petrol) can produce freeze injuries to the skin.
- The final member of the classical hydrocarbon solvent tetrad of toxicological effects is sensitisation of the myocardium to catecholamine-induced arrhythmias. As 'a general rule of thumb', myocardial effects usually require exposures sufficient to produce narcosis/CNS effects. There are also relatively clear structure-activity relationships for effects on the myocardial electrical system: (a) in general, the higher the C4-C5 aliphatic hydrocarbon content, the more potent the substance is as a myocardial sensitiser. Within this spectrum, butanes (C4) are generally considered to be the most potent myocardial sensitiser; and (b) the higher the simple aromatic content, the more potent the substance is as a myocardial sensitiser.

Thus if CNS or narcotic effects are present, or if the product involved in the exposure is known to have a high C4-C5 and/or high simple aromatic content, monitoring of cardiac electrical activity is an important feature of clinical management. Handling stress may also increase the risks associated with catecholamine sensitisation. There is some evidence that catecholamine sensitisation may last for several days.

6. PERITONEAL DIALYSIS FOR THE TREATMENT OF POISONING IN DOGS AND CATS

The purpose of this commentary is to review the rationale for the use of peritoneal dialysis in poisoning of small animals. The contraindications and potential adverse effects of peritoneal dialysis are also reviewed.

Rationale for the use of peritoneal dialysis

The basis for the use of peritoneal dialysis is to accelerate removal of the absorbed poison from the body with the hope of reducing toxic effects. The expected end result is a reduction in total systemic exposure (i.e. area under the curve or AUC).

Technique

It is beyond the scope of this brief review to describe in detail the technique of peritoneal dialysis. The following references describe how to perform peritoneal dialysis in small animals:

Bersenas AME (2011) A clinical review of peritoneal dialysis. *Journal of Veterinary Emergency and Critical Care* 21(6) 2011, pp 605–617

http://vsecrounds.com/wordpress/wp-content/uploads/2012/12/JVECC-Peritoneal-Dialysis2.pdf

Cooper RL and Labato MA (2011). Peritoneal dialysis in veterinary medicine. *Veterinary Clinics of North America Small Animal Practice* 41: 91-113

In terms of technique, it is important to use purpose-formulated peritoneal dialysis fluids, purpose-made peritoneal dialysis catheters and to warm the fluid to body temperature. Purpose-made peritoneal dialysis fluid warmers are available at a reasonable cost.

Which poisons can be removed by peritoneal dialysis?

To be available for removal by dialysis, a poison must have the following properties:

- Relatively water-soluble (hydrophilic)
- Not highly protein-bound
- Low molecular weight
- Small apparent volume of distribution
- Slow intrinsic rate of clearance (i.e. a long half-life)

Peritoneal dialysis is generally much less efficient (1/8 to 1/3) than haemodialysis, and therefore it cannot be assumed that because haemodialysis has been shown to make a difference to clinical outcome in a given poisoning, that peritoneal dialysis will do likewise.

It should be borne in mind that improved clinical outcome is unlikely unless the AUC is at least halved. In other words, a small decrease in AUC is not a worthwhile goal of therapy.

Peritoneal dialysis has been shown to be useful in the following toxicoses:-

Barbiturates	Borates (e.g. ant-killer)	Carbamazepine
Ethanol	Ethylene Glycol	Iron
Lithium	Methanol	Salicylates
Salt	Theophylline	

It is important to note that in ethylene glycol poisoning, peritoneal dialysis must be used early, while most of the poison is still present as the parent compound rather than as the more toxic metabolites. It should also be noted that peritoneal dialysis will also remove ethanol if this has been administered as an antidote.

While peritoneal dialysis may be useful in poisoning due to aspirin or other salicylate preparations, it is not effective for paracetamol.

Contraindications and complications of peritoneal dialysis

Peritoneal dialysis should not be performed if the poison is unlikely to be effectively removed, and is contraindicated in patients that have recently had abdominal surgery. Complications of peritoneal dialysis include:-

- Fluid and electrolyte derangements
- Perforation or laceration of viscera
- Peritonitis (from dialysis fluid, procedure or visceral puncture)
- Adhesions
- Hypothermia (if dialysis fluid is not appropriate temperature)
- Dyspnoea (if fluid volume is excessive)

If peritonitis does occur, the recovered dialysis fluid may be cloudy. Note that many antibiotics are removed by dialysis and therefore if peritonitis develops, peritoneal dialysis must be stopped and antibiotic therapy instituted.

Some recommendations

- Treat the patient, not the poison.
- Determine whether the poison or toxic metabolite is likely to be effectively removed before embarking on peritoneal dialysis, bearing in mind that only a substantial decrease in AUC is associated with improved clinical outcome.
- Be aware of the possible adverse effects, and follow appropriate procedure to minimise the risk of these.

7. INDUCING EMESIS FOR THE TREATMENT OF ORAL POISONING IN DOGS AND CATS

The purpose of this commentary is to review the rationale for the use of an emetic (apomorphine, ipecac, hydrogen peroxide or other emetics) following oral poisoning and to present the current generally accepted case against their usage within this context. The contraindications and potential adverse effects of inducing emesis are also reviewed.

Rationale for the use of an emetic

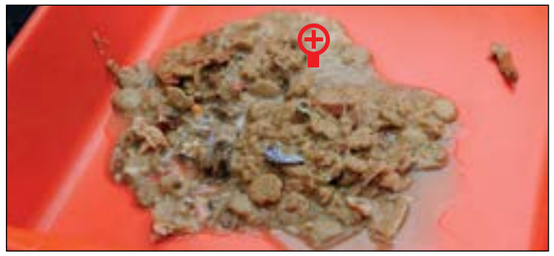
The usual basis for the use of an emetic is to reduce the gastric load of a toxicant with the hope of reducing absorption. The expected end result is a reduction in systemic exposure (i.e. area under the curve or AUC).

This rationale is based on three assumptions: (i) That inducing emesis is an effective way to remove gastric contents, (ii) that separation of the poison from the vehicle in the stomach is negligible and (iii) that absorption of the poison through the gastric mucosa is negligible. The first assumption has been clearly shown to be incorrect, the second is unlikely to be correct and the third is very often incorrect.

Common techniques

The most common emetic used in veterinary practice is apomorphine, followed by naloxone reversal. The medical profession abandoned the use of apomorphine in favour of ipecac syrup because of ipecac's superior safety profile, before almost wholly abandoning the use of emesis in poisoning since 1997.

Figures 3A-B. Apomorphine to induce vomiting and Methocarbamol muscle relaxant (courtesy of Anne Fawcett)



Figures 4 A-B. Vomit induced after chocolate poisoning (A) and contraceptive poisoning (B)

The case against the routine use of emesis for the treatment of oral poisoning

Inducing emesis in poisoning is a very ancient practice. Nicander of Colophon (185-135 BCE) employed linseed tea and a finger down the throat to induce emesis, while Galen (131-201 CE; physician to Marcus Aurelius) used chicken excrement as an emetic in mushroom toxicity. However, there are no peer-reviewed studies in the medical or veterinary literature that provide robust evidence that inducing emesis improves clinical outcome in a poisoning case. On the contrary, numerous well-designed and well-conducted studies in the medical literature have shown that inducing emesis makes no difference whatsoever to clinical severity of poisoning, length of hospital stay, clinical outcome or mortality. The clinical benefit, or lack thereof, of emesis in veterinary toxicology has not been explored. Based on extensive and robust medical evidence, however, the induction of emesis fails the requirement of Evidence-Based Medicine, which is that it improves clinical outcome. There is no evidence that it does, and it carries some disadvantages.

The absolute contraindications to inducing an emetic are well-recognized, and include:

- Ingestion of oils.
- Ingestion of hydrocarbons and other volatile substances.
- Ingestion of corrosive substances.
- Patient is exhibiting altered mental state (either excited or depressed mental status)
- Patient is at risk of seizures. Note that emesis itself can trigger seizures.
- Patient is at risk of stupor or coma
- Patient has or may have increased intracranial pressure, or other risk factor(s) of intracranial or intracerebral haemorrhage, such as thrombocytopaenia or abnormally low clotting factors.

It should not be overlooked by veterinary practitioners that oils **include waxes that melt at internal body temperature. Such waxes are routinely used in anticoagulant rodenticide baits.** The danger with oils is twofold. Firstly, if regurgitated material is aspirated, lipoid pneumonia is commonly severe and difficult to treat. Secondly, regurgitated oil may coat the larynx and enhance the ability of bacteria to enter the lower respiratory tract, resulting in opportunistic bacterial pneumonia.

Emesis is absolutely contraindicated if the animal has eaten a poison in bait formulated with wax.

Disadvantages of administering an emetic include the following:

- It is extremely unlikely to make any difference to clinical outcome, even if no clinical signs are present
- Emesis is likely to cause discomfort and distress to the patient
- Time spent administering an emetic, waiting for it to take effect, and administering naloxone, would generally be better spent observing the patient and, as appropriate, administering activated charcoal, antidote or other effective therapies
- There is clinical evidence that inducing emesis reduces the effectiveness of activated charcoal in human patients, by delaying its administration
- Risk of aspiration and secondary pneumonia. This may be less common in domestic pets than in human beings, but robust data are lacking
- Clinical signs of impending or current emetic effect, including hypersalivation and rigidity of abdominal muscles, may be confused with, or conceal, clinical signs caused by the toxicant
- Emesis will remove chloride from the body and, if abundant emesis occurs, may result in electrolyte imbalance
- Risk of CNS and respiratory depression from apomorphine. The dose of apomorphine that induces emesis also causes CNS and respiratory depression
- Rare but reported complications of inducing emesis include cerebral haemorrhage, oesophageal tear or rupture, hiatal hernia, gastric rupture, pneumothorax and pneumomediastinum

In addition, there is medicolegal risk to the practitioner who administers an emetic despite existence of a Material Safety Data Sheet (MSDS) warning against this practice. As MSDS documents are updated to reflect the fact that the modern medical profession almost universally regards induction of emesis as being of no value, it is increasingly common to find that the MSDS for common veterinary toxicants states that emesis should not be induced. As an example:

Baysol® Snail and Slug Bait MSDS

http://msds.duluxgroup.com/pdf/shess-en-cds-010-000000021405.pdf

This medicolegal risk has yet to be tested in a court of law.

In 1997 the American Academy of Clinical Toxicology and the European Association of Poisons Centers and Clinical Toxicologists issued a joint Position Statement on the use of ipecac syrup, the preferred emetic of the medical profession. The consensus statement represented a massive collaboration effort reflecting the opinions and experiences of clinicians, scientists and toxicologists from more than 60 countries. The consensus statement authors critically reviewed well-conducted clinical and experimental studies, and concluded that there was no evidence that ipecac improves the outcome in poisonings, and that its use in emergency departments should be abandoned. The 1997 Position Statement was reviewed and updated in 2004 and again 2013, with no change in the conclusions and recommendations.

The Position Statement reviewed the value of emesis in reducing absorption of marker substances in studies conducted in dogs and in human volunteers. The recovery of material was found to be highly variable but generally disappointing. Even when the emetic was administered within 30 minutes of ingestion, the mean recovery of material from dogs was 17.5% to 52.1%, and never exceeded 62%. Recovery of ingested material declined rapidly with time as material was absorbed and/or moved beyond the stomach. Significantly, the poor response to emesis was found in dogs even when barium sulphate suspension, which is not absorbed from the gastrointestinal tract, was used as a marker. The mean recovery of barium sulphate was only 62% when the emetic was administered immediately after the barium sulphate was administered, declined to 44% at 30 minutes and to 31% at 60 minutes. Emesis shortly following administration of barium sulphate in gelatin capsules to fasted puppies resulted in a recovery of only 2% to 31% of the marker. This is consistent with endoscopy studies that have shown that tablets can still be found in the stomach after administering an emetic, and the experience of one of the authors (RRD) that poison bait can still be found in the stomach of an animal that has died after vomiting copiously. The Position Statement also cited cases in which emesis failed to significantly alter the number of ferrous sulphate tablets seen on radiography of the stomach prior to administration of an emetic. The Position Statement concedes that emesis with ipecac 'occasionally produces impressive returns' but does not comment on whether that translates into improved clinical outcome.

One of the authors of this article (RBC) has published a paper in the peer-reviewed veterinary literature that clearly shows that poisoned animals are very rarely presented at a veterinary clinic within 30 minutes of ingestion of a poison. It should also be borne in mind that animals rarely confine themselves to eating less than 2 or 3 times the dose of a poison required to exceed the toxic threshold, so recovery of 40 to 60% of the gastric contents is unlikely to bring the total systemic exposure to below the toxic threshold in most clinical situations.

The Position Statement also reviewed toxicokinetic studies. These studies show that administration of an emetic within 30 minutes of ingestion may reduce the systemic exposure (AUC), but the decreases in AUC are generally modest. This is likely to be a reason why inducing emesis has never been shown to translate into an improved clinical outcome.

One study reviewed in the Position Statement suggested that ipecac may increase the movement of stomach contents through the pylorus and into the duodenum, although whether apomorphine would have the same effect is undetermined.

The Position Statement also reviewed a number of randomised prospective studies conducted in emergency rooms. These studies included hundreds of patients and therefore have great statistical power. No clinical benefit from inducing emesis was found. All other aspects of patient profile, poison exposure and treatment being equivalent, inducing emesis made no difference to outcome.

The Position Statement stopped short of stating that ipecac should not be administered as a first aid measure by the caregiver of an alert, asymptomatic child prior to calling for medical or paramedical assistance. Subsequent studies have shown that home use of ipecac in children did not reduce emergency department use or improve outcome in poisoning. In one study it was found that referrals from a Poison Center to an Emergency Department (ED) transiently increased, with no corresponding increase in overall morbidity or mortality, but the referrals then declined to baseline. Investigation showed that poison center personnel were initially reluctant to accept that it would not alter the outcome if emesis was not induced. As poison center personnel became comfortable with the fact that emesis made no difference to clinical outcome, referrals to an ED dropped back to baseline.

Overall in the EU and USA, there has been no adverse effect in human clinical populations of omitting emesis from the treatment of oral poisoning. Morbidity and mortality data have remained the same, validating the conclusions of the 1997 Position Statement.

There is no scientific evidence that inducing emesis in oral poisoning improves clinical outcome in either human beings or other animals.

In the view of the authors, the only situation in which we can see a possible, although unproven, benefit to inducing emesis is if a patient is presented to a veterinarian very shortly after ingesting a highly toxic substance in a delayed-release form. An example from New Zealand would be if a dog had eaten a sea slug, *Pleurobranchaea maculate*. Sea slugs contain the bacterial toxin tetrodotoxin, a very deadly bacterial toxin also found in puffer fish, blue-ringed octopi, and a number of other species. The body of the sea slug would act as a delayed-release formulation and if the sea slug could be recovered in vomitus in intact or near-intact form, this would be preferable to an emergency gastrotomy. Sea slugs would usually be too large to be recovered by gastric lavage.

Figure 5. Suspected puffer fish ingested then vomited by dog (courtesy of Anne Fawcett)

What about home emetics for first-aid use by clients?

The two emetics most commonly suggested for home use on pets by clients, and most likely to be available, are 3% hydrogen peroxide and table salt, either straight or in saline solution.

A factor to consider with regard to any use of an emetic by an owner is whether the owner will then delay bringing the animal to the veterinary clinic until after the animal has vomited. This delay in treatment could be detrimental to the outcome.

Hydrogen peroxide at a 3% concentration appears to be effective and fairly innocuous. It is not as commonly found in Australasian homes as it is in the USA where it is widely used as a disinfectant in first-aid. Hydrogen peroxide solutions break down over time to oxygen and water. The greatest risk with hydrogen peroxide is if the client inadvertently prepares it at too strong a concentration. Ingestion of concentrations over 10% can cause severe burns to the mouth, oesophagus and stomach, and fatalities have been recorded in the medical literature. In addition to this mechanism, hydrogen peroxide can cause gas emboli. The breakdown of 1.0 mL of 35% hydrogen peroxide can produce 115 mL of oxygen gas. In a canine study, investigators demonstrated that colonic lavage with only 0.75% hydrogen peroxide caused gas bubbles to form in the mesenteric circulation. Whether the same outcome could occur with oral intake, or whether the gas would be successfully eructated, has not been investigated. There has been a ▶

human case of acute cerebral gas embolism after ingestion of a small amount of 35% hydrogen peroxide. The veterinarian's level of comfort with suggesting that hydrogen peroxide should be used as a home emetic for pets is likely to be based on their assessment of the ability of the owner to ensure the correct dilution is used.

It is of particular concern to the authors that many websites, including some written and maintained by veterinarians or by state or federal governments, recommend the home use of salt or saline solution to induce emesis. It is also alarming that many rodenticide MSDSs advise that pets should be given a saline solution, mixed in 'a soft drink bottle' and 'squirt[ed] down the throat' as an emetic. There are numerous documented cases of fatal hypernatraemia in adult humans, children and domestic pets as a result of this practice. Even ingestion of homemade modeling dough or of seawater has resulted in lethal hypernatraemia in dogs. Hypernatraemia has a high mortality rate even with aggressive treatment.

Salt, in solution or as a solid, should never be used as an emetic in domestic pets, or in human beings, under any circumstances.

The reaction from the New Zealand veterinary profession

Our attempts to bring the New Zealand veterinary profession up-to-date with the science on the induction of emesis in small animal poisoning have been met with great reluctance on the part of the profession to abandon the use of emesis in light of scientific evidence. It may be instructive to review their objections and our responses, to clarify the science around this issue.

I need to see the scientific evidence before I change the way I practice.

Although this sentiment is generally commendable, in this situation, it is important to understand that the practice of inducing emesis was never based on science. The number of peer-reviewed scientific papers showing that inducing emesis makes a difference to clinical outcome is zero. So what this person is unknowingly saying is 'I need to see the scientific evidence before I abandon an unscientific practice'.

The 1997 Position Statement is in the public domain and readily available online, as are the subsequent reviews and a number of other scientific articles showing that inducing emesis does not improve clinical outcome in oral poisoning. Some further reading is included at the end of this article.

It makes intuitive sense that inducing emesis will help.

At risk of being facetious, it also makes intuitive sense that the sun orbits the Earth, and that incorrect intuitive conclusion misled the human race for thousands of years. In other words, beliefs that make intuitive sense are not always correct.

The 'intuitive' conclusion in this case is based on the assumption that inducing emesis is an effective means of emptying the stomach and/or lowering the AUC enough to make a clinical difference. As discussed above, these assumptions have been shown to be incorrect.

I've induced emesis in a great many poisoning cases and I know the patients would have done worse without it.

When asked how they 'knew' this, the practitioner admitted that they didn't have any proof, but the patients recovered and they supposed that the emesis, among the other therapeutic measures, contributed to the recovery.

Human beings are so prone to this sort of fallacious reasoning that it even has its own Latin name, *post hoc ergo propter hoc*, which translates as 'After this, therefore because of this.' *Post hoc ergo propter hoc* reasoning has generally served the human race quite well, because it often happens to be true. However the use of *post hoc ergo propter hoc* reasoning can also lead to false conclusions. If an animal recovers from poisoning and the therapeutic regimen included apomorphine, it doesn't mean the animal would not have recovered just as well if the therapeutic regimen had not included apomorphine.

I know inducing emesis helps if the animal has eaten metaldehyde or anticoagulant rodenticide because I've recovered a lot of bait with the green (or blue) colour still in it.

There are several false assumptions here:

(i) That the green or blue colour is the poison. In fact the green or blue colour is an inert dye. Both metaldehyde and anticoagulant rodenticides are colourless.

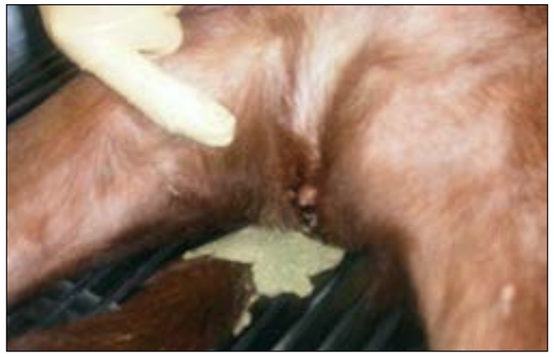
(ii) That the inert dye has the same absorption characteristics as the poison. This is an unfounded, and incorrect, assumption. By the same token, fluorescein in the vomitus does not signify that emesis has succeeded in recovering ethylene glycol.

(iii) That the stomach is an inert bag in which nothing happens to separate the poison from the bait, so recovery of a lot of bait implies recovery of a lot of poison. On the contrary, the stomach is anything but inert in digestive and absorptive processes. It is worth remembering that when a chemist wants to separate the different substances in a biological mixture, the first step is usually an acid digestion, because it is an extremely good way to separate different biological molecules. The stomach, which also features agitation and warmth to enhance acid digestion, is highly conserved throughout much of the animal kingdom, probably because it works so well at separating substances. It cannot be assumed that because the vomitus contains vehicles, fillers and dyes from the original bait, that it also contains the poisonous substance at the same concentration as the original bait. Unless emesis is induced almost immediately after ingestion, it is far more likely that it does not.

I know the dog's stomach was emptied by the apomorphine because it could not bring up any more contents and was just producing clear 'spits' of fluid.

It is not safe to assume that because an animal is no longer able to produce stomach contents other than fluid, that the stomach has been emptied. One of the authors (RRD) has performed necropsies, and reviewed necropsies performed by others, in which poisonous material was still present in the stomach even though the animal had, while alive, vomited repeatedly and had reached the point that it had not been able to regurgitate anything but fluid.

I gave apomorphine to a puppy that had just eaten some metaldehyde-containing slug baits, and it vomited up some baits and then did not develop clinical toxicosis, so that proves the benefit of inducing emesis.



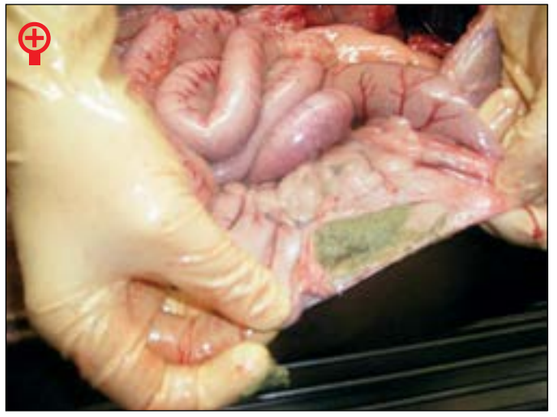


Figure 5 A-C. Case of Defender™ Snail & Slug Pellets poisoning (courtesy of Aine Seavers)

This is *post hoc ergo propter hoc* reasoning again, as discussed above. In this particular case, because the toxic threshold of metaldehyde in dogs is available in the literature, it was possible to show that the amount of bait consumed by the puppy, on a mg/kg bodyweight basis, would be very unlikely to exceed the toxic threshold. Therefore it was at least as likely that the puppy did not develop clinical signs because it did not eat a dose sufficient to exceed the toxic threshold.

Dr Safdar Khan and colleagues at the ASPCA Animal Poison Control Center in the USA have published a paper titled 'Effectiveness and adverse effects of the use of apomorphine and 3% hydrogen peroxide solution to induce emesis in dogs' in JAVMA *in 2012. They are experts and they say apomorphine is effective, so I'm going to keep using it.*

Both the authors of this article are very familiar with the paper of Khan *et al* 2012. The paper defines 'effectiveness' as whether or not the emetic was successful in inducing emesis, not whether emesis has any beneficial effect in poisoning. There are no studies that show that emesis **alone** has any beneficial effect on clinical outcome in dogs or cats. The only veterinary paper that the ASPCA Animal Poison Control Center cites to defend the practice of inducing emesis shows that emesis *together with activated charcoal* can improve outcome in anticoagulant rodenticide poisoning. The very real possibility that the beneficial effect is entirely attributable to the activated charcoal has not been explored. Studies in human beings have shown that activated charcoal alone is more effective than activated charcoal after emesis.

Brodifacoum rodenticides are formulated so that gastric emptying is delayed, so we should still use emesis for those.

This argument again assumes that the stomach is merely a bag that holds ingested material until it progresses further, which is far from the case, as discussed above. The acid stomach is extremely well adapted to separate biological substances from each other. In addition, brodifacoum is a lipophilic molecule even when ionized, as it would be in the stomach, and therefore it could be absorbed across the gastric mucosa before an emetic could be administered. It should further be borne in mind that brodifacoum is commonly formulated with waxes that melt to oils inside the body, and emesis is therefore contraindicated because of the risks associated with emesis of oils.

The C_{max} (maximum plasma concentration) of brodifacoum is not reached until 11 hours, so we have plenty of time to get it out of the gastrointestinal tract by emesis.

This assumes that the brodifacoum is sitting patiently in the stomach for the best part of 11 hours before suddenly proceeding through the pylorus and being absorbed, hardly a likely event even given the formulation to delay gastric emptying. In addition, as discussed in the previous response, brodifacoum is overall a lipophilic molecule with few sites for ionization, so even when ionized it is likely to readily cross biological membranes. So why is there a relatively late C_{max}? Where is the brodifacoum if not distributed evenly in the hydrophilic (including plasma) parts of the body? While pharmacokineticists and toxicokineticists generally use plasma concentration as a default way to determine the total concentration in the body, it is recognized to be inaccurate in the case of lipophilic substances. Calculating the apparent volume of distribution (V_d) of a lipophilic substance from the plasma concentration typically results in a V_d far greater than the total volume of the body itself, because most of the lipophilic substance is in the lipid stores such as adipose tissue, Ito cells of the liver and, for those substances that can cross the blood-brain barrier, the central nervous system. In fact, mice that have eaten toxic doses of brodifacoum have been shown to have 20 times the anticoagulant concentration in their liver than that in their plasma. Lipophilic substances such as brodifacoum are unlikely to reach C_{max} in the plasma until the capacity of lipid stores has been saturated.

Anything that reduces the total body burden of a poison must be doing some good.

Experimental and clinical studies have shown that this is not the case, and that for those substances that have been studied clinical outcome is not improved unless the AUC is decreased by more than half. Inducing emesis is unlikely to achieve this. Even if emesis is induced immediately after ingestion of a poison, the recovery is unlikely to be more than 50%. Even assuming 100% absorption of the remaining material, this would only be clinically beneficial if the animal had eaten exactly the lethal dose and no more. Animals are unlikely to show such restraint.

The studies on which the Position Statement was based only assessed mortality of patients. I believe inducing emesis can reduce morbidity and shorten treatment time.

This is incorrect. A range of endpoints including severity of toxicosis and length of hospital stay were assessed in addition to mortality. Inducing emesis has never been shown to reduce morbidity or shorten treatment time. On the contrary, when administration of activated charcoal is delayed in order to induce emesis, clinical severity and length of treatment time have been shown to be increased.

The studies on which the Position Statement was based were human studies and don't apply to animals.

On the contrary, the literature review on which the Position Statement is based includes a number of experimental studies in dogs. Furthermore, from the point of view of pathophysiology, there is no reason why extrapolation cannot be made from humans to animals. Toxicologists are very aware that extrapolation is routinely done from non-human animals to human beings, and many medical advances have been adopted into veterinary medicine. ▶

The Position Statement is based on the use of ipecac and does not apply to apomorphine.

The medical profession abandoned the use of apomorphine in favour of ipecac because they found ipecac to be safer, the reversibility of apomorphine notwithstanding. Because ipecac cannot be reversed, it may lead to greater delay in administering activated charcoal than does apomorphine, but that is the only difference. Otherwise, the information on which the Position Statement is based applies as much to apomorphine as it does to any other emetic.

Most human poisonings are pharmaceutical drugs and veterinarians treat a wider range of poisonings. Therefore you can't extrapolate from medical publications.

This is incorrect, as perusal of any medical toxicology textbook will show. Emergency room doctors deal with a much wider range of poisonings than veterinarians do. Small children tend to get into all the same substances that puppies do, including insecticides, molluscicides, antifreeze, household cleaners and poisonous plants. In addition, human poisonings encompass pharmaceutical drugs, industrial poisons, and recreational drugs among others.

We must be seen to be doing all we can to help the animal.

With respect, the authors of this article question the ethics of administering a treatment that has no scientific evidence to support it, and which causes the animal to suffer nausea, just to impress the client. We also question the business ethics of charging the client for ineffective treatment. Would it not be better to explain to the client that inducing emesis is not helpful, as the medical profession has done?

I still believe inducing emesis has a place in treating poisoned animals.

While we acknowledge that there are specific, rare situations in which we would consider inducing emesis justified, such as recent ingestion of a sea slug by a dog, we encourage all practitioners to practice Evidence-Based Medicine, to recognize that there is no evidence that inducing emesis is beneficial in the vast majority of cases of small animal poisoning, and to recognize that inducing emesis carries significant risks and contraindications.

Some recommendations

- Treat the patient, not the poison.
- In general, inducing emesis has no place in the routine management of oral poisonings, and it may reduce the effectiveness of activated charcoal and delay the administration of beneficial therapeutic measures.
- Salt or saline solution should NEVER be used as an emetic

Further reading

In the public domain

American Academy of Clinical Toxicology/European Association of Poisons Centers and Clinical Toxicologists joint Position Statement (2004 version) http://www.clintox.org/positionstatements.cfm

Gussow, L (2004). The last thing you'll ever have to read about ipecac. http://journals.lww.com/em-news/Fulltext/2004/01000/The_Last_Thing_You_ll_Ever_Have_to_Read_about.11.aspx

Höjer J, Troutman WG, Hoppu K, Erdman A, Benson BE, Garbane BMÉ, Thanacoody R, Bedry R and Caravati EM (2013) Position paper update: ipecac syrup for gastrointestinal decontamination. http://www.eapcct.org/publicfile.php?folder=joint&file=PS2013_IpecacSyrup.pdf

Roberts, JR (2002) Basic toxicology: Ipecac-induced emesis: Syrup of ipecac is a dinosaur that lacks efficacy in altering clinical outcome for toxic ingestion and carries serious potential for disaster: Part I in a series. http://journals.lww.com/em-news/Fulltext/2002/06000/Basic_Toxicology__Ipecac_Induced_Emesis__Syrup_of.11.aspx

Available by interloan

Albertson TE, Derlet RW, Foulke GE, Minguillon MC and Tharratt SR (1989) Superiority of activated charcoal alone compared with ipecac and activated charcoal in the treatment of acute toxic ingestions. *Annals of Emergency Medicine* **18**: 56-59

Barer J, Hill LL, Hill RM and Martinez WM (1973) Fatal poisoning from salt used as an emetic. *American Journal of Diseases of Children* 125 889-890

Bond GR (2003) Home syrup of ipecac use does not reduce emergency department use or improve outcome. *Pediatrics* **112**: 1061-1064

Calam J, Krasner N and Haqqani M (1982). Extensive gastrointestinal damage following a saline emetic. *Digestive Diseases and Sciences* **72**: 936-940

Casavant MJ and Fitch JA (2003) Fatal hypernatremia from saltwater used as an emetic. *Journal of Toxicology Clinical Toxicology* **41**: 861-863

Kasai CM and King R (2009). Hypernatremia. Compendium: *Continuing Education for Veterinarians* April 2009

Kornberg AE and Dolgin J (1991). Pediatric ingestions: charcoal alone versus ipecac and charcoal. *Annals of Emergency Medicine* **20**: 648-51

Kulig K, Bar-Or D, Canrtill SV, rosen P and Rumack BH (1985). Management of acutely poisoned patients without gastric emptying. *Annals of Emergency Medicine* **14**: 562-567

Lapus RM, Slattery AP and King WD (2010). Effects on a Poison Center's (PC) triage and follow-up after implementing the no ipecac use policy. *Journal of Medical Toxicology* **6**: 122-125

Moder KG and Hurley DL (1990) Fatal hypernatremia from exogenous salt intake: report of a case and review of the literature. *Mayo Clinics Proceedings* **66**: 439

Pond SM, Lewis-Driver DJ, Williams GM, Green AC and Stevenson NW (1995) Gatric emptying in acute overdose: a prospective randomized controlled trial *Medical Journal of Australia* **163**: 345-349

Türk EE, Schulz F, Koops E, Gehl A and Tsokos M (2005). Fatal hypernatremia after using salt as an emetic – report of three autopsy cases. *Legal Medicine* **7**: 47-50

8. WHOLE BOWEL IRRIGATION FOR THE TREATMENT OF POISONING IN DOGS AND CATS

The rationale for whole bowel irrigation (WBI) is that the induction and passing of a liquid stool using an osmotically balanced polyethylene glycol-electrolyte solution (PEG-ES) will help to reduce the absorption (and thus the systemic exposure and area under the curve; AUC) of ingested extended-release substances or substances with an a very slow rate of absorption. Materials other than PEG-ES (typically GoLytely®) have been used for this purpose (e.g. sodium picosulfate, other proprietary materials used for bowel cleansing prior to colonoscopy). However, currently the effectiveness and safety properties of these alternative materials have not been assessed. Provided correctly balanced materials are used, and despite popular misconceptions, WBI does not cause significant serum electrolyte abnormalities. This cannot necessarily be said for materials not specifically designed for WBI. Furthermore, this is one of the significant differences between WBI and traditional methods of catharsis/purgation.

Like many techniques for decontamination of the bowel during a toxicological emergency, WBI has been applied ill advisedly as a panacea. While WBI may be of value under specific defined circumstances, it should not be used routinely in cases of oral poisoning and it is not a first-line treatment modality in the majority of cases. There remain very large data gaps regarding the risk:benefit ratio and potential indications for the technique. It is particularly important to bear in mind the contraindications/risk factors for the technique.

Indications For Use and Effectiveness

As with catharsis, WBI has only been demonstrated to be effective and to have a reasonable risk:benefit relationship for materials that are: (a) slowly released or are/have extended release formulations; and (b) ingested in significant or life-threatening amounts; and (c) do not bind effectively to activated charcoal or the patient presents too late for activated charcoal to be effective (i.e. exposure to treatment intervals of greater than 60 to 120 minutes); (f) the animal presents for treatment while the material is still present within the small intestine (and possibly the large intestine for a very small minority of xenobiotics that are absorbed from this area of the gut – a rare situation); and (e) contraindicating factors are absent and the treatment is likely to have a good risk:benefit ratio. In order to make a clinically significant difference, administering WBI should have a reasonable possibility of reducing the xenobiotic AUC by ≥ 40-50% (approximately).

WBI has been demonstrated to have clinical benefit with the following xenobiotics: (a) orally ingested whole fentanyl transdermal patches; (b) orally ingested whole clonidine transdermal patches; (c) orally-ingested slow/extended release potassium chloride preparations; (d) possibly other orally-ingested sustained/extended release pharmaceuticals combined with late (> 60-120 minutes exposure to treatment interval) presentation for treatment; (e) orally ingested lithium pharmaceutical preparations; (f) possibly orally ingested iron supplements; and (g) drug packing of illicit narcotics/drugs of addiction (the author has encountered dogs that have been used for this purpose, although the author is yet to encounter a cat being used for such a purpose). Although unproven, there is some possibility that WBI may offer some benefit for plant poisonings that are associated with a relatively slow and sustained release of the toxin(s) from the plant material and the toxins are known not to bind to activated charcoal. However, there is a gross shortage of objective, well-controlled data. This lack of data makes it difficult to specific recommendations regarding which plant intoxications might benefit from treatment with WBI.

WBI has also been suggested in cases of life-threatening intoxications where the agent does not bind to activated charcoal and where no other method of gastrointestinal decontamination is possible. Again, there is a gross shortage of objective, well-controlled data that demonstrates either effectiveness or risk under such circumstances. Again, this lack of data makes it difficult to specify recommendations regarding which situations might benefit from treatment with WBI.

It is also critical to note that xenobiotic plasma TMax (i.e. time to maximum plasma concentration) is not a direct indicator of the effective post-exposure time window for WBI. A number of other factors come into play.

The Australian Poisons 'bible' by C&T Series contributor, Ross McKenzie, published by CSIRO, is available to CVE financial members at 15% discount. Go to: www.cve.edu.au/node/25910

Contraindications and Complications

WBI is generally inappropriate in the following circumstances:

- If the airway is unprotected or compromised i.e. if the gag reflexes are compromised. **It is important to note that it is often not feasible to perform WBI with a cuffed endotracheal tube in place;**
- If there is cardiovascular instability;
- If there is respiratory instability;
- If there is any evidence of bowel obstruction, significantly reduced bowel motility or ileus or bowel obstruction;
- If there is evidence or likelihood of gastrointestinal hemorrhage;
- If intractable vomiting is present. **Notably, vomiting is more likely if emesis has been previously induced or if the ingested material triggers vomiting;**
- In cases of drug packing where there is evidence of leakage of the drug packets (e.g. evidence of sympathomimetic overdoes, hypertension, hyperthermia, tachycardia, serotonin syndrome and so forth with cocaine). Stabilization (if possible) followed by surgical intervention is the preferred option under these circumstances.

Given that circulatory disturbance/hemodynamic instability (or exacerbation of these conditions) is a rare, but potentially serious, consequence of WBI, it is **particularly important to ensure that cardiovascular resuscitation and stabilization are complete and effective before beginning WBI. It may be reasonable and prudent to have already established an intravenous access prior to commencing WBI treatment.**

WBI is not without risk, although if administered correctly it appears to have reasonable safety properties (based on the limited available data). Most complications arise as a consequence of errors/injuries associated with nasogastric tube placement. WBI may result in nausea, vomiting, abdominal bloating and abdominal discomfort/cramping.

How To Do It

The equipment needed is as follows:

- 10% xylocaine spray or ophthalmic examination topical anesthetic (e.g. propariacaine 0.5%);
- 2% xylocaine jelly or other water-based lubricant;
- A 3.5 – 5 Fr. rubber, polyurethane or silicone nasogastric tube (avoid the polypropylene ones as they tend to kink). The larger the tube, the better given the volumes, flow rates and viscosity of the material. The author prefers brands of nasogastric tubes that incorporate a radiographic marker. Under most circumstances, the volumes involved are relatively large, thus oral dosing with a syringe may not be practical.
- Easily washable facilities that can cope with the volume of expelled bowel contents.
- At least 2-3 liters of GoLytely® (depending on the size of the animal). Use other colonoscopy preparations or sodium picosulfate preparations **at your own risk**!

Remember that WBI always comes after resuscitation/stabilization (airway, breathing and circulation in that order) and administration of known effective antidotes. Treat the patient, not the poison.

Different practitioners have different techniques and tricks for passing a nasogastric tube. Provided the tube ends up in the stomach and traumatic nasogastric intubation is avoided, the method is appropriate. The author's personal preference is to place a slight temporary bend in the tip of the nasogastric tube and chill/freeze the tube before use to keep the bend in place for a short period of time. Some practitioners will tie a loose knot in the end of the tube in order to produce the temporary bend (obviously *you need to untie the knot before you try to place the tube*, which brings up the situation that the author has observed where a practitioner could not untie the knot in the tube!). While this is happening, a couple of drops of ophthalmic examination topical local anesthetic are placed into the nostril. It is important not to use too much local anesthetic as the material may run back into the pharynx and anesthetize the arytenoid cartilages, disrupting the gag reflexes. The tube is placed with the slightly curved tip facing ventrally though the ventral nasal meatus with the aid of liberal amounts of lubricant. Once into the nasopharynx, the tube is rotated so that the curved tip points dorsally. This seems to aid passage of the tube into the esophagous. The author's personal preference is to then perform a thoracic radiograph to ensure that the tube is present in the esophagus/stomach and not in the respiratory system. An alternative method to check placement is to have an assistant rapidly inject about 50-60 mL of air into the tube while the practitioner auscultates over the stomach (hopefully a 'rush' of air will be heard). The pH of stomach suction aspirates can also be checked using litmus paper or pH indicator papers. Given the volumes involved, administering WBI fluids into the respiratory system is a catastrophic mistake. Any gastric aspiration samples should be collected *before* starting WBI.

Unfortunately there are no systematic dose response studies for WBI. Various dose regimens have been suggested for humans, however there are has been no systematic evaluation of these regimens (e.g. 500 mL/hour for children 9 months to 6 years of age, 1000 mL/hour for children 6-12 years of age; 1500 – 2000 mL/h for over age 12 years; alternatively 240 mL every 10 minutes until the stool is clear and contains no solid material). The author's preference is to start at a slow rate of ▶

administration and then carefully observe the animal for evidence of abdominal discomfort/pain, abdominal bloating or hemodynamic disturbance. If no adverse effects are present, the administration rate is slowly increased until the maximum practical rate of administration possible with the size of nasogastric tube or 400-500 mL/hour, whichever is the lower. Warming the irrigation fluid will reduce its viscosity and help the administration with the relatively narrow nasogastric tubes used in veterinary medicine. With cats and smaller dogs, it is important to reduce the maximal rate of administration and to increment the administration rate very slowly combined with careful observation of the animal. Administration of the irrigation fluid is continued until the stools are clear fluid with no solids. Usually clearance of the bowel will commence within 30-60 minutes following the start of the procedure and the whole procedure may take up to several hours, particularly with large dogs. **It is important to regularly monitor the circulatory status and the hydration status of the animal that is undergoing WBI. It is also critical to monitor for evidence of gastrointestinal hemorrhage. Orally administered medications given during WBI are likely to be less effective or ineffective because of reduced absorption.**

It is also absolutely critical to stop the administration of the irrigation fluid if no defecation has occurred within a reasonable length of time (e.g. within 1 hour or so) or if vomiting has started (especially if it is protracted) or if there is evidence of hemodynamic instability, or if there is evidence of undue abdominal discomfort/abdominal distress and/or pain and/or if there is no evidence of gut sounds on auscultation. If no defecation has occurred and/or there is no evidence of movement of gut contents and/or there is undue pain or other adverse events occurring, it is absolutely critical to re-evaluate the patient for the presence of a bowel obstruction (including abdominal radiographs). It is possible that there was no bowel obstruction at the start of the WBI procedure. A bowel obstruction can occur during the procedure (albeit rarely), particularly if there are large solid masses, foreign bodies or bezoars within the gastrointestinal tract or if there is a propensity for intussusception. Some practitioners deliberately add radiopaque marker beads to the irrigation fluid at regular intervals in order to monitor the progress of the procedure. However, this is not a critical aspect of the procedure and there is some evidence that the position of such markers does not directly correlate with the actual status of the procedure.

Figure 6. Pets access poisons with alarming ease (courtesy of Anne Fawcett)

Figure 7. New Members are directed to the CVe-library to read our June 2011 issue, devoted entirely to Small Animal Poisoning. 'Bosca' pictured with the issue (courtesy of Anne Fawcett)

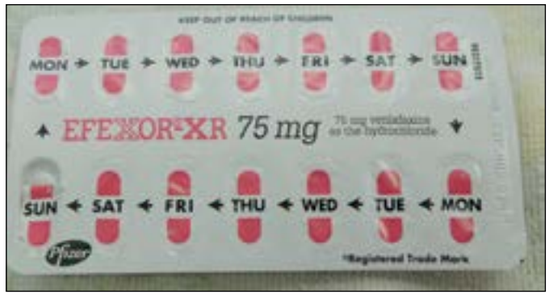


Figure 8. Efexor poison in a cat (courtesy of Anne Fawcett) – watch out for an upcoming article on this topic by Anne

Live Feline Vaccines

The best protection from Feligen®

Proven lack of shedding*

Strong cell-mediated & humoral immune response^

Unparalleled safety with no adjuvants, preservatives or mercury

BLDF906

Don't risk inferior protection.

Discuss the strengths of live feline vaccines with your Virbac Area Manager or call 1800 009 847.

* Virbac data on file, APVMA 40759

^ Greene CE, Schultz RD. Immunoprophylaxis and immunotherapy. In: Greene, CE, ed Infectious diseases of the dog and cat. Philadelphia: WB Saunders Co, 2006; 1069–1119.

Virbac

Passionate about animal health