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'Fifi', gracing this cover, won the most 'likes' in our Facebook Best 'Vet Pic' competition

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CONTENTS

MERRY XMAS, SEASON'S GREETINGS AND HAPPY NEW YEAR

from the CVE team and our generous sponsors

Producing and delivering the *Control & Therapy Series* in high quality print and digital format is expensive. We acknowledge and thank our long-term sponsors – Boehringer Ingelheim, Virbac Animal Health and Hill's Pet Nutrition – for their ongoing financial support.
We also thank ASFM, BOVA Compounding, Provet, Radincon, Vepalabs and VetApps for financing the extra 16 pages in this `bumper' Xmas issue.

Note: The CVE office will close in line with The University of Sydney's policy. Our last day will be Thursday 18 December and we re-open on Monday 5 January 2015.

FROM THE DIRECTOR

Another year is drawing to a close, and for the CVE it has been a big year as our new team members have come to grips with the idiosyncrasies of the veterinary profession. You will have noticed that our marketing materials have a fresh new look, which has had a positive impact on the promotion of our new membership categories and the appearance of everything we produce. At the end of last year, the CVE was restructured into three business units, which all interact with each other in many different ways. There is a marketing team, a business services team and an education team and everyone in each of these teams has been working hard to ensure that the CVE is offering and delivering what our members want.

Next year is going to be an even busier year with many events and workshops spread around the country. At the end of January, a cattle workshop will be held at Charles Sturt University. This workshop is similar to one run three years ago at Camden and is designed to be practical and invaluable to anyone who wants to explore or expand on a veterinary career with cattle. There will be 3½ days of combined practical sessions and interactive didactic sessions, starting early and finishing late, being led by experts in the field. Numbers will be limited to ensure that everyone who attends gets maximum benefit from this workshop.

In February, our traditional major Sydney conference will be held with four days of companion animal medicine with a sprinkling of surgery. On the fifth day a masterclass will be held in feline medicine, with limited numbers. This program will be presented by some of Australia's finest veterinary specialists and was convened by Andrea Harvey, who has written several articles for C&T. It's a dynamic course populated with a wide range of 'new' veterinary specialists.

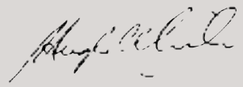
A two day pharmacology seminar is to be held in April at Rydges World Square in Sydney and will be presented by the CVE in collaboration with the pharmacology chapter of the ANZCVS. The program will cover a number of topics, which will provide the latest information on pharmacology and therapeutics and provide an important update for anyone in practice.

The midyear conference to be held in Melbourne has been convened by Aussie expats Geraldine Hunt and Bryden Stanley, who will ably be assisted by Arthur House. This conference will change the way you approach soft tissue surgery, as these three specialists will impart their combined knowledge gathered through years of experience in Australia, the UK and North America. The fifth day will be a wet workshop on practical reconstructive surgery.

In September, our holiday destination conference will be held over four days at Port Douglas. The topic will be dermatology with many of the speakers familiar to those of you who attended our very popular dermatology conference in Sydney in 2009. Dermatology is such large part of most companion animal practices and it is an ever changing and advancing field, so this conference will set the standard for best practice dermatology for the next five years.

As usual, throughout the year we will be running a number of shorter seminars and stand-alone workshops, so visit our website or watch out for our e-mails to make sure that no one in your team misses out on a CVE event in 2015.

Once again, we have produced a bumper issue of C&T for the final edition of 2014, so please take your time to enjoy the wide variety of articles contributed by our passionate and sometimes contentious contributors. Enjoy the holiday season and take the opportunity to recharge your batteries ready for an even bigger and better year.



2015 CALENDAR
CVE CPD PROGRAM

CONFERENCES & SEMINARS

16-20 Feb	Small Animal Internal Medicine Conference – with a dash of Surgery AND 1 day Feline Masterclass	Sydney
27 Feb	Zoobiquity Conference	Sydney
15 Mar	Gastrointestinal Surgery - Tips & Tricks	Launceston
16-17 Apr	Pharmacology Symposium	Sydney
3 May	Canine Internal Medicine	Adelaide
16-17 May	Cardio-respiratory Medicine & Diseases of the Chest	Brisbane
31 May	Gastrointestinal Surgery - Tips & Tricks	Canberra
15-19 Jun	Soft Tissue Surgery including 1 day workshop	Melbourne
25-26 Jul	Ophthalmology: Theory and Practice including workshop	Sydney
14-17 Sep	Updates in Clinical Dermatology	Port Douglas

WORKSHOPS

28-31 Jan	Beef and Dairy Cattle Workshop	Wagga Wagga
28 Feb	Hip & Stifle Workshop	Sydney
1 Mar	Bone Plating Workshop	Sydney
7 Mar	Basic Echocardiography Workshop	Sydney
8 Mar	Advanced Echocardiography Workshop	Sydney
14 or 15 May	Approaches to Bones and Joints	Sydney
18/19 Jul	Basic Echocardiography Workshop	Brisbane

TIMEONLINE

9 Mar	Respiratory Physiology, Monitoring and Support
6 Apr	Rabbits and Rodents
18 May	Small Animal Oncology
8 Jun	Practical & Advanced Dentistry

Visit **www.cve.edu.au** for the most up-to-date CVE CPD calendar.

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CONGRATULATIONS TO OUR DE EARLY BIRD WINNERS FOR 2015

Paying early ensured the following vets not only secured a place in the DE course of their choice for 2015, they also received a hefty discount and were the 3 lucky winners in our Early Bird draw, winning an iPad mini each.

Congratulations to **Ratanaporn Tangwangvivat,** Thailand – 2015 Dermatology,
Deborah Hope, UK – 2015 Feline Medicine and
Joanne Douglas, UK – 2015 Feline Medicine

and to all our DE Participants who secured a place in our 2015 program.

Ka-Tai Tangwangvivat (pictured above) was our happy winner from Thailand

IT'S NOT TOO LATE – PLACES STILL AVAILABLE IN SOME COURSES
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THANK YOU TO ALL CONTRIBUTORS

… and more C&T articles and Perspectives are 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.

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- **Adam Gordon**– What's your diagnosis?

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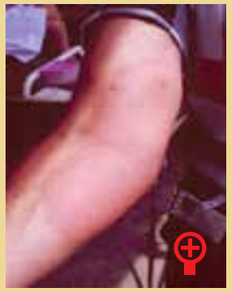
HUMAN TICK-RELATED DISEASES

Have you heard of red meat allergy…?

It is one of 3 common serious medical complaints caused by ticks in humans.

CVE is working with Royal North Shore Hospital's Tick-Induced Allergies Research and Awareness group www.tiara.org.au to disseminate this important information.

Download C&T No. 5416 'How Much Do You Know About Human Tick-Related Diseases' from www.cve.edu.au.



A very large local reaction to a tick bite on the inner aspect of the upper arm.

Download TiARA's informative pamphlet to hand out to your clients - Preventing and Managing Tick Bites

Established in 1965, the C&T is the brainchild of Dr Tom Hungerford, first Director of the PGF (renamed the CVE) from 1968-1987, who wanted a forum for uncensored and unedited material.
Tom wanted to get the clinicians writing.

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

WINNER OF BEST IMAGES

WHAT'S YOUR DIAGNOSIS?

C&T NO. 5424

Todd Browning

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213 Bexley Road, Earlwood NSW 2206
T. (02) 9718 5235 F. (02) 9789 1956
E. earlwoodanimalhospital@gmail.com

The patient, a desexed female cat, ex RSPCA, approximately 7½ years old from an indoor multi-cat household presented with multiple discrete small oval lesions on her sternum which literally oozed fluid, and she was also quite uncomfortable to touch.

Cytology from a lesion revealed suppurative inflammation. She was placed on doxycycline and did amazingly well and today you cannot tell there was an issue. She has been off doxycycline for 3 weeks now.

Go to our eBook to see A4 versions of these images.

Figures 1-3. The patient at presentation.

Figures 4-5. The patient 14 days later, after treatment.

Email your answer to Elisabeth.churchward@sydney.edu.au.

Author of the most correct answer wins a CVE proceedings of their choice. Go to: www.vetbookshop.com to peruse our titles.

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SUDDEN DEATHS IN YOUNG FREE RANGE PIGS

C&T NO. 5425

Jeremy Rogers
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DISEASE INVESTIGATION – 15TH JULY 2013
Government of South Australia
PIRSA Biosecurity – Animal Health, Murray Bridge

Introduction

A local farmer raising free range pigs in the lower Mallee area of SA contacted PIRSA on the 15th July to describe sudden deaths in his pigs. A Primary Industries and Regions SA (PIRSA) vet responded within an hour of the report, and a number of post mortem examinations were conducted, and samples collected for laboratory tests. Results are inconclusive but indicate a peracute clostridial disease as the most likely cause. One further death was noted on 18th July, and no further deaths after that.

History

A mob of around 200 mixed breed pigs are being raised in a 'free range' management system about 50 Km to the North of Murray Bridge. The area is a predominantly sheep and cereal cropping region with rainfall between 250 to 350 mm annually.

The owner has approximately 20 sows, 20 gilts and 60 other pigs of various ages between newborn and 6 months of age. With the exception of newborn pigs, all ages are run together on an area of approximately 1 Ha that comprises some Mallee scrub and ryegrass / mixed pasture. The pigs are contained by electric fencing and shelter is constructed of large hay bales stacked in the paddock. The pigs are fed hammer milled barley daily that is fed out on to the pasture. The diet has not changed for some time, and surviving pigs appear healthy and have good growth rates. There is no access to poison plants, fungi or rubbish dumps etc.

The weather during the event had been unusually cold at nights.

Date	Number & age	location
18/07/2013	1 10- 20 kg pig	In haystack
15/0720/13	1 bacon size, 4 weaners	In sheds/ haystack, found in morning
14/07/ 2013	5 dead young pigs ~ 20kg	In paddock
08/07/ 2013	3 dead all around 5-6 months	In paddock
Totals	14 age from 3-6 months age	

Clinical symptoms / PM findings

With the exception of 1 pig, all pigs were found dead in the morning with no symptoms prior to this. A common observation was that the carcasses putrefied very rapidly, even though the temperatures had been cold. With some of the earlier deaths, the owner noted some white froth from the mouth. All other pigs appeared healthy and in good condition. Dead pigs presented for necropsy were all highly autolysed, especially abdominal organs with advanced subcutaneous emphysema around the head and neck area, or generalised. In some cases abdominal musculature had ruptured with gas-filled intestinal loops just under the skin. These pigs had been dead in very cold conditions for perhaps 10-12 hours. In one fresher animal lungs and chest appeared normal, with no evidence of pneumonia. Meninges in all pigs appeared to be engorged, and turbinates appeared congested in all bodies. There was no evidence of inflammatory bowel disease in any pigs, and all bodies were observed to have stomachs full

Figure 1. This pig representative of others with rapid carcass decomposition and subcutaneous emphysema

of grass / grain mixture, indicating that death had occurred within a relatively short time after feeding with no signs of struggling. Petechiae or ecchymotic haemorrhages were not observed in any carcass. There was no sign of diarrhoea in any pigs.

Results from samples

Laboratory test results have not been helpful in identifying a specific cause, but have ruled out some common endemic diseases. Salmonella bootle was cultured from 1 sample of faeces.

Diagnosis

A list of causes of sudden death in pigs is attached . The condition that most fits the observed signs and findings is peracute malignant oedema (Cl septicum, Cl novyi).

Treatment

The owner has been advised to vaccinate the herd with Clostridial vaccines immediately, with a follow up booster in 4 weeks. This group of pigs had not been vaccinated previously.

Discussion

Free range farming operations offer a number of advantages in terms of infrastructure investment and animal health where the environment is properly managed, but disadvantages include the lack of adequate facilities to handle or treat animals in a situation of a disease outbreak. In this event it appears that the most likely cause was a vaccine preventable condition.

Recommendations

Vaccination against common pig pathogens is recommended, including Erysipelas, and the range of clostridial diseases. Although there are no specific registered clostridial vaccines available for pigs Ultravac 5 in 1 (Zoetis) is a recommended off-label vaccine. The dose is 2 mLs and should be administered subcutaneously using a short needle to avoid abscesses and carcass damage.

• A list of causes of sudden death in pigs – Vade Mecum series B, number 8 'The diagnosis of the diseases of pigs' University of Sydney Post Graduate Foundation in Veterinary Science (renamed the CVE), Nov 1987 (available in the CVe-library)

• Laboratory results (see eBook)

Acknowledgements: Dr Barry Lloyd for assistance in diagnosis and recommendations.

FROM THE DE FILES
WINNER!

UNCONTROLLED FELINE DIABETES MELLITUS

C&T NO. 5426

Betty Liem
DE Participant 2013 (Internal Medicine: A Problem Solving Approach)

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History

'Zander' is a 13-year-old male neutered domestic short hair cat that was presented for weight loss despite ravenous appetite and increased drinking and urination. He is an outdoor cat and has no history of vomiting and diarrhoea.

Physical examination

Physically Zander appeared normal except for moderate dental calculus, unkempt coat and a body condition score of 3/9. There were no palpable thyroids and chest auscultation was normal.

Investigations

Haematology

Test	Result	Ref	Test	Result	Ref
RBC	7.1	4.9-10.0x10^12/L	WBC	10.6	5.5-19.0x10^9
Haemoglobin	105	77-156 g/L	Neutrophil%	68%	
Reticulocyte%	1.8	0.0-0.4%	Neutrophils	7.2	2.0-13.0x10^9/L
Reticulocyte	128	3-50x10^9/L	Lymphocyte%	13%	
MCV	54	43-55 fL	Lymphocytes	1.4	0.9-7.0x10^9/L
MCH	15	13-17 pg	Monocytes%	4%	
MCHC	276	282-333 g/L	Monocytes	0.4	0.0-0.6x10^9/L
Platelets	Clumped and adequate		Eosinophils%	15%	
Platelet count	187	300-800x10^9/L	Eosinophils	1.6	0.0-1.0x10^9/L
Blood smear examination: Mild anisocytosis, mild macrocytosis White cell morphology normal			Basophils%	0%	
			Basophils	0.0	

Biochemistry

Sodium	144	144-158mmol/L	Calcium	2.6	2.1-2.8mmol/L
Potassium	5.4	3.7-5.4mmol/L	Phosphate	1.5	1.0-2.3mmol/L
Chloride	101	106-123mmol/L	Ca:P Ratio	1.7	1.1-2.3
Bicarbonate	23	12-24mmol/L	Protein total	96	60-84 g/L
Na:K Ratio	26.7	>29.0	Albumin	33	25-38g/L
Anion gap	25.4	15.0-31.0mmol/L	Globulin	63	31-52 g/L
Glucose,Flox	25.1	3.2-7.5mmol/L	A:G Ratio	0.5	0.5-1.1
Glucose Serum	28.5	3.2-7.5mmol/L	Bilirubin total	5	0-7 umol/L
Urea	12.5	5.0-15.0mmol/L	ALP	36	5-50IU/L
Creatinine	0.13	0.08-0.20mmol/L	AST	43	2-62 IU/L
Cholesterol	7.7	2.2-5.5mmol/L	ALT	91	19-100 IU/L
Gamma GI	Below limit of detection		CK	92	64-400IU/L
Serum appearance: Slightly haemolysed			T4 total	12	10-60nmol/L
			Beta-hydroxybutyrate	2.5	0.0-0.5mmol/L

Urinalysis (cystocentesis)

Blood	++++	Glucose	++++	USG	1.032
Bilirubin	Negative	Protein	+	Urine culture and sensitivity:	
Ketone	Negative	PH	5	<10 000 orgs/mL. Negative.	

Inhouse tests

FIV	Positive	FeLV	Negative	fPLi	Abnormal

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Management

Zander was diagnosed with diabetes mellitus based on the presence of hyperglycaemia, glucosuria and his typical presenting signs. He was also infected with feline immunodeficiency virus since he was not previously vaccinated against FIV, which may have contributed to his hyperglobulinaemia from chronic antigenic stimulation. We started Zander on glargine insulin at 1 IU SID and changed to a low carbohydrate diet (Royal Canin Prescription Diet Diabetic Dry®). Glargine is a long-acting insulin analogue, which can be administered once or twice daily. It provides excellent duration of action in cats, hence better glycaemic control than human or porcine lente insulin, NPH or ultralente insulins[1]. Blood glucose curves were performed every 10 to 14 days with an increment of 1 IU glargine twice daily per visit if required. His blood glucose curve was still uncontrolled at 6 IU (1.3 IU/kg) though his body weight has been stable (around 4.5 to 4.7 kg) and he has always been well at home based on owner's perception. His appetite has always been excellent and water intake test could not be performed as multi-cat household.

Insulin resistance is defined as decreased sensitivity to insulin[2]. Although there is no insulin dose that clearly defines insulin resistance, most cats achieved good control with a dose ≤1 IU/kg/dose. Insulin resistance should be suspected if glycaemic control is poor despite insulin dose ≥1.5IU/kg/dose. The most common causes of insulin resistance in cats are acromegaly, hyperadrenocorticism, obesity and renal failure. Other less common causes include hyperthyroidism, bacterial infection and steroid use[4].

Most likely potential reasons for Zander's poorly controlled diabetes were acromegaly and hyperadrenocorticism. In patients with acromegaly, physical examination may reveal organomegaly, inferior prognathia (undershot lower jaw), cataracts, clubbed paws, broad facial features, widened interdental spaces, cardiac murmurs or arrhythmias, respiratory stridor, and neurological signs. Even so, acromegaly could not be ruled out in Zander as some cats with acromegaly may be phenotypically indistinguishable from normal cats. Tentative diagnosis can be made by measurement of GH and IGF-1 concentrations and imaging with CT or MRI of the brain[2]. Whereas in patients with hyperadrenocorticism, physical examination may reveal hepatomegaly, seborrhoea, skin fragility, pot-bellied appearance, muscle atrophy and bilateral symmetric alopecia. Similar to acromegaly, a lack of clinical symptoms does not exclude the diagnosis. To confirm diagnosis, ACTH stimulation test and low-dose dexamethasone suppression test can be performed and urine cortisol:creatinine ratio can be used as a screening test for hyperadrenocorticism[2] and abdominal imaging (ultrasound or CT) can be used to evaluate adrenal size. At that stage, we have decided to continue to increase insulin dose by 1 IU every 2 weeks up until 10 IU BID before considering further testings for the above possibilities. We have also modified his diet to include more protein source- with boiled chicken as main diet and prescription dry diet to graze on during the day to further improve his glycaemic control, since carbohydrate is thought to be responsible for insulin resistance and diabetes mellitus in domestic cats[3].

WEBCAST SHARED FROM THE ISFM FORUM

Prof Tony Buffington, Professor of Veterinary Clinical Sciences, Ohio State University, USA presents 'FROM FUS TO PANDORA SYNDROME: How to manage stress and cystitis in cats?' at École Nationale Vétérinarie D'Alfort.

In English: vo-live.fr/vod/video.php?1gzN

or French: vetoquinol-contact.fr/webconference

Conclusion

Zander's blood glucose was finally under good control at 8 IU glargine BID. His insulin resistance was probably only mild and glycaemic control can still be achieved at higher insulin dose at 1.7 IU/kg, combined with a high protein, low carbohydrate diet. Zander will be re-evaluated in a month time and owner will continue to monitor his demeanor, appetite, urine glucose and body weight at home.

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Follow up

Zander's diabetes has been under good control since initial management. He has maintained his body weight at 6.2 kg with 8 IU glargine BID. Subsequent frutosamine levels at 1 month and 3 months were 281 and 273 umol/L indicating excellent glycaemic control (249-406 umol/L).

CONGRATULATIONS TO OUR BEST 'VET PET' FB COMP WINNERS!

Fifi' (346 likes), won her owner Kay Gerry first prize of CVE$500 toward CPD of her choice.

Second place Melissa Kozaruk's dog Evie (255 likes) and third place Kerrie Rodgers' cat Frankie (178 likes.)

A big thanks to Hill's Pet Nutrition for providing first and second prizes, and to the 72 people who entered the comp, and everyone who voted.

www.facebook.com/cvesydney

Like

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HOW TO MAKE A 'CAT FRIENDLY' WAITING ROOM

C&T NO. 5427

Andrea Harvey
RCVS Recognised Specialist in Feline Medicine
European Veterinary Specialist in Internal Medicine
CVE/ISFM Feline Medicine Distance Education Tutor

Read these articles in the eBook

	ISFM Cat Friendly Clinic Accreditation Scheme is Launched in Australasia, Sept 2014, Issue 273
	Creating a 'Cat Friendly Clinic' & working towards ISFM Accreditation, Dec 2014, Issue 273

In 2 issues of C&T last year (September 2013, Issue 272 & December 2013 Issue 273), 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 was launched in Australasia in 2013, with the support of Royal Canin and CEVA. In previous articles, Andrea discussed the importance of stress in cats, the impact of this on feline patients and the veterinary clinic and some practical advice for how veterinary practices can become more 'cat friendly'.

Since that time nearly 300 Australasian practices have registered their interest in the ISFM Cat Friendly Clinic Accreditation scheme, and around half a dozen have already become accredited. However, many people find some of the requirements a little daunting and don't know where to start, or are put off by some criteria which may at first seem unachievable for their clinic. One of the most common stumbling blocks is creating a cat friendly waiting room, and this article specifically addresses just that: how to get creative and convert any waiting room into a cat friendly one!

INTRODUCTION

Firstly, with any potential changes you may be planning in transforming your clinic to a 'cat friendly clinic', remember the basics of what you are trying to achieve and why. It can then be easier to think more creatively about 'how' to achieve these aims.

Stress and anxiety has many impacts on both the cat's wellbeing (reduced eating, urination, grooming, adverse effects on pain and healing, recrudescence of viral infections etc) and our ability to practice good feline medicine. A stressed/anxious cat is likely to be less easily handled and increases the risk of staff injuries, as well as reduced ability to carry out examinations and procedures. Stress also affects many parameters such as heart rate, respiratory rate, temperature, blood pressure, blood glucose etc and so makes interpreting all of these parameters very difficult. Signs of pain are also similar to signs of stress, and therefore pain is very difficult to optimally manage and monitor in a stressed cat.

Figure 1. Make the most of your available space with vertical shelving

Managing stress in cats within a vet clinic environment is therefore paramount to be able to practice a high standard of feline medicine and offer the best overall care to our feline patients. Furthermore, the stress of bringing a cat into a veterinary clinic has been recognised as an important factor for clients being less likely to seek veterinary attention for their cats, both when sick and for preventative health care.

A veterinary clinic situation typically comprises most of the factors that cause anxiety and fear in cats; an unfamiliar and often busy environment, noise, smells, strangers, dogs, intervention, loss of control of their environment and inability to use their normal coping mechanisms such as running away or hiding. The aim of a 'cat friendly clinic' is to try and minimise all of these factors to reduce fear and anxiety in our feline patients.

THE WAITING ROOM: THE GATEWAY TO THE CLINIC

The waiting room is a key area, as this is the gateway to the clinic. It sets the scene for the rest of the clinic, and sets the cat's mood for the consult room!

The overall aim with the waiting room is to create a calm and unthreatening environment for the cat to wait in so that it is not even more fearful by the time it reaches the consultation room, and also an atmosphere that reassures feline owners that this is a clinic staffed by people who care about both them and their cats.

The first step is to evaluate your current waiting room, trying to think about 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? 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?

The best way of creating a cat friendly waiting room will be different for every clinic, since it depends on the physical size of the clinic, the caseload, the cat vs dog proportions, and how the appointment times work in terms of the typical number of clients that you may have in the waiting room at any one time. The key is thinking about your individual clinic, and how from the moment a cat client walks in the door, to when they go through to the consult room, what can be done to minimise any visual, aural and olfactory threats that the cat may feel.

SEPARATE CAT WAITING AREAS

Obviously the ideal is to have some way of separating cat and dog waiting areas, as being in close vicinity to dogs is one of the major factors contributing to the stressors discussed. For most practices, space doesn't allow for completely separate areas, but often barriers can be used to at least go some way in dividing the waiting area. These may be multipurpose and contribute to the décor of the practice; for example, a barrier can be used as a notice board, information board, or the barrier may be shelving that could contain information, or could be shelves for placing cat carriers on. The images overleaf show some examples of how barriers can be utilised.

YOU CAN'T ACCOMMODATE A SEPARATE WAITING AREA FOR CATS? SO, GET CREATIVE!

This all sounds ideal, but is your waiting room too small for separate areas, or vertical barriers of any type? If so, don't panic, but instead just get more creative and think outside of the box!

- **Utilise vertical space**
  - Remember, cats feel safer at a height, and using vertical space can be a good way of getting them out from underneath dogs' noses and avoiding eye contact with other animals. Figure 1 illustrates a good example of how this may be achieved without taking up extra floor space (Image courtesy of Village Vets, UK)
- **Utilise space outside of the traditional waiting room**
  - Small practices may have limited waiting room space, but this also means they are likely to have minimal cat clients waiting at any one time, perhaps only one. In this situation, rather than leave them sitting next to a dog, there may be a small space or shelf behind the reception desk that can be utilised for placing cats in their carriers whilst the client sits in the usual area. Or perhaps there is a spare consult room that the client can go directly into rather than staying in the waiting room. When you look carefully and creatively, the vast majority of clinics will have somewhere that can be utilised as a quieter raised area for waiting cats.
- **Use space in time**
  - If it is impossible to have any sort of physical separation between cats and dogs, perhaps you could set up cat only consulting times? This may even be possible to extend to the hospital for routine procedures such as desexing and dentals – try and schedule to do cats on a different day from dogs.

OTHER WAITING ROOM TOP TIPS

Also consider the route to the cat waiting area and from the cat waiting area to the consult room and reception desk. There is little value creating a great cat waiting area if the cats are then

placed on the floor next to a dog at the reception desk, or have to walk through waiting dogs to get to the consult room.

- **Raise baskets**
  - Regardless of the type of waiting area, cats feel safer at a height, and having raised surfaces to place cat carriers can make a big difference
- **Cover carriers**
  - Similarly, covering cat carriers goes a long way to giving the cats an opportunity to hide, and avoiding direct visual contact with other cats as well as dogs. Simple gestures like providing a box of towels in the waiting area, with a sign telling cat clients to use these to cover their cats' carriers to help make them feel safer whilst waiting, goes a long way not just for the cats, but also clearly demonstrating to cat owners that you understand cats and care about trying to reduce their anxiety in the clinic
- **Display clear notices**
  - Ensure that the cat waiting area is clearly labeled, and that signage directs dog and cat clients to their appropriate areas
  - Have additional notices politely asking clients with dogs to keep them away from any cats in the waiting area
  - Use notices to instruct clients to use the raised surfaces, and cage covers, explaining why these are important

Utilise feline pheromones

- Consider having plug in Feliway® diffusers in the waiting area and spraying any cage covers with Feliway®

Feline information in waiting area

- Display useful staff information such as who the 'cat advocate(s)' are, information about cat carriers, transport advice, medicating advice etc

Reception desk area

- This can be an important consideration in trying to stop cat and dog encounters occurring at the reception desk. A narrow area in front of the reception desk encourages these close encounters. Conversely, a low wide reception desk encourages owners to place cat carriers on top of the desk, out of the reach of dogs' noses

CAT FRIENDLY RECEPTIONISTS PLAY A VITAL ROLE

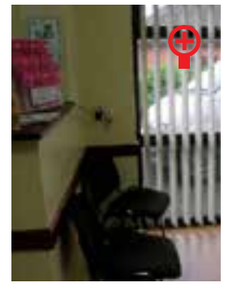
Do your receptionists show an understanding and empathy for the feline patients? The cat friendly waiting area will only work well if receptionists are also aware of what factors cause anxiety to cats, and how to address these. They are the staff that see what happens in the waiting room, and need to notice the potential for anxious cats, or where dog and cat encounters may occur. They can help to reinforce any separation by asking dog-owning clients to be considerate of cats in the waiting area, ensuring that they notice and correct the situation if a dog client sits in the cat area. Training receptionists to encourage cat clients to use the raised surfaces and carrier covers, and explaining why they should use these, helps to educate cat owners, reinforces utilisation of these aids, and again helps to demonstrate to cat clients that all the clinic staff understand cats and are doing all they can to minimise their anxiety in the clinic.

For more detailed information, to apply for a FREE 'ISFM Cat Friendly Clinic' information pack which includes a full veterinary guide, all the details of the scheme and how to apply for accreditation, visit: **tinyurl.com/isfmcfc** (or go to the International Cat Care website for further information: **www.icatcare.org:8080/.**

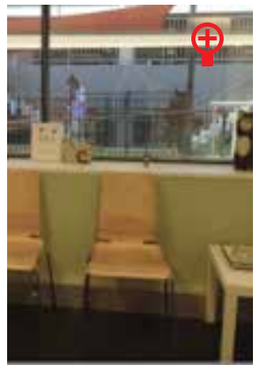
This article sponsored by CEVA and Royal Canin

RECEPTION AREA

SEPARATE CAT WAITING AREA



COVERED CARRIERS



RAISED CARRIER

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DELIBERATE BEFORE YOU MEDICATE

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SOMETIMES MORE IMPORTANT TO KNOW WHEN NOT TO USE A DRUG THAN IT IS TO KNOW ONLY WHEN TO USE!

I put this series of info bits together after inheriting a flurry of second opinion cases some time ago. The common thread was that all had minor to major clinical problems induced by vets racing to embrace new medications without considering possible pitfalls. When the patients experienced negative unexpected outcomes, often at potential major expense to the owner, no one had reviewed the possible responsibility said new drugs might have played. Hopefully, these snippets below will save other pets and vets some stress and grief early on in a puzzling case.

1. Unstable Epileptics

A. Don't go on an extended **Honeymoon with Keppra®)** – cheaper to stick with a **3-day break**.

Levetiracetam (Keppra®) is a good (albeit very expensive!) drug that has not been used thoughtfully by some who raced to embrace its use. Rather than use as a daily or as a stand-alone, replacement anti-seizure medication, this drug is suggested to work much better when used as a pulse therapy for cluster seizures so as to overcome tolerance issues. Pulse therapy, rather than maintenance therapy, avoids depressed and despairing clients, who further down the track think all hope is lost when the seizures return with a vengeance, even whilst their pet is on this daily new expensive supposed top-of-the range anti-epilepsy medication.

The pulse therapy can be dosed in one of 2 ways;

- In addition to phenobarbitone, Br etc maintenance therapy: 30mg/kg Keppra® PO every 6-8hours for the duration of the cluster (2-3days normally) then stopped once the patient is seizure-free for 24hrs and not resumed until the next cluster occurs. We have used this in a small number of cases, but equally found lower doses – 10-30mg/kg max every 8-12 hours – worked in the cohort of dogs also taking long term, daily, anti-epilepsy medication.
- As a stand-alone treatment, where the seizures occur within a short period of time (e.g. 24-48 hours) followed by a long inter-ictal period of weeks or months, Keppra® can be given during these severe clusters to reduce the number and severity of the seizures. Pulse therapy dosing is at the higher level; 30mg/kg tid to qid in this mono therapy cohort.

This way, some of the high costs of the drug are avoided, as well as reducing the possibility of tolerance.

My personal impressions are that there is less likelihood of owners self-medicating themselves with Keppra® (whereas I sometimes got concerned with owner requests for home Valium® supplies) and that it works better than home use Valium®. Unlike Valium, owners have not reported any increased aggression in their pets. However, you do need to follow these pulse clients up and make sure they have actually stopped the drug as you advised, since 'over compliance' from an owner who perceives the drug is 'curing' the epilepsy and is afraid to stop using Keppra® can also be a problem. (I got caught out on that with a good client).

B. But best of all – before you add in any new epileptic drugs – **First check the phenobarbitone blood peak and trough** to make sure you don't have any ***habituation, compliance or enzyme auto induction issues*** that occur with long term use. Clinically, auto induction will show as a controlled, epileptic-throwing-increased-seizure activity – this dog is NOT refractory to the phenobarbital until you have checked the serum concentration – wherein you will often find that the dog may now just need a higher dose to remain in the therapeutic serum phenobarbital concentration.

C. If using **Bromide**, remember: High salt diets or salt ingestion affect clearance; so check carefully the salt content of your epileptic diets – especially supermarket dog treats which can often have 28X salt levels compared to the 0.2% seen in many premium diets. Kathy Lin's excellent article available in the eBook (below) or the CVe-library is a must read on this salt diet content issue.

	'Hypernatraemia: The importance of understanding labelling in commercial dog diets', SA Perspective, C&T June 2011.
	Tables 1-3 enlarged for download here

D. Rectal **Valium**
Send Valium home in syringes but **don't use plastic syringes** if possible to avoid the diazepam/plastic interface reaction issue. Most wholesalers, especially those who have a human medical supply division, have very cheap glass syringes you can fill and then plug for rectal home use in the epileptic patient.

E. Gabapentin – I love gabapentin! I'm going through bucket loads, especially for palliative arthritic care to add-in when NSAIDs have reduced efficacy. I have not got to Ariane Goerlich's emergency analgesic rush load of 30mg/kg yet. I use her 5mg/kg x BID 2 days then 10mg x 2 days step up and most dogs stabilise on 10mg/kg with no sedation effects. Cats on 5mg/kg bid.

This lower induction dose regime avoids inducing diarrhoea and ataxia in the home-treated patient. **Don't stop suddenly**,

especially if the dog is a known epileptic, but also in any patient as it can flush out your occult or borderline epileptic if you withdraw long term treatment abruptly. If the patient is on it for more than 4 days, then I take the same amount of time to wean it off the drug as it was on it for. Always check kidney and liver function before starting the drug.

Wear gloves. Child-bearing age women should avoid handling the drug.

The full dose range is 10-60mg/kg total divided bid-tid in dogs and 5-10mg/kg bid-tid in cats.

Recently I am finding gabapentin very helpful:-

- Early-on in the onset of clinical signs of IVDD cases –Types 1-3 disc protrusions/extrusions as gabapentin is both an excellent neuropathic drug in these instances as well as calming the distressed and anxious dog.
- Superb! for Feline Idiopathic Ulcerative Dermatosis where gabapentin co-use allows much lower doses of prednisolone to be used.

Our local chemist or any compounder can compound up a liquid version for small pets with no xylitol, no artificial sweetener and no propylene glycol in it and can add flavour as optional extra e.g. beef/chicken/fish and liver. Cost: 25mg/per mL in 100mL is $50 and the 50mg/per mL in 100mL is $60.

Note: I would love feedback from C&T Series readers on gabapentin use in feline facial pruritic syndromes as well.

2. Killer Pain Relief LITERALLY! Opioid Induced Anorexia (OIA)

Panting, **trembling**, hiding, salivation, aggressiveness, and anorexia can all be signs of pain but equally they can all be the signs of a dog having an adverse or idiosyncratic response to a drug. Know the signs of pain but when you are loading up a dog with more **Temgesic/Torbugesic** for its recalcitrant pain and yet nothing seems to be touching the dog's distress – STOP.

An animal in pain may pace, but mostly they prefer to sit still and reduce movement, so if in constant motion, move the analgesic opioid/opiate drug reaction thought up your d/d list.

An animal in pain will often not eat. But if the inappetence persists after a good dose of opioid/opiate and that inappetence seems to be a severe and recalcitrant expression, (especially if in a stomach on legs like a Dachshund, Beagle or Labrador), remember that there is a syndrome called **Opioid Induced Anorexic**. Stop the drug, and the dog will often eat several hours later.

(*Cerenia may be a much better choice for pain and emesis control in such cases.)*

3. Tapeworms – a blast from the past now back with us in increasing numbers, so remember the 'Dexamethasone 50%' rule.

With the advent of combination flea and partial deworming products came a reduction in oral all-in-one gastrointestinal deworming usage. With that followed a slow, but consistent, reappearance in tapeworm cases.

Given many tapeworm-infested dogs are concurrently pruritic (often throwing erythematous lesions along the ventral skin, perianal and rectal tissues), remember that glucocorticoids like dexamethasone used at the same time as dewormers such as praziquantel could, in theory, cause ***50% reduction in efficacy of the tape dewormer***. So, if you co-address the pruritus but not the potential reduction in dewormer efficacy, the tapeworm problem may persist. Given how incredibly cost effective a bulk 100 tab pot of Virbac Praziquantel is to the vet, then an easy solution is to extend the repeat 14 day interval re-dosing to perhaps a course of 3 or 4 repeats, not the normal course of 2 treatments.

4. Cardiac Meds

I would ask Vets who prescribe **spironolactone** to be very cognisant of the potential side effect this drug can have on the gastrointestinal tract, and to be alert to the first signs of gastric bleeding in these patients.

We can all agree that cardiac cases and massive digestive tract ulceration are not 2 clinical syndromes we would traditionally put together.

Yet, in recent years I have been involved in 3 cases where the animal was placed on spironolactone. When poor appetite, reflux and cachexia became apparent, no one thought to do an open-minded full physical exam including a basic faecal scoop, wherein dark bloody stools would also have been discovered…

The imminent fatal gastric bleed in one case might have been prevented if the following had all been considered:-

- The breed – GSD
- The drug – Spironolactone
- The co-presence of ACE inhibitor medication
- The signs of gastric pain – drooling, reflux, poor appetite
- The dark black stools

None were...

The dog was too far gone for me to save by the time I first saw her. This gorgeous dog must have been in horrendous pain, unaddressed, due to the focus on her primary cardiac condition and not on her overall presentation.

The others dogs' ulcers (Chihuahua and GSD) were detected and treated in time but, again, no thought initially had been given to the Spironolactone before I suggested it as a potential cause of the presenting signs.

If:-

1. High potassium concentrations can lead to gastric ulcerations and stenosis.
2. *ACE inhibitors, angiotensin II receptor antagonists, trimethoprim, heparin, potassium supplements and potassium-containing medications (e.g. penicillin G potassium) can increase the risk of hyperkalaemia developing in patients receiving spironolactone, especially in the presence of renal impairment (renal disease, elderly patients). These agents should be used with caution and serum potassium levels monitored when the substances are concurrently administered with* spironolactone. *AND*
3. *In the human field, a cohort study and 2 case-control studies concurred to show an approximately twofold, dose-dependent, increase in the incidence of upper gastrointestinal bleeding in patients receiving Spironolactone. The postulated mechanism is that spironolactone's aldosterone antagonist activity delays gastro duodenal healing. However, gastrointestinal bleeding is not a common adverse effect of aldosterone antagonists. In practice, although these studies have a low level of evidence, the role of spironolactone should be suspected in the event of a gastrointestinal bleed and caution should be exercised with prescribing spironolactone in patients who have predisposing factors to gastrointestinal bleeding.*

Then we need to be more vigilant in our use of Spironolactone. I don't use Spironolactone, but if I did, then I would:-

- Pick my cases carefully
- Pick my breeds – avoid use, if possible, in GSD and Dachshunds, breeds which historically appear to be at higher risk for gastric/duodenal ulceration, respectively, than others.
- Evaluate temperament and not use it on fussy eaters with capricious appetites and poor regularity of eating.
- Remain alert for any non-primary cardiac signs beginning to appear.

Good old cheap Furosemide may not have the double cardiac remodelling diuretic action of Spironolactone but that doesn't negate the efficacy and safety of Furosemide; in fact, Lasix may just be a much safer drug generally.

One valid use of Spironolactone may be in ***liver*** disease and ***ascite***s but an ***alternative drug*** – ***Zaroxolyn*** – is very popular with USA vets for ascites.

For more information on methods surgical, mechanical and drug related treatment for severe ascites read:

C&T No. 5316 Fulminant ascites options – drugs and scalpels, June 2013.

The same arguments to 'Deliberate Before You Medicate' apply when you select OMEPRAZOLE/LOSEC® (PPIs- proton pump inhibitor) rather than drugs like CIMETIDINE.

I initially submitted this article to C&T about 4 years ago but never got around to finishing it off until now – when, by happenstance, cimetidine is not currently available in Australia. However, the argument below still stands to redress the imbalance against the drug and those who used it and show that the logic used to promote an alternative (still available) was incomplete and incorrect. All the recent 2014 new guidelines for PPI use further validates my concerns that we were being told to replace good, proven practice with a newer unfamiliar drug without fully investigating this newer class of drug. Well, I had done my due diligence and investigated these new proton pump inhibitors and was dismayed at the partial facts taught to vets by others. Hence this article below:-

I always had Tagamet/cimetidine on the shelf and so was amused to find the discovering of my almost blasé use of this drug quite startling to many Australian vets who have been taught that only ***Bad vets use Tagamet and Good vets use Losec®. Hmmh...***

Cimetidine (Tagamet-Movicol) was the ONLY REGISTERED DRUG vets in Europe could use as first-cab-off-the-rank for gastric conditions. **It has been used in possibly millions of dogs by good and great vets over the years to excellent effect.** Cimetidine is cheap and easily to give and has a multitude of uses in paracetamol poisonings, pancreatitis, oesophageal reflux and also of co-use in some German Shepherds with EPA.

Whilst I have no issue with Losec® (in fact use it on myself on occasion and also in my patients), I was intrigued as to the bad press about Tagamet. I was even more surprised when the reasons why were explained to me by other vets who had never use Tagamet. Then I did have a problem!

But not with Tagamet, but with the arguments used against it – arguments which showed a huge degree of under-education by its opponents on the reasons why they hate Tagamet and love Losec®.

Not one vet was aware of the proposed 8 week time restriction usage of Losec®.

Everyone 'talked' about the slow onset of Losec® and the need to transition into it over 3 days and the need to keep using cimetidine for a few days to tide over the switch off it onto Losec® – but this is only partially true...

But mostly, the main problem with the arguments for not using the one drug, Tagamet, and choosing another, omeprazole, '*because of a hypergastrinaemia or a gastrin rebound effect concern'* is that those concerns are flawed, incomplete and incorrectly exonerate omeprazole. It, too, has that very same, in fact sometimes worse, hyper-gastrin side-effect!

Historically, cimetidine once got bad press about gastric rebound in the 1970s because when humans stopped taking it, their ulcers flared back up. Not surprising, given we now know that those ulcers needed *a triad of antibiotic etc – not Tagamet alone – to cure them.*

Let's look at omeprazole

a. Omeprazole is 10 times more potent than cimetidine inhibiting gastric acid secretion and has a longer duration of activity (>24) hours.
Chronic suppression of acid secretion in lab animals leads to hypergastrinaemia and mucosal cell hyperplasia, rugal hypertrophy and development of carcinoids so a MAXIMUM OF EIGHT WEEKS THERAPY is recommended. (Trials have indicated extended use is safe in humans but the animal caution remains valid.) How many omeprazole vets use only an 8 week course?

b. Claim: 'Omeprazole takes 3 days to work' – not completely correct. *'After oral administration, the onset of the anti-secretory effect of omeprazole occurs within 1 hour, with the maximum effect occurring within 2 hours. Inhibition of secretion is about 50% of maximum at 24 hours and the duration of inhibition lasts up to 72 hours.'*

Because initial absorption is rapid, with peak plasma levels of omeprazole occurring within 0.5 to 3.5 hours, (data sheet for Prilosec®), I and other vets have found that a percentage of dogs and people will vomit on omeprazole within an hour of taking the drug and will continue to do so each time it is administered. This drug is of no use in those patients.

So, don't ignore a client who suggests Losec® makes their dog vomit and neither panic that the emesis is a sign of patient deterioration or unstable treatment. Just stop the drug, and the vomiting stops very quickly.

Equally, other clients will report fairly rapid onset of gastric pain relief within hours of commencing the omeprazole.

To back up the first-day rapid-onset claim, rather than third-day lag-effect claim, evidence is found within the mounting concern that newer anaesthetic drugs increase gastric reflux and oesophagitis risk post-surgery, especially with propofol. The drive is to give omeprazole the night before. This protective efficacy can only occur if omeprazole takes effect far quicker than in the 2-3 day lag effect.

c. The anti-secretory effect lasts far longer than would be expected from the very short (less than 1 hour) plasma half-life, apparently due to prolonged binding to the parietal H^+/K^+ ATPase enzyme. The inhibitory effect of omeprazole on acid secretion increases

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with repeated once-daily dosing, reaching a plateau after 4 days. When the drug is discontinued, secretory activity returns gradually, over 3 to 5 days.

The down side of omeprazole's parietal cell persistence is that it is difficult to titrate out of this drug. The shorter persistence of cimetidine allows any potential rebound gastrin effect to be reduced/eliminated by tapering off the cimetidine. The longer persistence of omeprazole makes that phasing out harder to achieve, as it's much harder to step down and to titrate out the omeprazole. ***As a result, we can get an omeprazole-induced recalcitrant hypergastrinaemia syndrome that can persist for 14 days unabated by any drug we have to use against it! Yet, I'm told if I want to prevent hypergastrin rebound to use omeprazole not cimetidine – you can see why I am unhappy.***

'Because gastrin secretion by the G cells of the antral mucosa is inhibited by an acid pH in the gastric lumen, hypergastrinaemia may be induced by any treatment that decreases gastric acidity. For example, the administration of H_2-receptor antagonists causes an increase in plasma gastrin levels proportional to the dose [6]. Omeprazole administration causes a greater increase because it is a more potent inhibitor of gastric acid secretion. In one study, the magnitude of the gastrin response corresponded with the degree of acid inhibition and pH increase. Therefore, the data support the hypothesis that the hypergastrinaemia caused by omeprazole is dependent on gastric pH and GAS suppression.'

d. Few vets were aware that Tagamet only gets the pH to 4-5, and omeprazole to neutral ph. Whilst the latter is of great benefit in, say, Zollinger Ellis gastrinoma syndrome, **a neutral pH brings other baggage**. A neutral pH carries concerns about increased bacterial overgrowth and reduction in Vitamin B absorption. Omeprazole can take acid pH to neutral – at this low pH you have poor inhibition of bacteria – hmmm…

So, the canine pancreatic case that didn't need antibiotics initially (because canine anatomy V feline means former pancreatitis cases less likely to need antibiotic) now need antibiotics if you use omeprazole...?

Equally, you will now have the potential for poor Vitamin B absorption. As we all know the important role Vitamin B plays in feline IBD – **using omeprazole in an IBD case can drop the VIT B needed to control the IBD...?**

So how on earth does this make proton pump inhibitors safer, better, more effective than cimetidine...?

Cimetidine doesn't reduce the pH to neutral, so bacterial overgrowth and poor Vitamin B absorption are not such a risk, so reduce the use of antibiotics. It's cheaper, you can titrate out of it to reduce gastric rebound and you can use in poison cases. So how come my using it makes me a BAD vet...?

The medical profession 2012 advised: PPIs should not be used in advance of H_2RAs, an argument against 'step-down' therapy and other popular practices.[2]

Many human drugs we now use in our patients such as Fosamax and Clopidogrel can have serious side-effects/ interactions with PPI. Likewise, the reduction in magnesium levels and the increase in blood pressure now noted as side effects in humans using these drugs should give vets pause about being blasé prescribing PPIs in general.

I don't believe in there being 'Good Vets' or 'Bad Vets'. The sooner. as a profession, we stop feeling fit to judge our colleagues and peers by the drugs they use rather than the knowledge they use to select said drug, then the healthier and more collegial the Australian Vet Profession would be.

So, by all means use proton pump inhibitors, spironolactone etc. But give those of us who also use some good old-time medicants – in addition to the 'new best practice drugs' – the benefit of the doubt. We might actually know what we are doing in adopting recalcitrant attitudes and not just embracing the new drug on the block and throwing out the old favourites which have served our patients so well.

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PELGER-HUËT ANOMALY IN AN AUSTRALIAN SHEPHERD DOG

C&T NO. 5429

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History

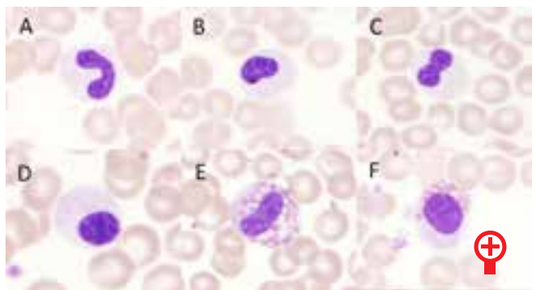
A 1-year-old entire male Australian Shepherd dog was presented for annual vaccination. The owner reported the dog to be in good health and physical examination findings were unremarkable. Blood samples were collected and submitted to the laboratory for routine screening (complete blood count and a complete biochemistry profile).

Laboratory findings

Blood film evaluation showed almost all neutrophils and eosinophils to be hyposegmented. Nuclear morphology resembled band, metamyelocyte and occasionally myelocyte forms but with mature, condensed chromatin and normal cytoplasmic features (**Figure 1**). A mild eosinophilia was present (2.1 x 10^9/L: reference interval 0.1 -1.3 x 10^9/L). No other haematologic or biochemical abnormalities were present.

The hyposegmented granulocytes were consistent with a diagnosis of Pelger-Huët anomaly.

Figure 1. ***Blood smear. Note the nuclear hyposegmentation of neutrophils (A-D) and eosinophils (E, F). Wright-Giemsa stain. 100x objective.***



Discussion

Pelger-Huët anomaly (P-HA) is an hereditary disorder of leucocyte development. It has been documented in humans, dogs, cats, horses, and rabbits, and is characterised by failure of terminal nuclear differentiation. This manifests as granulocytes and monocytes with nuclei that are hypolobulated/hyposegmented but with condensed, mature chromatin. In dogs, it has been reported in both mixed-breed and pure breeds, including Cocker Spaniels, Basenjis, Border Collies, English and American Foxhounds, Samoyeds, Australian Shepherds, Australian Cattle Dogs, Boston Terriers, German Shepherds, and Coonhounds. While it is regarded as rare in the general canine population, Latimer et. al (2000) reported an incidence of 9.8 % in a study of 842 Australian Shepherds in the United States.

P-HA is an autosomal dominant trait, with incomplete penetrance documented in Australian Shepherds in contrast to human beings and rabbits which show complete penetrance. The human and mouse mutation is in the gene encoding the lamin B receptor, which is involved in nuclear segmentation. The mutation has not been characterised in other species to date. Heterozygote animals are clinically healthy; however in homozygotes the mutation is usually lethal in utero.

Leucocytes of animals and people with P-HA function normally. The significance of recognising P-HA is to avoid misinterpretation of the leucogram as being due to severe inflammation or infection (severe left-shift), thereby avoiding unnecessary treatment and additional diagnostic testing. P-HA granulocytes maintain mature, condensed chromatin and have normal cytoplasm, in contrast to immature granulocytes seen in inflammatory states which have immature, dispersed or granular chromatin and may be associated with toxic changes (increased cytoplasmic basophilia or cytoplasmic foaminess, Dohle bodies etc). Demonstrating similar abnormalities in the sire, dam or siblings helps to confirm the diagnosis of P-HA. A genetic test for dogs is not yet available.

Following this diagnosis, the sire (an American import) and a sister of this dog were subsequently also confirmed to have P-HA. Given the high incidence of P-HA in Australian Shepherds, it is recommended that all breeding animals are screened as the potential for small litter sizes and stillbirths is high if both parents are affected.

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MAJOR WINNER
WIINER BEST FILM CLIP

THE USE OF DISTRACTING FIXATEURS TO ASSIST IN PELVIC REPAIR

C&T NO. 5430

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'Bindi' is a 5.2kg Jack Russell terrier who suffered a shattered pelvis due to a car accident. The injury was deemed to be at least 1 week old by the time of presentation.

Cage confinement for some 7 days prior to presentation had determined that she had sciatic nerve function and bladder control. Radiography identified a bilateral sacrosciatic fracture with an avulsed portion of sacrum. Her pubis and ischium were fractured and significantly comminuted. Both acetabula were intact.

She also had a 30mm left inguinal hernia to be repaired.

Figure 1: Original DV view

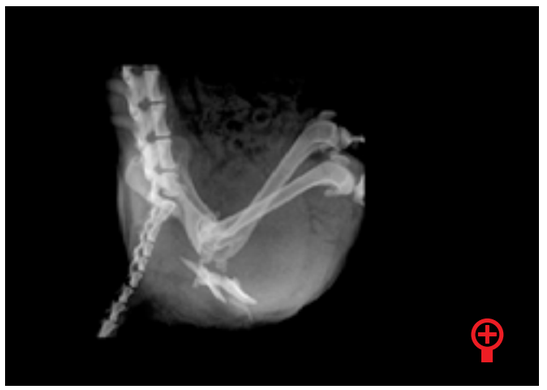


Figure 2: Original lateral view

Immediate priority was given to stabilising the sacrosciatic fractures to establish a pelvic passage and mobility. Second priority was given to hernia repair.

A bilateral dorsal approach was used to expose the articular facets of the sacrosciatic joints. This was not difficult as trauma had separated the tissues, necessitating only skin incisions over the iliac wings with the patient in ventral recumbency to allow visualisation.

The right ileum was reflected medially to expose the articular surface of the sacrum and a drill used to place a transverse drill hole across the sacrum. Visualisation of a bone specimen is a help for drill placement. Alternately, if the long axis of the sacral joint facet is visualised, running approximately ventral to dorsal, placing the drill hole a measured 4/10ths of the distance from the ventral extremity to the dorsal extremity should penetrate the sacral body centrally. If a sharp drill is used, and drilled by hand with care, any penetration of the neural canal should be felt before damage is done. (See Ref 2 for drill sharpening)

This hole was deliberately angled anteriorly so that the natural forward movement of the pinned right ileum tended to compress the joint.

A 1.6mm K-wire was drilled through an estimated corresponding position on the articular facet of the right ileum, penetrating the skin to allow a retrograde placement into the predrilled sacral hole, thus transfixing the joint.

The K-wire was similarly continued into a predrilled hole in the left ileum.

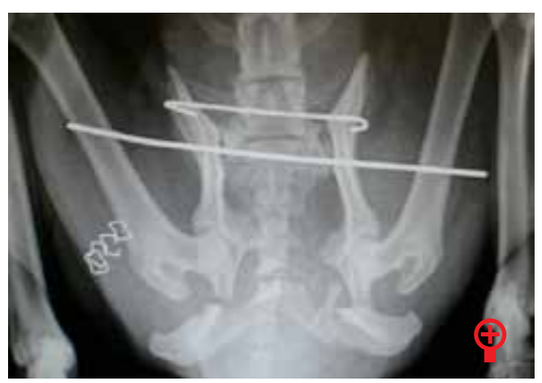


Figure 3: Initial fixation VD view

To prevent anterior movement of the left ileum the K-wire was bent caudally then withdrawn to bear on the left ileum so as to prevent joint separation.

Figure 4: Initial fixation lateral view

To ensure an open pelvic inlet a 1/6mm K-wire was placed through the wing of each ileum and clenched thus holding the wings together and the shafts apart.

The patient was walking but not able to extend the hind legs. Possible causes included pressure on the sciatic nerves or lack of leverage for the caudal leg muscles due to the anterior displacement of the pubic and ischial fragments during healing. Some two weeks post-accident it was decided to place these fragments in traction using a fixateur.

Under anaesthetic the original trans-sacral K-wire was pushed through with a longer K-wire to act as a bilateral anchor for a fixateur.

A second K-wire was placed across the wings of the ileum as a second bilateral anchor point for a fixateur. Each side was bent forward and held thus in spring tension for stability (by distraction) to allow traction hooks to be placed on the tuber ischium. The degree of traction can be estimated from the flex visible in both the threaded rods and the K-wire.

Figure 5: Traction applied to the ishial tuberosities; earlier application would have probably helped.

Figure 6: Bindi was comfortable after fixation.

Traction was increased at 1mm per day, the optimal rate determined for osteogenic distraction in dogs tibias by Ilzarov (Ref 3) and the hooks replaced as and when they tore through the bone.

Daily adjustment for traction requires no pain relief or tranquilisation.

Distracting fixateurs only require some thought as to anchor and traction points which can easily be reselected if needed. Application is only limited by the surgeon's imagination and can be easily modified.

The device was well tolerated and allowed immediate function as expected. Bindi was hospitalised for maintenance and supervision, but was not caged except at night, having the run of the hospital and a grassed yard by day. Strenuous activity was discouraged.

The fixateur was removed 8 weeks after admission and Bindi was discharged.

The cost of disposable orthopaedic material was in the order of $50.00.

A video was taken during healing and prior to discharge.

Points of the technique

Fixateurs are flexible in application and function, allowing optimal wire or pin size selection for the bone.

The elasticity of K-wire is utilised to allow it to be tensioned against itself to provide stability. In long bones the bone is in overall traction against muscles and each fragment is in traction.

Infection from contamination is unlikely to be a problem in stabilised bone, though antibiotics are used for 5 days after insertion or pin replacement. Tissue can be incised to the bone to allow wound drainage.

Penetration of muscles is never a problem.

Vaseline or Vaseline 5% chlorhexidine emulsion can be used to stop serum scalding on the skin at pin insertion sites.

Fixation of intermediate bone fragments is not required and has been shown to be counter-productive.

Holding the fractured bone extremities in their normal position is adequate for function and fast healing. Bone deficits regenerate well from the periosteum if stability is established and functionality of the damaged bone restored. Minimal iatrogenic trauma is a prerequisite for the latter. K wire insertion is virtually atraumatic.

Joints can be spanned to keep broken bones in extension, i.e. the radius and metacarpals can be used as traction points to hold a crushed carpal joint in extension and allow weight bearing. Traction gives good axial alignment and overextension causes no problems.

Preservation of the fracture clot is a distinct bonus with closed fracture reduction.

There is no need for special wound care in the yard; infection of the theoretically draining wounds seems to never occur if the K-wires are stable, i.e. kept tensioned against each other.

Figure 7. Cage rest is not usually required.

Figure 8: Small pin chuck

A small pin chuck was used with a sharp drill for sensitivity when drilling through the sacrum so that inadvertent penetration of the neural canal could be felt before damage was done. If a drill is sharpened properly it should pull itself through bone with rotation alone allowing fine control of penetration. (Ref 2)

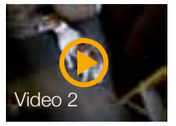
A selection of fixateurs allows an appropriate fitting for a range of patient sizes. The author is happy to supply the system and assist with application to allow others to use this simple and effective method of repairing fractures.

Note: If you would like more information, advice or assistance, Grahame is very happy to be contacted by telephone or email.

Figure 9: Any size fixateur is possible

Videos of Bindi walking.

Video 1 Video 2



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Read more articles on Fixateurs by Grahame, Bob and Alan here:

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eBOOK	Baker, G. Sep 2011, External Fixateurs: The use of fine wire external fixation to apply distraction and compression in bone repair – MAJOR WINNER, pg 22, C&T Series, Issue 264, C&T No. 5150.
eBOOK	Baker, G. Sep 2011, External Fixateurs: Distracting Fixateurs for optimal healing and distraction osteogenesis: Three cases of bone repair using traction fixateurs, pg 23, C&T Series, Issue 264, C&T No. 5151.
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VACCINE STORAGE FOR TRANSPORT: IMPORTANT INSIGHTS FOR THE VETERINARY INDUSTRY

C&T NO. 5431

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Background

The cold chain involves all the people, equipment and procedures which ensure that vaccines are maintained within safe temperature ranges, usually +2°C to +8°C at all times. The network of people, equipment and procedures that ensure that vaccine is manufactured, distributed, stored and used within the safe temperature range is called the cold chain (Nyda et al., 2001). Vaccines are delicate products whose effectiveness is dependent on their storage in specific temperature ranges. Vaccine freezing during transport has been shown in several studies to be a major cause of damage to freeze-sensitive liquid vaccines in the cold chain for human vaccines worldwide, often more problematic than warming for liquid vaccines (Techathawat et al., 2007; MoH Indonesia 2013; Muegge, 2012; Nelson et al., 2004; Chojnacky et al., 2010; Wirkas et al., 2007 and Kartoglu et al., 2009).

In the USA alone, millions of dollars' worth of vaccine for humans is lost annually due to improper storage. In resource poor settings, this level of waste is an alarming source of compromise to resources already under strain. Avoidance of vaccine damage due to temperature aberrations therefore has huge safety and cost implications (Techathawat et al., 2007). Further, vaccine effectiveness has impacts on project and community members' faith in animal health services and probably their willingness to adopt and trial other interventions in the future. It is highly likely that the situation is the same for animal vaccines.

Packs containing water (ice packs), gel, or phase change coolants are used to maintain an optimal temperature range during vaccine transport. Freeze-sensitive vaccine may be damaged if it is placed in contact with such packs taken directly from the freezer. In addition, packs containing coolants rather than water contain chemicals that lower the melting point and ensure the coolant remains colder than 0°C for longer than water ice packs. This also presents a risk of freezing. The use of 'conditioned' icepacks is recommended by the World Health Organization (WHO) to avoid freeze damage to vaccines during transport. Conditioning involves removing an icepack from the freezer and keeping it at room temperature until it reaches 0°C (Kartoglu et al., 2009). Icepack conditioning is a time-consuming process (more than 1 hour is required at 20°C) and recent surveys have shown that the practice is difficult to enforce and is widely ignored (Kartoglu et al., 2009).

This report explores a different approach to maintenance of optimal vaccine temperature conditions – frozen water ice packs wrapped in a variety of materials. Wrapping of ice packs is recommended by some human public health agencies, for example the Guidelines from the California Department of Public Health recommend that frozen packs should be covered in bubble wrap (Muegge, 2012). This set of experiments forms part of a broader manual which aims to provide a concise and practical guide to all aspects of storing and transporting veterinary vaccines, with particular focus on the thermotolerant 'wet' I-2 Newcastle disease (ND) vaccine (Alders et al., 2010) packaged in plastic dropper bottles.

Water-based ice packs, liquid chemical cool packs, and phase change coolant packs are used to maintain an optimal vaccine storage temperature range. Packs containing coolants rather than water contain chemicals that lower the melting point and ensure the coolant remains colder than 0°C for longer than water ice packs. This presents a risk of freezing unless they are appropriately conditioned, a step in the cold chain which is time consuming and often inadequately performed (Kartoglu et al., 2009). For this reason, water ice packs were used instead of ice packs containing coolant in the series of experiments described in this report.

Aims

Water-based vaccines such as the I-2 ND vaccine are susceptible to damage caused by freezing. The aim of this specific set of experiments is to determine the wrapping technique(s) that are most appropriate for storage and transport of water based vaccines for veterinary use.

The type of wrapping tested was that commonly available in the working environment of sub-Saharan Africa. These included newspaper, bubble wrap and dry and wet cloth. Water ice bricks were used because the use of gel ice bricks involves a higher risk of freezing (DHA, 2005). It has been theorised that these wrapping materials would not be able to maintain temperatures low enough for long enough for optimal vaccine storage and transport, but that the physical properties of the water in wet cloth wrapping is able to regulate temperature and maintain an optimal temperature range for longer (Schlussler, 2011), and these experiments explore this in a practical manner.

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Materials

- Polystyrene boxes. Dimensions: 16cm X 18cm X 30cm; Density: 24 grams per litre, *Medibox* by Polyfoam Australia (referred to herein as cool boxes)
- Freezer (previously determined to maintain temperatures between -15 to -22°C)
- Refrigerator (previously determined to maintain temperatures between 2 and 4.5°C)
- Ice bricks, Esky 350mL 85mm X 165mm X 35mm
- Data logger (Tinytag Plus 2 Temperature Logger with integral sensor, measuring range -40°C to +85°C with 10K NTC Thermistor probe, measuring range -40°C to +125°C)
- Newspaper, thick bubble wrap, 100% cotton tea towels
- Plastic jars containing 20mL tap water ('mock vaccine')

Methodology

As the variable tested in these experiments was the type of wrapping around the ice bricks, the equipment used (cool boxes, ice bricks, plastic jars, freezer, refrigerator, thermometers and data loggers) and location of the experiments, and were constant, i.e. all the same type and specifications. The mock vaccine and ice bricks were placed in the same position of the refrigerator and freezer respectively for all experiments. The experiments were performed at the same time of day in order to optimise consistency in ambient temperature.

Data loggers were programmed to record data from the probe and the body of the logger every 1 minute. The probes were then placed inside the 'mock vaccine', which was placed inside the refrigerator, on the bottom shelf. One data logger was placed immediately outside the refrigerator and the other two inside the refrigerator so that both the ambient temperature and the air temperature inside the refrigerator were recorded. The plastic jars, data logger probe and data logger were kept in this position for 24 hours.

Esky ice bricks were filled with 350mL tap water and placed in the freezer, on the top shelf, for 24 hours.

After 24 hours, the ice bricks were removed from the freezer, wrapped and placed in the cold boxes. The 'mock vaccine' was removed from the refrigerator immediately after and placed in the cold box. Three ice bricks surrounded each container of 'mock vaccine'. The ice bricks were either not wrapped, wrapped in 8 layers newspaper, 1-layer thick bubble wrap, 2-layers dry 100% cotton tea towel or 2-layers 100% cotton wet tea towel. When wet tea towels were used, the tea towels were wet under running tap water until they were dripping wet. The data logger probe remained inside the 'mock vaccine' and the body of the data logger was placed outside the cold box. Data were subsequently recorded for 36 hours.

Figure 1. Cold brick, Cold brick wrapped with tea towel, data logger in polystyrene box. Photo courtesy of Robyn Alders

Results

Table 1: No wrap, 8 layers Newspaper and 1 layer thick Bubble Wrap

Wrapping type	No wrap	Newspaper	Bubble wrap
Average (range) initial probe temperature, °C	3.4 (3.04 -4.01)	5.6 (4.4-6.8)	4.8 (3.8-5.3)
Average (range) minimum probe temperature, °C	-1.7 (-2.6-0.1)	-1.240 (-2.75-0.49)	-1.0 (-2.2-0.3)
Average (range) duration to minimum temperature, minutes	36 (14-48)	57.5 (52-63)	33.5 (20-47)
Average (range) time probe temp <0 °C, minutes	52 (0-88)	66.5 (0-114)	50 (23-84)
Average (range) probe temp at 15 minutes, °C	-0.2 (-0.5-0.1)	1.2 (0.8-1.6)	0.2 (-0.6-1.5)
Average (range) probe temp at 30 minutes, °C	-1.4 (-2.3-0.3)	-0.2 (-1.3-1.4)	-0.3 (-1.6-0.4)
Average (range) duration probe ≤8, hours	29.3 (27.2-30.7)	35.3 (29.5 – 40.7)	29.0 (26.4-30.8)
Average (range) duration probe ≤10, hours	30.8 (28.5-32.3)	38.0 (31.4 – 43.4)	30.9 (28.2-32.6)

Table 1 shows the average and range of results from 4 replicate experiments. In Table 1, the newspaper wrapped ice brick maintained the temperature of the mock vaccine vial lower for longer than no wrap and bubble wrap. However, none of the types of wrapping protected the 'mock vaccine' from temperatures below zero. There was considerable variation in some parameters, for example the average number of minutes the water temperature remained below zero degrees for the newspaper set of experiments ranged by 0 to 114 minutes.

Table 2 shows the average and range of results from a second set of four replicate experiments. In Table 2, temperatures above zero were maintained consistently by the ice bricks wrapped in the wet tea towel. The dry tea towel wrapping did not stop the 'mock vaccine' from reaching freezing temperatures. The wet tea towel wrapping allowed the 'mock vaccine' to be maintained between an average of 1.02°C (range 0.6 – 1.4) and 8°C for 33.8 (range 32.4 – 35.3) hours.

The ambient temperature varied between 16.0°C and 23.4°C over the period of the experiments and the average ambient temperature over the course of the experiments was 19.6 °C.

Discussion

The results of these preliminary experiments show that wrapping water filled ice packs in two layers of wet 100% cotton tea towel maintained the temperature of 'mock vaccine' close to optimal storage and transport temperatures for nearly 34 hours on average, under trial conditions. These results are likely to be significant despite the relatively large amount of variation in the results and are consistent with the outcomes described by Schlussler (2011). The ambient temperature varied between 16.0°C and 23.4°C throughout the time course of the experiments. This could have contributed

Table 2: No wrap, 2 layers Dry tea towel, 2 layers Wet tea towel

Wrapping type	No wrap	Dry tea towel	Wet tea towel
Average (range) initial probe temperature, °C	3.4 (3.03-3.8)	4.0 (3.6-4.6)	4.9 (4.3-5.8)
Average (range) minimum probe temperature, °C	-0.2 (-1.3-1.0)	-2.0 (-3.9--0.5)	1.0 (0.6-1.4)
Average (range) duration to minimum, minutes	26 (11-39)	56 (48-61)	2.3 (1.8-2.7)
Average (range) time water temp duration <0°C, minutes	28 (0-56)	104 (50-148)	0 (0-0)
Average (range) water temp at 15 minutes, °C	0.5 (0.1-1.0)	0.5 (-0.5-1.0)	3.8 (3.1- 3.7)
Average (range) water temp at 30 minutes, °C	-0.03 (-1.05-1.14)	-1.1 (-2.7--0.2)	2.2 (1.6-2.7)
Average (range) duration probe ≤8°C, hours	29.9 (27.4-32.8)	39.4 (35.7-43.5)	333.84 (32.4- 35.3)
Average (range) duration probe ≤10°C, hours	31.4 (28.9-34.5)	42.1 (37.8-46.4)	36.13 (34.2- 37.6)

to the variation in the results and future experiments should be performed in tightly temperature controlled rooms. The WHO recommends testing cold chain equipment at a constant ambient temperature of +43°C (WHO, 2012). The optimal environment to test cold chain set-ups is in the field conditions in which the vaccine will actually be used, and readers are encouraged to do so.

Users of the veterinary cold chain could adopt findings from the relatively large body of research available on the human vaccine cold chain. However, it is important to remain mindful of the differences between the human and veterinary cold chains. The veterinary cold chain is generally less well-resourced than that of the human health system, in terms of equipment and personnel. The use of cool, non-frozen water packs has been the recommended outcome of recent studies of human vaccine cold chains (MoH Indonesia, 2013) but is possibly not a suitable recommendation for the veterinary cold chain due to the poor insulation quality of cold boxes used routinely in the veterinary cold chain.

Another difference between the human and animal health sector vaccine cold chains is that vials used for animal vaccines are often multi-dose and contain larger volumes of vaccine than the single dosage vaccines used in humans, which has potential implications for temperature. It is also likely that in remote areas where refrigerators are scarce, storage capacity will be prioritised for the human cold chain over the animal cold chain, arguably placing more importance on the cold box for the animal cold chain.

There are many potential future experiments that could be performed to identify optimal storage and transport conditions for freeze and temperature sensitive vaccines. These include experimenting with different numbers of layers of wrapping, different numbers of ice bricks, different types of cold bricks, different types of cold boxes, different length of time of freezing of ice bricks, different volumes and types of 'mock vaccine', wrapping of 'mock vaccine' or actual vaccine vials instead of or as well as wrapping ice bricks.

Acknowledgements

Financial support from the Australian International Food Security Research Centre is gratefully acknowledged.

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REPLIES & COMMENTS

COMMENT ON C&T NO. 5421 'RAT BAIT' (ISSUE 276, SEPT 2014)

C&T NO. 5432
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I believe Dr Nikki Frost would have been correct with either treatment selection she made – giving fresh dog's blood to the feline patient to address the coagulopathy as well as the anaemia, or giving fresh plasma to address the coagulopathy but not the anaemia.

Red blood cell transfusion is certainly indicated for the treatment of anaemic hypoxia, but the critical haemoglobin (Hb) or haematocrit (Hct) below which all critically-ill patients require transfusion has not been established.

Anaemia seems to be well tolerated by the majority of critically-ill patients and the body will accept a marked degree of anaemia as long as the blood volume is maintained.

In anaemic states, as oxygen delivery (DO2) to the tissues lessens, the tissues ability to extract oxygen (ERO2 = oxygen extraction ratio) increases so that the oxygen uptake (VO2) remains constant until a critical level is reached where this can't continue and hypoxia with lactic acidosis, etc ensues. As DO2, ERO2 & VO2 cannot easily be measured outside the research setting, other end-points of resuscitation measurements can help us decide – systolic BP (80-100mmHg), mean arterial pressure (60-80mmHg), base deficit (-2 to +2 mEq/L), lactate (1 – 1.5 mmol/L), pH (>7.32), mixed venous oxygen saturation (SvO2 >70%), central venous oxygen saturation (ScvO2 >65%) – with the use of blood gases & blood pressure measuring devices.

There is a nice review paper (Journal of Feline Medicine & Surgery 2012; 15(2)62-67) on Xenotransfusion with canine blood in the feline species involving 62 cats which deduced that cats do not appear to have naturally-occurring antibodies against canine red blood cell antigens and no severe acute reactions were reported (in this series) in cats receiving a single canine whole blood transfusion. These anaemic cats improved clinically within hours of receiving canine blood. However, antibodies against canine red blood cells can be detected within 4-7 days of the transfusion, leading to destruction of the transfused canine red cells in a delayed haemolytic reaction – the average lifespan of transfused canine red blood cells is less than 4 days. Any repeated infusion with canine blood after 4-6 days following the first transfusion causes anaphylaxis which is frequently fatal.

Hence if the kitty is coping with its anaemia and is normotensive, then maintaining blood volume & addressing the coagulopathy with plasma would seem prudent, the kitty then manufactures its own red cells once blood loss is halted – in this case, the cat was able to increase its haematocrit from 11% to 39% in 10 days.

However, if red cells were deemed necessary (hypoxic anaemia – hypotension), then a one-off xenotransfusion of the cat with dog's blood would be likely to save the cat's life with low likelihood of severe adverse reactions.

REPLIES & COMMENTS

REPLY TO C&T NO. 5409 'ANISOCORIA & NYSTAGMUS IN A CAT' (ISSUE 276, SEPT 2014)

C&T NO. 5433
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Re C&T 5409, I have just had exactly the same situation happen with my own cat who is a 3yo DSH. It started with my observing a 5mm abscess on top of her head (she is indoor/outdoor and often gets into scraps with other cats and possums, plus bringing in the odd rat). I elected not to treat this as it appeared to be healing. A few days later she began walking tentatively and had difficulty jumping and then progressed to inappetence, anisocoria, nystagmus and what appeared to be a Schiff-Sherrington Phenomenon-like reaction.

I, of course, was overseas as this was occurring so my daughter placed her in the care of my colleagues. She was placed on a drip, given IV Solu-Delta-Cortef® and IV Timentin®. She improved remarkably the next day but still had anisocoria and inappetence. We started her on clindamycin 75mg and IV Valium® to encourage eating. Bloods showed an inflammatory rx, FeLV/FIV testing was negative. Toxoplasma testing was also negative. We tried to get X-rays of her head and chest, but by this stage she had become feisty and we could not get an IV into her. We continued for at least 2 weeks giving mirtazapine to encourage eating. Improvement was slow but steady.

We came to the conclusion that she had had a middle/inner ear infection which had resolved with the clindamycin, most likely related to that abscess I didn't treat.

My take home from this was to treat every cat bite abscess, regardless of how small they appeared to be!

FURTHER COMMENT ON MY C&T NO. 5410 CLOPIDOGREL (ISSUE 276, SEPT 2014)

C&T NO. 5434
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In regards to a C&T reader's query about my recent C&T No. 5410, specifically asking if the dose mentioned, 10-20 mg/kg, was correct or whether it should have said per cat, I reiterate that the dose was **correctly stated**. 10-20 mg/kg daily is the recommended dose as per Hogan et al (2004) referenced in the C&T article.

I use 75mg tablets, and dose at 1/8 - 1/4 tablet once or twice weekly long-term.

In regards to Richard Malik's query as to why I preferred once or twice weekly long-term instead of daily, the answer is that there doesn't appear to be any long-term usage studies on cats at present regarding safety.

I am sure someone will eventually research a safe dose for long-term therapy, but until then, I tend to err on the safe side.

Editor's Note: Most people we have contacted use ¼ tablet per average cat once daily.

DIAGNOSTIC IMAGING

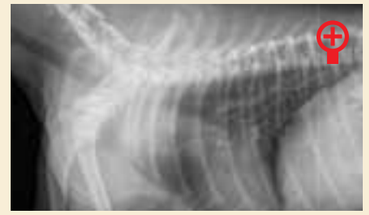
The CVE is keen to encourage our Members/Readers to embrace the eBook version of the quarterly C&T, as it's a great complement to the print version and allows the inclusion of multi-media. To encourage reading of our C&T eBook, Robert and Graeme, tutors for the CVE's highly regarded Diagnostic Imaging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

If you have forgotten your Username and Password please email cve.enquiries@sydney.edu.au or call (02) 9351 7979.

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

WHAT'S YOUR DIAGNOSIS?

C&T No. 5435



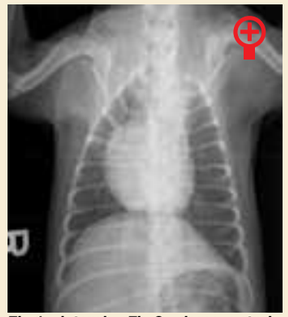


Fig 1 – lateral *Fig 2 - dorsoventral*

Signalment: 1 year old Pug.

History: Owner reports that dog appeared to be choking and has since vomited twice.

Views: Neck, thorax and cranial abdomen – right lateral and dorsoventral.

QUESTIONS

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

Solution including 4 images available in the eBook

WHAT'S YOUR DIAGNOSIS?

C&T No. 5436
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This case presented to me about 15 months ago at my previous employer, Karingal Veterinary Hospital. A 7yr FS DSH cat presented for a slow growing (about 6 months) lump on her back left leg and a problem with her left eye. This cat has a history of being in cat fights.

On examination the left eye seemed to have swollen conjunctiva at the lateral canthus and between the lower eyelid and 3rd eyelid. On closer examination it appeared to be lymphoid tissue (photos attached).

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

Figure 2. Swollen conjunctiva lower eyelid near third eyelid.

REPLY TO POSSIBLE ALFAXAN REACTION (C&T NO. 5422, SEPT 2014)

C&T NO. 5437

Mark Boyhan

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Note: These replies and comments were supplied independently i.e. Mark's arrived unsolicited and CVE invited Christina and Sanaa to provide Invited Commentary.

1 2 3	C&T No. 5422 Possible Alfaxan Reaction (Sept 2014)

I read the C&T No. 5422 Possible Alfaxan Reaction by Dr Geoff Hayres, along with the replies by Dr Malik and the Jurox team. I thought that both replies were well reasoned and very measured in answer to the concerns raised by Dr Hayres.

In my opinion the medetomidine dose of 100ug for a 1.3kg cat is excessive as it works out to be 77µg/kg. The suggested premed dose rate for medetomidine in the 2nd edition of the BSAVA Manual of Canine and Feline Anesthesia is 10-20ug/kg IM or IV for cats and dogs. Dr Richard Bednarski in Chapter 26 of the 4th Edition of Lumb and Jones Veterinary Anesthesia and Analgesia suggests a dose rate of 4-6ug/kg for cats as a premed when combined with an opioid. Dr Polly Taylor, in her article on anaesthesia for kitten desexing on the old Feline Advisory Bureau website, warns against using high dose medetomidine protocols for desexing kittens less than 16 weeks of age and is more comfortable with doses around 10ug/kg. Dr Jeff Ko from Purdue University also discussed doses of medetomidine around 20ug/kg for cats, except when combined with ketamine.

The only induction protocols I have seen using medetomidine doses in the 60-80ug/kg dose range have been where medetomidine has been combined with ketamine, which has potent cardiovascular stimulating effects. Dr Hayres used 3 drugs all with recognised potential for cardiovascular suppression. The medetomidine dose was nearly 4 times the widely accepted dose of 20ug/kg as outlined above.

Most likely cause of near death experience would have to be left ventricular failure due to anaesthetic overdose in my opinion, which is consistent with what Dr Malik suggested.

The information in the table below was extracted from the following anaesthesia references.

Medetomidine Dose For Cat Premedication	Reference
4-6ug/kg IM*	Bednarski (2007)
5-10ug/kg IV	Gaynor & Muir (2009a)
20ug/kg SC*	Gaynor & Muir (2009b)
10-20ug/kg IM* 5-10ug/kg IV*	Lamont, L. (2009)
5-20ug/kg IM*	Lamont & Matthews (2007)
10-20ug/kg IM or IV*	Murrell (2007)
2-10ug/kg IM*	Pawson, P. (2008)
5ug/kg IM*	Robertson, S.A. (2009)

*In combination with an opioid analgesic

The only published work I have seen using High Dose medetomidine (100ug/kg) in conjunction with Alphaxalone/Alphadolone, presumably Saffan was in:

Short, C.E. (1992) Chapter 4. Alpha-2 Adrenergic Agonists/Antagonists in Cats. In: Short, C.E. (ed.) Alpha2-Agents in Animals – Sedation, analgesia and anaesthesia. *Veterinary Practice* Publishing Company: Santa Barbara, California.

This is from some very early experimental work in mature laboratory cats. I remember when Dr Charles came out to Australia in about 1993/94 when Domitor® was first released by Ciba-Geigy/Novartis, at the product launch he was very comfortable with the dose rates of Domitor® (80ug/kg IM) in combination with Ketamine (5mg/kg) in cats, but said that more work needed to be done with other combinations at that time. The information he presented for dogs was more comprehensive as they had completed more trials with dogs at that point. This is also reflected in Chapter 3: Alpha-2 Adrenergic Agonists/Antagonists in Dogs from the same book. The more contemporary references above suggest far lower doses.

I use medetomidine quite often and the CEPSAF study in the UK by Dr David Broadbelt showed that at clinically appropriate doses in the right cases it decreases the incidence of perioperative mortality to the same extent as Acepromazine does. I guess the important part is 'clinically appropriate doses'. Dr Sheilah Robertson at the University of Florida bases much of her approach to dog and cat anaesthesia on this study. I also think that such a heavy handed approach with medetomidine in an immature desexing case is ill advised. Although Dr Geoff doesn't tell us the age of the cat, the bodyweight is consistent with being under 4 months, and possibly quite considerably so.

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Comment from Richard Malik, Co-Veterinary Editor

Thank you Mark for taking the time to submit a reply, which is what the C&T Series is all about – stimulating constructive criticism. I did some calculations at the time I read Geoff's draft but obviously didn't do them as carefully as you did. I personally don't use Alpha-2 agonists, and even though the Europeans seem to like them and find them safe, I prefer to keep away from them.

COMMENT ON POSSIBLE ALFAXAN REACTION (C&T NO. 5422, SEPT 2014)

C&T NO. 5438

INVITED COMMENTARY COURTESY OF:

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I was asked to provide comments – additional to those provided by Richard Malik, Stephen Page and the professional group from Jurox – on the report of an adverse reaction to Alfaxan in a kitten. I presume my role is to provide an expert judgement. Expert judgement is a common and popular method for assessing causality of adverse events, the determination whether there is a reasonable possibility that a product is casually related to an adverse event. Expert judgement is not the most reliable method for causality assessment for there is room for subjectivity, preconceived opinions, variability in expert knowledge, experience and skills, and lack of standardised clinical evaluation of case reports. Algorithms have been developed to at least provide guidance to assessing of case reports. The WHO-UMC developed a system which provides a practical tool for case report assessment and which I would like to apply to this case. The WHO-UMC method groups causality into 6 categories ranging from certain to unassessable/unclassifiable. For each category assessment criteria are given. The case report in question complies with the following criteria:

A) Event (or laboratory test abnormality), with reasonable time relationship) to drug intake

The report does describe an adverse event, namely pulmonary oedema diagnosed on the basis of presence of excessive fluid in the respiratory tract. Although not specifically reported the time interval between the administration of Alfaxan and first recognition of the adverse reaction can be assumed to be reasonable.

B) Event could also be explained by disease or other drugs

Should Alfaxan in fact have caused pulmonary oedema it would be reasonable to assume that the pathogenesis was altered pulmonary vascular permeability. However pulmonary oedema in the kitten could also be explained by haemodynamic abnormalities. Relatively obvious haemodynamic abnormalities would include pre-existing heart disease – as suggested by Richard Malik – and α-2 agonist induced haemodynamic changes. The concurrent administration of sedatives and anaesthetics with a variety of effects on cardiovascular function could have initiated decompensation of pre-existing abnormal cardiac function resulting in pulmonary oedema. α-2 adrenergic agonists alone have been implicated for causing pulmonary oedema. In sheep, vasoconstriction resulting in increased pulmonary vascular pressure is the initiating cause of α-2 agonist induced pulmonary oedema. There are anecdotal reports of medetomidine causing pulmonary oedema in healthy dogs. In both dogs and cats medetomidine has been shown to have minimal direct effect on pulmonary vascular resistance/pressure. Rather pulmonary vascular pressure may be indirectly affected as medetomidine increases left ventricular preload. The combination of medetomidine induced bradycardia and increase in afterload (peripheral vasoconstriction) has been suggested as an explanation for the increase in preload. In isoflurane anaesthetised cats IV medetomidine caused similar changes. In contrast to studies in sheep, pulmonary oedema with α-2 agonists and in particular with medetomidine has not been observed in studies in dogs and cats. It is noteworthy, however, that medetomidine doses used in the studies which revealed above information were considerably lower than the dose used in the kitten. Haemodynamic effects of α-2 agonists are dose related. Because of the high dose of medetomidine given to this kitten, haemodynamic changes may have been enhanced resulting in blood stasis in the pulmonary capillaries and as such may have caused pulmonary oedema. Cardiopulmonary effects and in particular effects on pulmonary vascular resistance/pressure of high doses of medetomidine in dogs and cats are not known.

Less likely causes of excessive fluids in the respiratory tract may include respiratory irritants such as residual cleaning agents on the endotracheal tube and in the breathing circuit, and the age of the patient. Although not mentioned specifically the weight of the kitten would indicate she was a paediatric patient. Relatively poor compliance of the immature myocardium could have made pulmonary oedema in this patient a more likely consequence of the medetomidine induced haemodynamic changes, in particular the increase in preload.

C) Information on drug withdrawal may be lacking or unclear

The information provided on the course of recovery in the kitten is limited in particular with regards to recovery from pulmonary oedema which is at the centre of the adverse event. Regardless and due to the concurrent administration not only of drugs to produce sedation and anaesthesia but also drugs administered in response to the seemingly life threatening event it remains unclear if and to what extent the resolution of pulmonary oedema was related to the eventual elimination of Alfaxan.

According to the above applied WHO-UMC system if an adverse drug response report, such as the one in question, complies with the above criteria drug causality is **possible.** It can therefore be concluded that Alfaxan is one of other causes for pulmonary oedema in this kitten.

Reports on cases of adverse drug reactions are fundamental for pharmacovigilance and as such for optimising drug safety. Dr Geoff Hayres should be commended on reporting the adverse event in the kitten, a process which takes time and effort and therefore is often forgone.

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COMMENT ON POSSIBLE ALFAXAN REACTION (C&T NO. 5422, SEPT 2014)

C&T NO. 5439

INVITED COMMENTARY COURTESY OF:
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There is often unwillingness by veterinarians to openly discuss anaesthetic complications and emergencies such as that described by Dr Geoff Hayres, especially when it results in the death of a patient or it is unclear what may have contributed to the incident. I would like to commend Geoff for successfully resuscitating the kitten, and thank him for his willingness to share this experience and the CVE for providing a safe forum in which discussions such as this can be had.

We will never know why this young healthy cat developed what appears to be acute pulmonary oedema following induction of anaesthesia. However, there are a number of factors that may have directly contributed to this incident, or at the very least increased the likelihood of such an event. Careful consideration of these factors can help all of us to adopt safer anaesthetic practices that both reduce the occurrence of such events and improve the outcomes when they do occur.

1. What signs did the animal display and is this consistent with an adverse drug reaction?
2. What type of reactions can be caused by anaesthetic drugs, what is the mechanism and how common are these reactions?
3. What are the cardiovascular effects of the anaesthetic drugs used and could this simply be an overdose?
4. Could the cat have had undiagnosed disease?
5. What is the most effective way to manage the symptoms displayed by this cat?

Anaphylaxis and anaphylactoid reactions

Every sedative, analgesic and anaesthetic agent we administer has the potential to cause a fatal anaphylactic-type reaction; however, they are extremely rare. These reactions often involve the release of histamine, although the underlying mechanisms for such reactions differ between drugs and individuals.

The reaction can be a direct drug-receptor interaction that is independent of the immune system, such as can occur when pethidine is administered intravenously. Alternatively, administration of any drug can result in an acute immunological response (a hypersensitivity reaction), and again the response is primarily histamine-mediated. An example of this is reactions to particular IV antibiotics and colloids.

Classic allergic reactions (anaphylaxis) usually involve the production of IgE in response to exposure of a drug. First time exposure (as is likely in this case) results in a minor reaction that is often asymptomatic. Subsequent exposures may result in severe cardiovascular collapse and bronchoconstriction that is mediated via histamine, released from mast cells following the binding of previously formed IgE to mast cell and basophil membrane receptors.

First exposure reactions that are severe are sometimes termed 'non-allergic' or anaphylactoid. The mechanisms behind such reactions are not well understood and probably involve a number of different pathways including direct histamine release and complement-mediated processes. Cremophor (the previous solvent combined with alfaxalone) is one example of an agent that causes profound histamine release mediated via activation of the complement cascade.

Regardless of whether the reaction is allergic or non-allergic, the response can be severe and is usually characterised by:

- acute sudden onset
- involvement of skin and mucous membranes (erythema, swelling)
- respiratory compromise (dyspnoea, bronchoconstriction, hypoxaemia)
- hypotension (with or without bradycardia)

Acute pulmonary oedema (as observed in this case) is not common although cases have been reported in humans.

Based on what Geoff described (young cat and first time exposure), it is unlikely that this was an anaphylactic reaction. An anaphylactoid reaction is also less likely, based on the clinical signs that were observed; although it cannot be ruled out since any one of the drugs that were administered (medetomidine, alfaxan, or isoflurane) has the potential to cause an anaphylactoid reaction.

Anaesthetic overdose

The cardiovascular and respiratory effects of medetomidine, alfaxan and isoflurane are well described in both cats and dogs. However, less is known about the effects of these drugs in very young cats, in particular when these drugs are used in combination.

Medetomidine is a synthetic α_2-adrenoceptor agonist marketed for its rapid onset of action, analgesic and sedative effects, its enhanced α_2-to-α_1 binding specificity and convenient reversibility. The following cardiovascular effects were observed in healthy adult cats (2-7 years) administered 20µg/kg IM.

15 minutes after administration:

- HR decreased to 58% of baseline
- Systemic vascular resistance increased
- Systolic, mean and diastolic arterial blood pressure was unchanged
- Cardiac index decreased to 37% of baseline
- Stroke index decreased to 65% of baseline
- Central venous pressure increased
- Left ventricular stroke work index decreased to 61% of baseline
- Right ventricular stroke work index decreased to 43% of baseline
- Pulmonary vascular resistance increased

Translated, this is a massive decrease in cardiac function, yet if all you are measuring is BP, it would look fine. This dose also resulted in reliable sedation, analgesia and muscle relaxation.

Alfaxalone is a neurosteroid anaesthetic solubilised in a beta-cyclodextrin and marketed as Alfaxan®.

The following cardiovascular effects were observed in healthy adult cats administered 5mg/kg Alfaxan IV slowly over 60 seconds. At 1 and 5 minutes after administration:

- HR decreased slightly
- Systolic, mean and diastolic arterial blood pressure decreased slightly
- Pulmonary arterial pressure was unchanged
- Cardiac output decreased slightly
- Systemic vascular resistance increased slightly

However, at this dose all of these parameters were still within the acceptable normal range. At this dose anaesthesia was achieved and endotracheal intubation was possible.

Summary

In this incident, the cat was very young and received 76µg/kg of medetomidine IM and 2.3mg/kg alfaxan IV. The administration of isoflurane appears to coincide with when the cat deteriorated which does not rule out isoflurane as the cause of an adverse drug reaction. However, it is unlikely that the cat inhaled sufficient isoflurane before resuscitation was commenced for it to have caused any direct cardiovascular effects that may have contributed to the emergency.

The cat was healthy; however, we cannot rule out any sub-clinical co-morbidities.

The combined effect of medetomidine and Alfaxan administered at these dose rates in young healthy cats has not been reported, however based on their pharmacodynamic profiles as summarised above, it is feasible that this cat experienced an inadvertent anaesthetic overdose following the administration of this drug combination that resulted in acute left-sided heart failure.

The reason I propose this is not to be in agreement with Richard Malik (it's much more fun to argue with him), but rather to highlight another valid and potentially more likely cause, based on the information given by Geoff.

It is also important to emphasise that the risk of anaesthetic overdose is much higher than the risk of an adverse drug reaction, simply because both severe anaphylactic and anaphylactoid reactions secondary to drug administration are extremely rare in our domestic species, except in drugs that are known to directly cause release of histamine. Alfaxalone, medetomidine and isoflurane are not reported to cause histamine release.

Management

Regardless of what caused this event, Geoff Hayres did a great job of resuscitating this cat. I want to highlight the key strategies that I believe made all the difference.

- Immediate reversal of the reversible drugs (i.e. medetomidine) and discontinuation of anaesthesia. Although it was some time after administration of atipamezole that the cat started to recover, it must be remembered that this cat was in shock, and so any drugs administered will have a delayed response.
- Oxygen supplementation
- Administration of adrenaline (indicated for management of anaphylaxis, bronchoconstriction and cardiovascular resuscitation)
- Manual removal of excess fluid obstructing the larger airways (if suction is not available, use gravity)
- Administration of frusemide and dexamethasone (to manage pulmonary oedema +/- airway inflammation of unknown origin)

Geoff's decision to not use medetomidine or alfaxan for subsequent anaesthesia I think is also sensible. Firstly, we can never completely rule out that this was not an adverse drug reaction and either drug could have caused it. Secondly, acepromazine does cause vasodilation; however, its overall effect on cardiovascular function (especially cardiac output) is much less than that of medetomidine, especially when medetomidine is used in high doses.

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I have attempted to provide an evidence-based response, and hope that it has added positively to the discussion. Thank you CVE for giving me the opportunity to comment on this C&T.

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WINNER OF BEST IMAGES

WHAT'S YOUR DIAGNOSIS?

C&T No. 5440
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'Stanley' is an 11-year-old male neutered Chinchilla cat. Stanley visits once or twice a year to have a general anaesthetic and have the thick mats shaved from his coat. He also usually leaves one of us with bloody track marks along our arms as he is a very fractious cat.

Yesterday, he was premedicated with acepromazine (0.03mg/kg) and methadone (0.15mg/kg) subcutaneously. We were able to put the stethoscope on his chest and his heart sounded OK, no murmurs or arrhythmias. Induction was ketamine (4.5mg/kg) and diazepam (0.23mg/kg) intravenously. Loud gurgly noises were emanating from caudal oral cavity, and when he was intubated copious amounts of haemorrhagic froth filled the ET tube. HR dropped to 140/bpm, was in respiratory arrest and we ventilated him. We had to keep suctioning the bloody froth from his ET tube. SpO_2 was initially 60% and over 20 minutes climbed to 90%. Loud crackles auscultated over lung fields. Gave frusemide 4mg/kg IV. His HR went to 220-240/m. He was eventually extubated, then kept on oxygen (100%) by mask then intranasal O_2.

Whilst he was still very sedated, we were able to get thoracic radiographs and do a very brief echo. I think the radiographs and echo are consistent with HCM and congestive heart failure, but there are some aspects of this that are troubling to me.

I'm confident the pulmonary changes represent pulmonary oedema, and there was also a mild pleural effusion. There is unequivocal improvement after frusemide administration (Figure 3). His cardiac silhouette seems on the small side if anything, which initially had me wondering about spurious echo measurements but he was definitely not dehydrated.

The copious haemorrhagic froth expectorated via the endotracheal tube makes me think that the cat was already in congestive heart failure. It seems inconceivable to me that the clinical and radiographic signs could appear within minutes of administration of the aforementioned drugs. The owners (anaesthetist and theatre nurse respectively) had not noted any problems at home and he certainly did not appear tachypnoeic/dyspnoeic prior to premed/anaesthesia.

I also would have thought with pulmonary oedema of this severity (assuming it is pulmonary oedema) associated with HCM, that there would be some degree of left atrial enlargement. Unfortunately, during the echo Stanley started to become aroused and stressed and we had to stop before we measured the left atrium to aortic root ratio (and other) measurements.

Stanley's right heart also appears enlarged on echo (right atrium and right ventricular internal ventricular dimension). I have not seen a heartworm case (dog or cat) in our area for many years but he had not had an antigen or antibody test.

	video

Please send your replies and comments to: Elisabeth.churchward@sydney.edu.au for publication in our December 2014 issue.

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See www.vetbookshop.com to peruse our lists of titles.

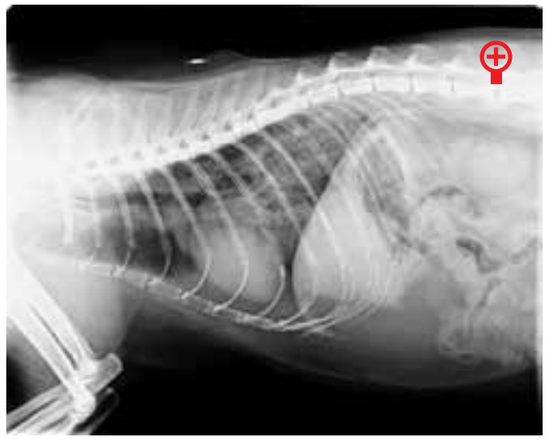

Figure 1. Lateral thoracic radiograph at the time the cat 'crashed'.

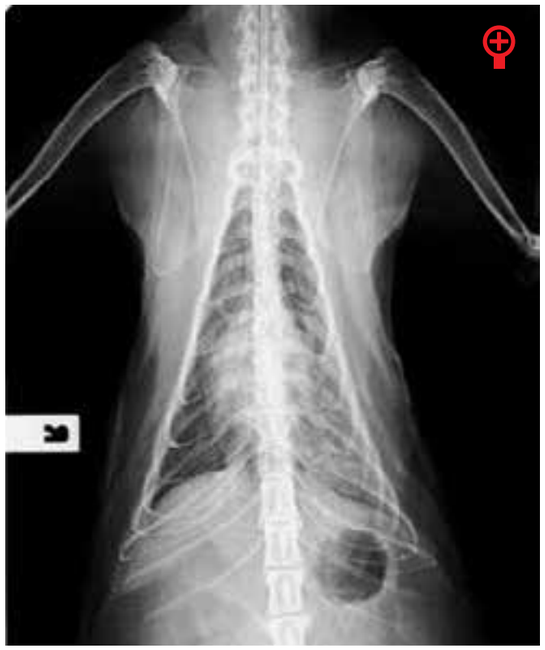

Figure 2. Dorsoventral thoracic radiograph at the time the cat 'crashed'.

From the ISFM Forum –
CVE's Feline Distance Education partner

ANSWER TO WHAT'S YOUR DIAGNOSIS?

(C&T NO. 5397, JUNE 2014, ISSUE 275)

C&T NO. 5441

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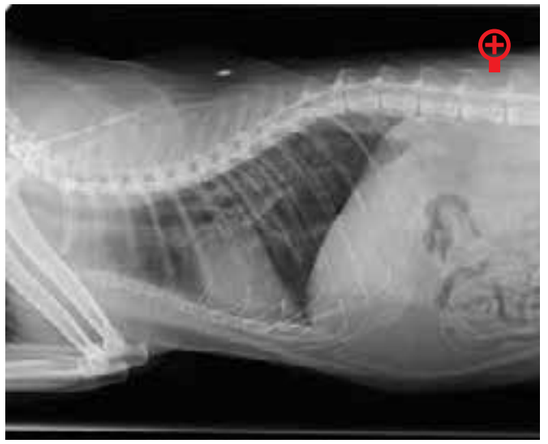

Figure 3. Lateral thoracic radiograph 3 hours after administration of frusemide.

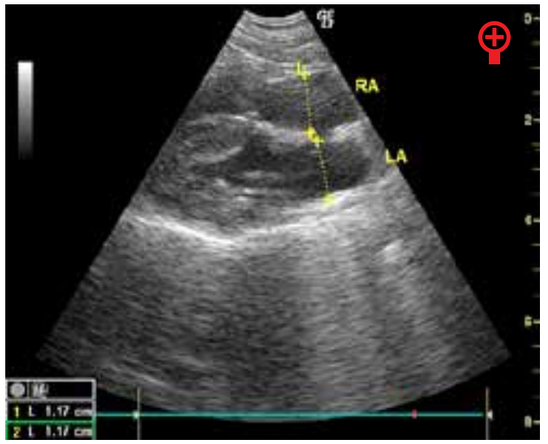

Figure 4. Right parasternal long axis view optimis

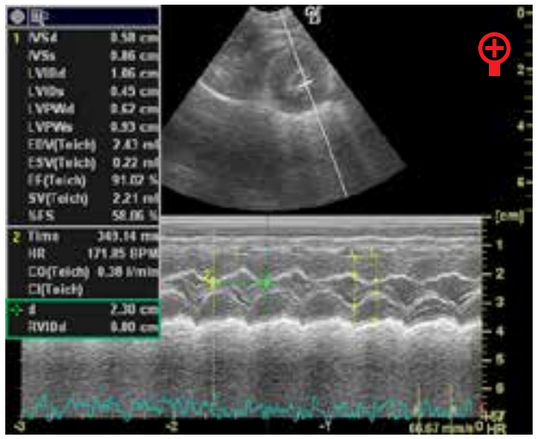

Figure 5. M-mode study showing symmetrical thickening of the left ventricular free wall and the sternum

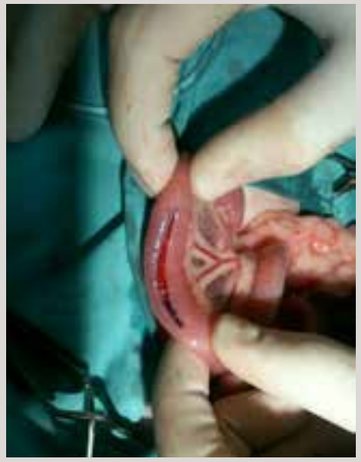

Figure 1. Bella's bowel.

'Bella' was 14-years-old and presented to me in early 2013 with a history of acute onset vomiting and abdominal pain. She was treated symptomatically (buprenorphine/maropitant/fluids/antacids/antibiotics) and recovered her appetite and was discharged eating well after 3 days.

She presented again 2 weeks later with inappetence; this time there was a history of being caught in a door.

Her radiographs showed peritonitis so an exploratory laparotomy was performed (see Figure 1) but no biopsies sadly due to the history of trauma.

Bella recovered well after surgery to suture that 'tear' and remained well for about a month. She then presented with intermittent vomiting and diarrhoea which was uncontrolled by medication but waxed and waned for approximately 2 months. Then she began to lose weight. At that point we decided to trial a combination of prednisalone and amoxicillin-clavulanate treatment on the basis that she may have had an inflammatory bowel disease or diffuse small cell lymphoma. Sadly, she did not respond to steroid therapy nor to a combination of prednisalone and chlorambucil. A post mortem was performed and histopathology revealed intestinal lymphoma. In hindsight, a biopsy at the first surgery would have been a good idea.

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PERSPECTIVE 110

GREY MATTER – PERSPECTIVES ON SMALL ANIMAL IMAGING

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Zoe attended the University of Sydney and obtained a Bachelor of Veterinary Science in 1999. She worked in small animal practice in inner Sydney for 4 years, before moving to Perth, Western Australia.

At Murdoch University, Zoe undertook a residency in Diagnostic Imaging (2003-2006) and became a Fellow of the Australian and New Zealand College of Veterinary Scientists in Radiology in 2007. Zoe is a Director of the Veterinary Imaging Centre and Perth Veterinary Specialists.

WHEN TWO RADIOGRAPHIC PROJECTIONS WOULD HAVE GIVEN YOU THE ANSWER!

An 11-month-old Male entire German Shepherd dog presented with a 4 week history of left forelimb lameness after running into a stationary object. The dog initially responded to non-steroidal anti-inflammatories but lameness of the left forelimb persisted. The lameness was localised to the shoulder joint and radiographs (a single lateral projection) were obtained at the referring practice. The initial radiographs are no longer available, but showed pathology that was highly suggestive of an aggressive bone lesion in the distal aspect of the left scapula (see figure 1), including apparent cortical lysis, a stippled appearance created by poorly organised sclerosis and disorderly periosteal reaction along the cranial and caudal margins of the distal scapula.

Given the dog's youth, breed and geographic location in Perth WA, a differential of fungal bone disease (e.g. Aspergillus spp.) was considered highly likely, with primary bone tumour (e.g. osteosarcoma) unable to be ruled out. Both diseases potentially have a guarded prognosis and may lead to forelimb amputation. A complete abdominal ultrasound was performed (which was unremarkable) and urine was collected via cystocentesis for fungal culture (which was negative). Fine needle aspirates of the distal scapular lesion were obtained using ultrasound guidance but were inconclusive.

The owner was presented with options of:

- guided biopsy of the lesion
- more imaging (e.g. obtaining a CT of the thorax for staging, including the scapulae bilaterally, or less optimally, a repeat radiographic study with two orthogonal views to check for progression) in an attempt to characterise the extent of the disease further, or
- amputation and subsequent histopathology.

Two weeks later the dog returned for an ultrasound guided, tru-cut biopsy, in order to attempt to characterise the nature of the disease further. With ultrasound, the abnormal bone at the supraglenoid tubercle of the scapulae was easily identified and biopsied. Interestingly, at this visit, the dog was no longer lame in the left fore limb at all, which is highly unusual with both neoplastic and fungal bone disease.

Histopathology diagnosed calcinosis circumscripta (syn. canine tumoural calcinosis) – a benign condition characterised by local deposition of mineralised salts in tissues, surrounded by a fibrous response (Scott and Beurger). These lesions are common in juvenile German Shepherd dogs, less than 2 years of age and typically occur over bony prominences. Whilst they are often located in the around joints in the peripheral limbs, they are reported to occur in association with the shoulder joint (Roudebush et al). They are local and well circumscribed. At this point, interest piqued, a complete radiographic study was obtained and the craniocaudal projection of the shoulder joint shows the typical, stippled appearance of the lesion in the soft tissues medial to the distal aspect of the scapula (Figure 2). At a revisit 3 weeks after biopsy, the dog was barely lame. The owners elected not to proceed with excision of the lesion, given the resolution of clinical signs. No evidence of other calcinosis lesions were detected on physical examination in this dog. It is presumed that the initial lameness may have been caused by the lesion (e.g. compression on the medial aspect of the shoulder joint) but equally so, other causes of lameness (like sprain of soft tissues secondary to collision) could not be excluded.

This case eloquently demonstrates why two orthogonal (at right angles to each other) are ALWAYS indicated in radiographic assessment. Even when we look at simple structures (like the shoulder joint) with radiography, one projection does not allow you to take into account the effect of superimposition of structures over your area of interest. Practitioners are well aware of this principle in thoracic and abdominal radiography, where you nearly always need two orthogonal projections for a diagnostic study. There can be a tendency in looking at bones to want to skip this step, especially with a lesion like this. Practitioners in primary and referral practice need to remember not to skip steps, even when we think the answer is obvious! Retrospectively, the working diagnosis of an aggressive bone lesion proximally in the limb did not fit with the clinical signs of a minimally lame dog. This dog had a protracted work up (involving abdominal ultrasound, needle aspirates, urinary and bone culture) before the correct diagnosis was made. Arguably the outcome may have been worse if forelimb amputation had been performed, as it was the dog returned to normal function. Plain, old-fashioned radiographs would have likely led to a more rapid accurate diagnosis if a complete study had been obtained in the first instance.

Acknowledgements:

Jessica Finlay (Resident in Medical Oncology), Mika Frances (Resident in Radiology), Devon Thompson (Veterinary Radiologist), John Jardine (Pathologist)

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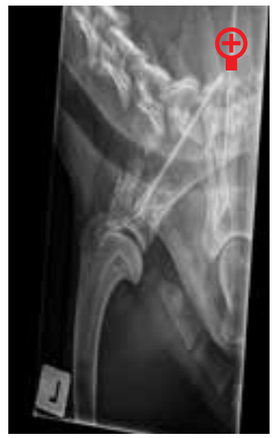

Figure 1: lateral projection of the left shoulder joint. Surrounding the distal third of the scapula is a large and poorly defined region of apparent bony lysis, sclerosis and irregular periosteal reaction. The lesion appears confined to the scapula (monostotic) and does not appear to cross the joint space. (Note, this study was obtained after diagnosis). A radiographic diagnosis of aggressive bone disease was made, based on this projection.

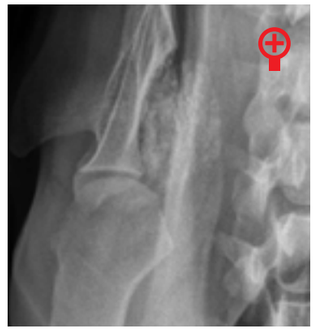

Figure 2: Craniocaudal projection of the left shoulder joint. A focal, well defined and stippled appearing mineralised aggregate is present in the soft tissues medial to the distal aspect of the scapula consistent with calcinosis circumscripta. The scapular is separate from the lesion and no evidence of cortical lysis is present. This projection underscores the importance of getting a proper, two-view projection of the joint in order to minimise the effect of superimposition of structures.

PERSPECTIVE 111

RIGID ENDOSCOPY

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After graduating from Colorado State University with a BSc in biological sciences/anatomy and neurophysiology, Elise relocated to the UK in 1998 to study veterinary medicine at the Royal Veterinary College, University of London. She graduated with honours in 2003 and instead of moving back to the USA, she ended up staying permanently in the UK! She focused her clinical work and studies on feline medicine and was awarded Membership of the ANZCVSc in feline medicine in 2008, and later Diplomate status of the American Board of Veterinary Practitioners (ABVP) in 2012. She operates a visiting feline referral and endoscopy service for those in first opinion practice in SE England. Elise's professional interests include medical endoscopy and endosurgery (laparoscopy). She's received her formal training from both veterinary and human consultant surgeons, in the USA, Germany, France and the UK. In her spare time Elise enjoys playing the cello, travelling, running events (10K and half marathons) and triathlon and her new love is now Ironman 70.3.

Abstract

Most veterinary practitioners will have at least some vague familiarity with flexible diagnostic endoscopy, and its use in diagnosing alimentary and respiratory diseases. Rigid endoscopes, on the other hand, can also be widely used in both diagnostic and surgical interventions. These endoscopes are often preferred for use in non-tubular structures due to better manoeuvreability and superior optical images compared to traditional fibre-optic flexible endoscopes. Up until now, these 'high-tech' procedures had been reserved for those practicing in veterinary teaching hospitals and private referral institutions. One of the most exciting of these procedures is the implementation of rigid diagnostic and interventional endoscopy/endosurgery into our everyday caseload.

Introduction to Rigid Endoscopy

The decision to incorporate endoscopy into small animal practice can be complicated and should take into account the financial viability of supporting this type of service within the veterinary business. It will depend on practice demographics, staffing, practitioner interest, investment in training, and relative proximity to practices offering similar services. A committed practitioner can easily learn basic endoscopic techniques; however, the novice endoscopist should strongly consider participating in hands-on wet lab courses provided by experienced endoscopists to rapidly achieve a level of competence that justifies the high initial investment in providing this type of service.

This article is intended to briefly provide the general practitioner with an introduction to the rigid endoscope, imaging/accessory equipment, and a few basic 'oscopies' that can be performed in practice.

Procedures that can be performed using rigid endoscopy include:

1. Rhinoscopy
2. Urethrocystoscopy/Vaginoscopy
3. Otoendoscopy
4. Laryngoscopy + Tracheoscopy
5. Laparoscopy
6. Arthroscopy
7. Thoracoscopy

What is a Rigid Endoscope?

In simple terms, a rigid endoscope is a long slender stainless steel tube with a series of solid glass rod lenses which allow for the transmission of light and image (Figure 1). Light transmission is achieved from the use of an extracorporeal light source attached to the optical end of the endoscope. The image is then viewed via an oculus, or eye-piece, directly to the operator's eye, or a video camera head which can be attached to the occulus. This image can then be transmitted to a video monitor and stored in an archiving system.

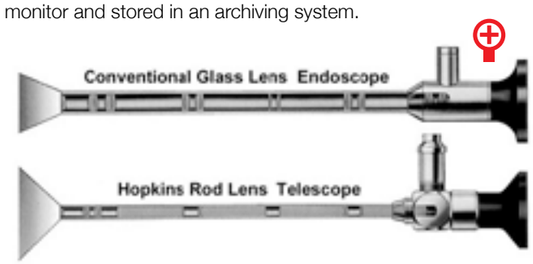


Figure 1: Two types of optical systems used in rigid endoscopes. The HOPKINS rod system is capable of transmitting considerably more light, and can produce a wider field of view, compared to conventional glass lens endoscopes. (Courtesy of Lhermette & Sobel, BSAVA Manual Endoscopy & Endosurgery 2008)

Rigid endoscopes can vary in diameter, length and 'viewing angle' (Figure 2). The viewing angle, at the distal tip, affects both the orientation and visualisation of the operative field of interest. The

simplest orientation is the 0°, or forward-viewing, endoscope. It is the most intuitive of the viewing angles because the visual field is in line with the true field. In extremely small cavities, for example the feline nasal cavity, this view would be considered the most limited. One can widen the visual field by using an angled endoscope and rotating the light post along its longitudinal axis. Rigid endoscopes are available with viewing angles of 30°, 70°, and even 120°; however, the 30° oblique is the most suitable in regard to increasing visual field and ease of use.

Figure 2: The top endoscope is an example of a 2.7mm 30' oblique endoscope. The bottom endoscope is a 5mm 0' forward viewing endoscope.

It must be appreciated that takes significantly more practice and patience using an angled tip endoscope as the angled view is opposite to the insertion of the light cable. **Practical Tip:** When endoscope is held with the light cable up, the surgeon can achieve a more realistic and anatomical spatial orientation.

The choice of size, length and viewing angle of an endoscope is based on its intended use (i.e. rhinoscopy, otoendoscopy cystoscopy, vaginoscopy arthroscopy, laparoscopy, or thoracoscopy). A 5mm x 0 degree rigid endoscope (Figure 3) is considered standard for most small animal laparoscopic and thoracoscopic procedures, while a 2.7-4.0mm x 30 degree oblique endoscope is more appropriate for cystoscopy/vaginoscopy, otoscopy and rhinoscopy. In the author's experience, 1.9mm x 30 degree oblique endoscope with integrated operating sheath is the most appropriate for performing rigid rhinoscopy in cats. A working length of 30cm is considered universal for laparoscopic procedures, while 18cmm endoscopes are frequently used for most other applications (i.e. rhinoscopy, otoendoscopy, and cystoscopy).

Figure 3: This is a 5mm, 0 degree rigid endoscope most commonly used in small animal laparoscopic procedures.

A large variety of different cannulae are available to facilitate entry of the endoscope into a particular body cavity. For instance, an endoscope can be inserted within a cystoscopy sheath and thus turning the endoscope into a 'urethrocystoscope'; whereas a cannula can used to traverse the abdominal wall and thus turning the endoscope into a 'laparoscope' (Figures 4 and 5).

Figure 4 (left): Threaded cannulae (EndoTip®) can be used in laparocopy. The threaded profile can improve retention within the body wall.

Figure 5 (right): A reusable 5mm trocar-cannula unit consisting of an insufflation port, trocar with pyrimidal tip, smooth cannula and valve.

Each procedure incorporating a rigid endoscope has a variety of accessory instrumentation. This allows for biopsy, aspiration, cautery, or other surgical interventions. Additional devices are usually employed for particular endoscopic procedures, such as carbon dioxide (CO_2) insufflators for laparoscopy (Figure 6), motorised shaver systems for arthroscopy and diode lasers for endoscopic laser surgery (Figure 7). Regardless of what procedure is being performed, it is important to remember that the basic rigid endoscopic chain will always consist of the light source, light guide cable, endoscope, sheath/cannula, camera system, and video system. Considering its versatility, the rigid endoscope and imaging chain is considered a very cost-effective purchase given the large breadth of endoscopic procedures that can be performed with relatively minimal initial investment on equipment.

Figure 6: Carbon dioxide insufflator required for laparoscopic surgery.

Figure 7: Diode laser can be used for therapeutic laser endosurgery (Courtesy of Philip Lhermette).

Figure 8: Endoscopic towers can range from a 'mix and match' of different makes/models, to very expensive and top-of-the-range endoscopy stacks. Regardless, a tower typically will routinely incorporate a monitor, light source, camera system, video processer, and archiving system.

A camera system and monitor (Figure 9, 10, 11) are essential for all forms of endoscopy. Although it is physically possible to squint down the oculus of rigid (or flexible fibreoptic) endoscope, the image obtained is rarely adequate for diagnosis, can be considered relatively unhygienic (e.g. rhinoscopy, cystoscopy and vaginoscopy) and renders surgical endoscopy virtually impossible. Many camera systems can be adapted to fit both rigid and flexible fibreoptic endoscopes and even video endoscopes. This will easily enhance your diagnostic and surgical capabilities as well as enabling you to record and archive images for client communication, animal medical files, and to accompany pathological samples for laboratory interpretation.

Figure 9: Video camera head that attaches to the oculus of rigid endoscope

Figure 10: The video camera head cable attaches to the camera control unit (CCU) top, and the light cable attaches to the xenon light source, bottom

Figure 11: Image is displayed on video monitor

A suitable light source (Figure 10) can also be used for both flexible and rigid endoscopy. It's best to choose a xenon or metal halide light source that is sufficiently powerful for the largest of your patients. In large open spaces such as the stomach or abdomen, light will significantly absorbed, so whilst output is less of a problem in smaller spaces (e.g. rhinoscopy or cystoscopy), if you plan to do any laparoscopy or upper gastrointestinal endoscopy then a powerful light source is absolutely essential. Halogen light sources are generally considered unsuitable for a variety of procedures. It's important to ensure that the light source can be adapted to fit both the flexible endoscope and the light guide cable (Figure 12) of the rigid endoscope.

Figure 12: Fibreoptic light cable that attaches to light post of rigid endoscope

Where Do I Buy My Equipment?

Access to various online auction sites offering endless supply of 'bargain' priced endoscopes can be enticing; however, buyer beware! There's usually a good reason why these endoscopes were retired from their original job in the first place! Spare parts may no longer be available and defects may not be apparent on cursory examination. What appeared to be a good deal can turn out to be wasted money on a product that is unusable, unserviceable, or not appropriate for the vast majority of procedures encountered in everyday practice. As a result, the endoscope eventually ends up in a storage cupboard collecting dust. It's therefore recommended to purchase the best quality equipment that you can afford and suitable for your practice needs. Eventually this purchase will pay for itself over several years due to superior diagnostic capabilities, and subsequently increased use. With appropriate care, these endoscopes will also last for many years and you will see a relatively quick return on your investment. Currently available instrumentation includes endoscopes designed for use in humans and endoscopes manufactured specifically for veterinary use. Purchasing new equipment not only provides you with a manufacturer's guarantee, but also includes staff training on equipment cleaning and maintenance, thus enhancing the life of your investment!

INTRODUCTION TO THE 'OSCOPIES'!

Rhinoscopy

Endoscopic examination of the rhinarium and frontal sinuses is referred to as 'rhinoscopy'. It can be performed quite easily on most canine and feline patients over 5kg using a 2.7 mm 30 degree angled rigid endoscope (a smaller outer diameter sheath can be used for smaller patients with the same endoscope) (Figure 13). The author usually and preferentially uses an integrated 1.9mm 30° oblique endoscope for feline rhinoscopy (Figure 14 +15). A cystoscopy sheath can be used with a 2.7mm endoscope which has an instrument channel that allows for the passage of accessory instruments as well as two-way stopcocks to continuous fluid ingress and egress to remove blood, mucus or other tissue debris from the field of view. Another advantage of continuous fluid irrigation is that it can act as a superior medium and enhance tissue magnification compared to that of air. The entirety of both the dorsal and ventral nasal meati can be examined adequately to the level of the ethmoid turbinates. Given the minimal trauma to the nose with endoscopy, and the constant irrigation of the field of view, the visualisation is in many ways superior to that of traditional open surgery. Some endoscopists will use a diode laser fibre within the endoscope to remove polyps and tumours without the need for aggressive open surgery (Figure16).

Urethrocystoscopy

The reproductive tract, urethra and bladder of the female patient can be examined with similar instrumentation to that described above for rhinoscopy. With careful fluid irrigation, the vagina, urethra and bladder can be well distended to allow for complete examination, assessing for ectopic ureters, urolithiasis, tumours, polyps and strictures. In addition, biopsy samples can be collected and a diode laser fibre can allow for surgical resection or debulking of proliferative tissue. The lower urinary tract of male canine patients or smaller female patients can also be accessed using a rigid endoscope via a prepubic percutaneous approach (laparoscopic-assisted cystotomy). The trocar and endoscope can be inserted into the fluid distended bladder via a ventrally placed abdominal cannula. This allows for the operator to examine the bladder and proximal urethra in patients whose urethral lumen will not permit passage of the endoscope. It can also allow for retrieval of uroliths from the bladder.

Figure 13: Photo 13: A 'universal' rigid endoscope can be used for rhinoscopy and cystoscopy. This is a 2.7mm 30 deg ree oblique telescope with cystoscopy sheath.

Figure 14: A 1.9mm 30 degree oblique rigid endoscope with integrated sheath is useful for feline anterograde rhinoscopy.

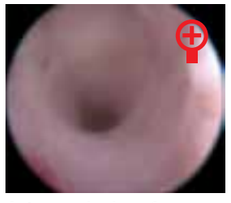
Figure 15: Distal tip of 30 degree oblique rigid endoscope and integrated sheath.

Figure 16: Use of a diode laser fibre inside operating channel of rigid endoscope sheath. Note the 'ingress' and 'egress' ports on either side of the endoscope which allows for the attachment of continuous irrigation fluid.

Otoendoscopy

The use of videootoscopy has grown it its popularity in veterinary practice. With the aid of a video camera attached to the eyepiece of a rigid endoscope, the ear canal and tympanic membrane can be brightly illuminated and magnified, allowing for a more detailed visual examination. In some cases, the videootoscope can be used in the presence of the client in the examination room. This can enhance owner communication and compliance in treating chronic ear disease. The videootoscope may be used in conscious as well as in the sedated or anaesthetized patient for ear irrigation procedures. The universal 2.7mm 30' oblique rigid endoscope used for cystoscopy and rhinoscopy can also be used with a cystoscopy sheath and 'converted' into an

Image of mucosal 'blunting' indicative of chronic inflammation associated with chronic 'polypoid'/ LP rhinitis in cat (middle meatus)



Anterograde view of nasopharyngeal stenosis in cat with 13 year history of chronic rhinitis

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otoendoscope. The ingress port can be connected to a fluid giving set resulting in controlled continuous irrigation and enhanced magnification of ear structures. The biopsy port can be used as a suction channel to aspirate fluid and debris. The ears can be continuously flushed and foreign objects, debris, or parasites can also be retrieved under direct visualisation using grasping forceps. Soft tissue masses can be biopsied using cupped biopsy forceps, and myringotomy performed with a catheter. With an attachable dual-port adapter, suction and saline may be used simultaneously to completely clean the ear.

Conclusion

In conclusion, the author's experience using rigid endoscopy has provided an easy and rewarding minimally invasive alternative to traditional diagnostic and surgical interventions for upper respiratory, urologic, and otologic conditions. If rigid endoscopy is going to be profitable, quality equipment needs to be purchased and needs to be readily available to be used on virtually every appropriate patient seen in the practice. Endoscopy can be an extremely valuable and versatile part of the clinician's diagnostic and therapeutic armamentarium.

Suggested Reading

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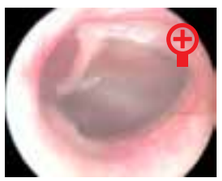

Figure 17: Normal feline tympanic membrane (Courtesy of Philip Lhermette)

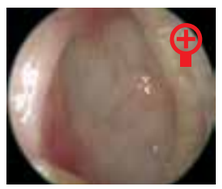

Figure 18: Opaque feline tympanic membrane due to chronic otitis externa/media (Courtesy of Philip Lhermette)

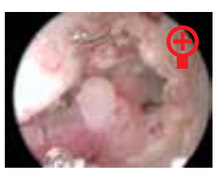

Figure 19: Adenocarcinoma affecting middle ear in cat (Courtesy of Philip Lhermette)

Figure 20: Otoscopic lavage and suction under direct visual guidance (Courtesy of Philip Lhermette)

CVE/ISFM FELINE MEDICINE DE COURSE

FROM THE DE LISTSERVE

ACUTE PANCREATITIS CASE

C&T No. 5442

Donald Wiggins – 2013 Feline Medicine Participant

United Kingdom
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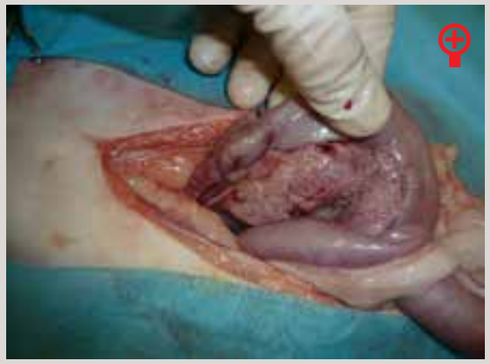
These images are from a recently deceased Burmese cat, 4yr, MN, BCS 6/9. Presented with acute onset vomiting, depression and quite marked abdominal pain (for a cat).

In spite of analgesia, ultrasound was tricky because of the pain but the bile duct was obviously dilated. The owner did not want any 'heroics' but allowed exploratory surgery after i.v. fluids. At surgery the whole pancreas was very red, and the bile duct was dilated and tortuous. The owner requested euthanasia at this point.

After euthanasia the duodenum was opened and the bile duct cannulated – it was obstructed. The pictures were taken after euthanasia so the marked erythema of the pancreas has gone. It is a shame we did not get a chance to try managing the case.



Figures 1 & 2. Picture of pancreas at exploratory laparotomy

PERSPECTIVE 112

MANAGEMENT OF FELINE PANCREATITIS: ROUNDTABLE DISCUSSION

Associate Professor Vanessa Barrs
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Special thanks to our Guest Editor, Andrea Harvey, for coordinating this Perspective

Associate Professor Vanessa Barrs

BVSc(hons) MVetClinStud FANCVSc (Feline Medicine) GradCertEd

Associate Professor Vanessa Barrs is the Director of the University Veterinary Teaching Hospital, Head of Small Animal Medicine at the University of Sydney and a registered Specialist in Feline Medicine. She has served as President of the Feline Chapter of the Australian and New Zealand College of Veterinary Scientists, Specialist representative of the NSW Board of Veterinary Practitioners and trustee of the Australian Feline Health Research Fund. She on the Board of Directors of the Australasian Society of Feline Medicine and is currently scientific editor for The Veterinary Journal. Vanessa's research interests include feline infectious diseases and alimentary lymphoma. In addition to over 80 refereed publications and book chapters, Vanessa is a busy practitioner, enjoys all aspects of feline medicine and loves nothing more than helping sick cats get better.

Kath Briscoe

BVSc (Hons I) MVetStud (Small Animal Clinical Studies) FANZCVS (Feline Medicine) Registered Specialist in Feline Medicine

Katherine graduated from the University of Sydney in 2003, graduating with first class honours and the University Medal. Kath worked in private small animal practice in Sydney and the UK for three years. In 2006, Kath completed her internship in small animal medicine at the University Veterinary Teaching Hospital, Sydney after which she completed her Fellowship training program in feline medicine. In 2008, Kath attained membership of the Australian and New Zealand College of Veterinary Scientists in feline medicine. During her residency, Kath completed a research project on the pathology of feline low grade alimentary lymphoma and inflammatory bowel disease. Kath has a keen interest in all aspects of canine and feline medicine, and is also passionate about providing continuing education for veterinary practitioners. After 5 years at the University of Sydney, Kath joined the team at the Animal Referral Hospital, Sydney where she continues to work. Kath passed her Fellowship examinations in June 2012.

Andrea Harvey

BVSc DSAM(Feline) DipECVIM-CA MRCVS MANZCVS (Assoc) Registered Specialist in Feline Medicine

Dr Andrea Harvey graduated from University of Bristol Veterinary School, UK in 2000, and after a couple of years in small animal practice, returned to Bristol Vet School to undertake a 3 year residency in feline medicine, funded by the Feline Advisory Bureau (now International Cat Care). She then remained at Bristol Vet School as FAB Lecturer in Feline Medicine until 2011 after which she moved to Sydney where she runs the feline medicine service at Small Animal Specialist Hospital. Andrea is also the International Society of Feline Medicine (ISFM) Australasian Representative, bringing various initiatives such as the ISFM 'Cat Friendly Clinic' scheme to Australia, and helping to develop the international profile of the feline chapter of ANZCVS. Andrea has lectured widely internationally and contributed to numerous textbooks, including being co-editor of the BSAVA Manual of Feline Practice. Andrea has wide interests in all aspects of feline medicine, and is passionate about both providing the best care for her patients, and helping to support other veterinarians to do the same.

Amy Lingard

BVSc (hons) FACVSc (Feline Medicine)

Amy graduated from the University of Queensland in 2001 and quickly made her way into feline-only practice. She spent three years as a feline practitioner at The Cat Clinic in Brisbane, before undertaking a residency in feline medicine at the University of Sydney Valentine Charlton Cat Centre in 2005. She attained Fellowship of the Australian and New Zealand College of Veterinary Scientists in feline medicine in 2010 and is now a registered feline medicine specialist. Amy is currently secretary of the feline chapter of the Australian and New Zealand College of Veterinary Scientists. She has published many peer-reviewed journal articles. Amy currently operates the Cat Clinic in Prahran in Melbourne, along with her partner Dr Richard Gowan. Her main clinical interests are alimentary disease and endocrine disorders.

1. WHAT SUPPORTIVE TREATMENTS DO YOU FIND MOST USEFUL FOR MANAGING CATS WITH ACUTE PANCREATITIS?

VB **Vanessa Barrs:** In the last 5 to 10 years, with the widespread availability of feline pancreatic lipase assays and our improved ability to recognize changes indicative of pancreatitis with abdominal ultrasound, I have seen prompt and dramatic responses in cats to relatively simple treatment regimens using combination therapy with fluid therapy, antiemetics, analgesics, appetite stimulants and early enteral support.

Fluid therapy is indicated, especially in acute pancreatitis (AP), to correct fluid losses and electrolyte/acid-base imbalances, to maintain intravascular volume and to improve pancreatic perfusion. I routinely use Hartmann's solution (compound sodium lactate). In humans, the use of lactated ringer's solution (LRS), which is very similar to Hartmann's, has been found to produce better patient outcomes in patients with AP compared to normal saline. An important component of fluid therapy is monitoring and correcting electrolyte imbalances – both hypo- and hyperkalaemia have been associated with poor patient outcomes in cats with AP. I also monitor ionized calcium, since hypocalcaemia is a poor prognostic factor in cats with AP, and supplementation with calcium gluconate may be indicated.

In addition to crystalloid therapy, I sometimes use colloids, especially in patients with tissue oedema, hypovolaemia or low oncotic pressure, e.g. hydroethyl starch (Voluven 6%, hydroxyethyl starch 130/0.4) (5 mL/kg over 15 minutes, repeated up to 4 times daily). In cats with severe acute pancreatitis that are not responding to other therapies, although there is little in the way of evidence-

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based medicine to determine efficacy, I will administer plasma as a source of albumin, clotting factors and protease inhibitors such as α-macroglobluin. Plasma can be given at 10 – 40 mL/kg/d in 5 mL/kg aliquots. If concurrent anaemia is present, a typed and/or cross-matched whole blood transfusion can be given instead of plasma.

The use of antimicrobials in AP is controversial. I try to avoid routine use unless there is a specific indication, for example concurrent bacterial cholangitis, or evidence of gastric ulceration. Having said this, in a patient with pyrexia and a left-shift neutrophilic leukocytosis and/or leukopenia, although this could be due to non-sterile inflammation from the pancreas, I will add in antimicrobial therapy. Haematogenous and transmural spread of E. coli from the colon to the liver and pancreas has been demonstrated in an experimental model of AP in cats (Widdison et al, 1994).

Early enteral support is important, especially as cats are at risk of developing concurrent hepatic lipidosis. In one study 45% of cats with AP had ultrasonographic evidence of hepatic lipidosis (Kimmel et al 2001). In cats that are critically ill but not persistently vomiting naso-oesophageal tube-feeding is a good short-term solution for providing enteral support (Klaus et al, 2009). For more stable patients, or for patients that are having other procedures done under anaesthesia, for example intestinal or pancreatic biopsies, I will routinely place an oesophagostomy tube. The quick reference guides in the BSAVA Manual of Feline Practice are an excellent resource for further information about enteral support in cats, including tube placement and caloric feeding guides. Unlike dogs, there is no evidence to date that fat restriction is beneficial in cats with AP. A low carbohydrate, high-protein, moderate-fat diet is generally indicated.

Other supportive therapies that I will prescribe for AP depend on the clinical signs that the patient is showing. Since complications associated with systemic inflammatory response syndrome/multi-organ dysfunction syndrome are not uncommon, I will perform thoracic radiographs and/or abdominal ultrasounds to monitor for effusions where signs are suggestive of these. Similarly, I will use CBC/coagulation profiles to monitor for disseminated intravascular coagulation in critically ill patients with severe AP.

Amy Lingard: Intravenous fluid support is possibly the most important treatment for acute pancreatitis; as one of the major factors in the progression of pancreatitis is disturbed pancreatic microcirculation. Like Vanessa, I use Hartmann's solution (Compound sodium lactate). In my experience, the majority of cats with acute pancreatitis present with hypokalaemia (rather than hyperkalaemia) and in these cats potassium chloride supplementation is indicated.

I strongly agree with Vanessa that early enteral nutritional support is extremely important in cats with acute pancreatitis. Once emesis is controlled, I would generally start an appetite stimulant, such as mirtazepine 1mg once daily orally (compounded) or cyproheptadine 2mg twice daily orally. If the patient remains anorectic for > 3 days, I would place an oesophagostomy tube (or a naso-oesophageal feeding tube in less stable patients), and reintroduce nutrition gradually over the next few days. I use the KITTY KOLLAR®, a washable, fabric collar designed to wear in conjunction with an oesophageal feeding tube. The comfortable collar replaces the bandaging normally used to hold the tube in place, keeping it stable and more protected against inadvertent removal (www.kittykollar.com/Product.php). I am hesitant to forcibly syringe feed, as it can induce food aversion.

I do not routinely use antimicrobials in cats with acute pancreatitis, unless there is a specific indication as Vanessa suggested. However, I am aware that there are some studies using culture-independent methods suggesting that greater consideration needs to be given to the role that bacterial infection may play in feline pancreatitis.

For me, the key to successfully managing acute pancreatitis is early recognition of severe cases and institution of aggressive and prompt therapy.

Kath Briscoe: Like Amy and Vanessa, my top 4 supportive treatments for AP in cats are: IV fluids, analgesia, anti-emetics and nutritional support. My preference is for placing an oesophageal feeding tube in any stable patient that hasn't eaten for 2-3days, as it is a simple, quick procedure and you can use the tube almost immediately after placement. One word of caution, though, is to make sure you check that you have placed the tube correctly by taking both lateral and DV thoracic radiographs after the procedure and while the patient is still anaesthetized. Unfortunately, I have witnessed the effects of food being placed directly into the thoracic cavity from an incorrectly placed tube and it didn't end well. I also use the Kitty Kollar described by Amy and find it really useful. Other than that, I don't have much to add!

Andrea Harvey: I also agree largely with the comments made by the others and don't have much to add, again my top treatments would be IVF, analgesia and anti-emetics, with nutritional support if the patient isn't eating after 2-3 days. Although I totally agree with the comments about oesophagostomy tubes, I do often use naso-oesophageal tubes initially in these cats (I use the soft silicone ones), and especially if I anticipate that they are likely to start eating within a few days, and are going to need to be in hospital during that time. Then, often they are eating again by the time they are ready for discharge and you can avoid the need for an oesophagostomy tube, but it all depends on the severity of the disease. I also tend to delay starting appetite stimulants until they have at least had a couple of days of anti-emetics and analgesia, just because I have observed suspected food aversion occurring when using appetite stimulants in a cat that may still be in pain or nauseous.

2. WHAT ARE YOUR FAVOURITE ANTI-EMETICS IN CATS WITH PANCREATITIS?

Vanessa Barrs: Maropitant (1 mg/kg SC, PO q 24 h), an NK-1 receptor antagonist that blocks centrally and peripherally mediated emesis, is generally the antiemetic of choice for me, because of its antiemetic, antinausea and visceral analgesic properties. In patients with severe vomiting that is not controlled by maropitant I will add in the 5HT3 receptor antagonist ondansetron (0.2-1 mg/kg IV, SQ, PO, q 8 – 12 h). Mirtazapine (1.88 mg q 24 h or 3.75 mg q 72 h), which is highly effective as an appetite stimulant in cats, also has anti-nausea properties and is a 5HT3 receptor antagonist. I do not routinely use metoclopramide for its anti-emetic properties, as it is less effective anti-emetic than maropitant or ondansetron, due to a lack of receptors in the chemoreceptor trigger zone. Where it can be beneficial, is for its prokinetic properties, in patients with ileus, administered as a CRI (1-2 mg/kg per 24 h).

Amy Lingard: For me, anti-emetics are indicated in all cats with pancreatitis regardless of whether vomiting or nausea is observed. I also use maropitant given its antiemetic and visceral analgesic properties, which makes it doubly useful in cats with pancreatitis. There are reports of off-label intravenous use of maropitant, but I have only ever used it subcutaneously. Like Vanessa, I would use ondansetron in patients with intractable vomiting. I only use metoclopramide as a prokinetic in patients with functional ileus, never as an anti-emetic.

Kath Briscoe: Maropitant is by far and away the most useful anti-emetic in my experience. Ondansetron would be my next choice, and like Amy and Vanessa, I only really use metoclopramide for prokinetic effects.

Andrea Harvey: I agree, I love maropitant for these cases, and like Amy I use it in all pancreatitis cases whether or not vomiting or nausea is observed. This is largely because I believe that nausea, even if not obviously present, is a key reason for inappetence in these cases. I think the visceral analgesia properties are also useful. I have sometimes used it off-label IV. Once vomiting is controlled, I most commonly use it orally to avoid the 'sting' of injection, and this is also useful if costs need to be minimized. Like my colleagues, for intractable vomiting (which I find uncommon in cats) I use ondansetron, and metoclopramide CRI for prokinetic effects if significant ileus is present.

3. WHAT ARE YOUR FAVOURITE ANALGESICS IN CATS WITH PANCREATITIS?

Vanessa Barrs: While abdominal pain can be detected in some cats on abdominal palpation, or during ultrasonography when the probe is placed over the pancreas, failure to detect it does not rule it out. In cats with severe AP and refractory abdominal pain I will use combination analgesia, e.g. ketamine continuous rate infusion (CRI) (2 – 10 µg/kg/min) combined with a fentanyl CRI (.02 - .06 µg/kg/min). In cats with less severe pain I will use methadone (0.1 – 0.2 mg/kg IM or IV 1 4-6h). I find buprenorphine is extremely useful for mild to moderate pain (0.01 – 0.03 mg/kg) and I usually administer it IV and then transition to transmucosal oral dosing at 0.01 – 0.02 mg/kg q 6- 8 h when patients are off IVF.

Amy Lingard: I strongly agree with Vanessa that the absence of abdominal pain on palpation does not rule it out. Anecdotal evidence suggests that abdominal pain is present in up to 75% of cats with pancreatitis, so my recommendation is to provide analgesia regardless. Rather than a fentanyl CRI, I tend to use fentanyl transdermal patches in cats with severe pain. For a >5kg cat, I would apply a 25µg patch; for a 3-5kg cat: 17 µg (2/3 patch); and for a <3kg cat: 12.5µg (1/2 patch). To ensure good contact, I tend to not only clip, but also shave the area with a disposal razor. The site needs to be thoroughly dried prior to applying the fentanyl patch which is then covered with op-site and elastoplast dressing. Adequate fentanyl blood levels are attained 3 to 12 hours after patch placement in cats, so in the interim I administer methadone 0.1-0.2mg/kg IM / IV. For patients with moderate pain, I would typically use buprenorphine 0.01–0.02 mg/kg every 8-12 hours IM / IV, or sublingually in outpatients. In the past morphine has been associated with increased sphincter of Oddi activity, but more recent studies have failed to show any adverse effects when treating human pancreatitis patients with opioids.

Kath Briscoe: Like my colleagues, I use analgesic drugs in any patient that I suspect has acute pancreatitis, and even for most cats with chronic pancreatitis. I tend to use morphine/ketamine infusions for the more severe cases, and methadone either as an infusion or bolus doses for the "standard" acute pancreatitis case. For cats with chronic pancreatitis, transmucosal buprenorphine (0.01-0.02mg/kg q 8-12hours) is my preferred treatment, though I have also used oral tramadol to some effect. Of course, when dispensing oral buprenorphine I make sure that I comply with the regulations for dispensation of S8 drugs for home use (e.g. each individual syringe is labelled, no needles are provided with the syringes, and the syringes are dispensed in a child-proof container).

Andrea Harvey: Again I agree with all my colleagues here, and always use opioid analgesia in any confirmed or suspected pancreatitis cases. I most commonly use methadone bolus doses and transmucosal buprenorphine at the doses stated above. Buprenorphine provides excellent analgesia for the vast majority of cases, but like my colleagues I will use more aggressive analgesia in cases of acute pancreatitis with more severe intractable abdominal pain. I commonly dispense buprenorphine for transmucosal use at home, and believe that along with nausea, abdominal discomfort can be a key reason for persisting inappetence in cases where adequate analgesia is not provided.

4. DO YOU ROUTINELY USE ANTACIDS AND, IF SO, WHICH ONES? AND WHY?

Vanessa Barrs: Like antimicrobial use, this is another controversial area. AP is thought to predispose to gastric ulceration because of hypovolaemia and local peritonitis. In addition, the rationale for its use is that increasing the gastric pH will result in decreased exocrine pancreatic stimulation, and proton-pump inhibitors may also decrease the activity of the ATPase pump of pancreatic cells. There is no evidence base to support the routine use of antacids in humans or dogs with AP. Therefore, I do not routinely use antacids in the treatment of AP in cats unless there are specific indications such as evidence of gastric ulceration, e.g. melaena, haematemesis or haematochezia.

Amy Lingard: Despite a lack of evidence supporting its use, I have to admit that we often use ranitidine or esomeprazole IV in cats with acute pancreatitis to suppress gastric acid. Ranitidine also has prokinetic effects, which may be beneficial in cats with functional ileus secondary to pancreatitis. And there is some evidence to suggest that pantoprazole possesses reactivity toward hydroxyl radicals. An experimental study in rats showed that pantoprazole reduced inflammatory changes and leakage of pancreatic acinar cells in severe acute pancreatitis. Unfortunately, oral omeprazole (Losec) is of little benefit in cats, as once the enteric-coating is broken the drug is rendered ineffective. For this reason, we would typically use famotidine if an oral antacid is required.

Kath Briscoe: I use antacids, predominantly esomeprazole 0.7-1.0 mg/kg IV SID, in some but not all cases of acute pancreatitis, and it is not part of my routine treatment for chronic pancreatitis. Those cases of acute pancreatitis for which I would consider antacids include any with evidence of gastrointestinal (GI) ulceration, or those in which I suspect acute necrotizing pancreatitis (i.e. the more severely unwell patients). Again, there is no evidence to support its use but it may be of benefit and is unlikely to do harm.

Andrea Harvey: Like Vanessa I don't routinely use antacids in these patients, but do use them in certain situations, namely in cases with melaena or haematemesis, or where there is an unexplained ongoing and significant drop in packed cell volume (with the assumption that there may be GI blood loss in these cases). I also use them in some cases of severe acute pancreatitis where the cat is not improving, and in that situation I use them with the rationale as described by Vanessa. In these situations I use esomeprazole IV or omeprazole PO (1mg/kg). I must admit that I do tend to use it once daily, although there is recent evidence that twice daily administration should really be used in cases of GI ulceration, so in a more severe case I would use twice daily administration. I use ranitidine for its prokinetic effects in cases where I suspect GI ileus largely based on ultrasonographic evidence of ileus.

5. WHAT IS YOUR APPROACH TO MANAGING CHRONIC PANCREATITIS IN CATS?

Kath Briscoe: Unfortunately, while we know that chronic pancreatitis occurs with reasonable frequency, we don't know the best way to treat it. My approach to the cat

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with suspected or confirmed chronic pancreatitis is to manage the symptoms associated with the disease, and try to minimise the frequency of episodes of acute pancreatitis. I feel that vomiting is best controlled using the NK1-receptor antagonist, maropitant. Maropitant has excellent antiemetic properties, and may also have some effect in managing visceral pain. It is also my opinion that nausea in cats is not well recognised, so I counsel my owners to observe for signs of nausea such as hypersalivation and/or lip-smacking, and to administer maropitant even if the cat is not necessarily vomiting. Provision of analgesia is a little more challenging, as we really have no idea of whether our patients with chronic pancreatitis are painful or not. Again, I tend to err on the side of caution and assume that the patient is uncomfortable if they show any signs of discomfort. My preference is for use of oral buprenorphine (with the caveat that the drug must be labelled and stored as compliant with the law) or oral tramadol.

In addition to this, evaluating for evidence of intercurrent disease e.g. inflammatory bowel disease (IBD), diabetes mellitus, chronic renal disease, and managing these as appropriate is of paramount importance to the overall health of the cat.

Andrea Harvey: I largely agree with Kath. We don't really know how to best manage chronic pancreatitis, and currently it is most common just to manage symptoms as they arise. The most common sign with chronic pancreatitis is usually inappetence. I feel that this is attributed to a combination of nausea and pain, and so like Kath I frequently use maropitant in these cases even if they are not vomiting, for the reasons Kath has outlined. I do believe that pancreatitis is painful, and in inappetent cats with pancreatitis I assume they may be in pain and use buprenorphine administered on buccal mucous membranes, usually until the cat is eating again. I personally don't like tramadol in cats and find cats often get quite 'spacey' on it, and it is harder to dose than buprenorphine. I also often use mirtazapine as an appetite stimulant. If loss of appetite is due to pain and nausea, appetite should return when these are appropriately managed. However, many cats develop food aversion and mirtazapine can be helpful in overcoming this. I only use it though once analgesia and anti-emetics are on board – because my feeling is that you can make food aversion worse if appetite is stimulated before pain and nausea is controlled.

As Kath also mentioned, managing any concurrent disease can be a mainstay of managing chronic pancreatitis, with it commonly occurring in association with inflammatory liver disease and IBD. Inflammatory liver disease is generally easier to recognize as liver enzymes and/or bilirubin are usually elevated. However, IBD can be more difficult to recognize and I think is under-recognised in cats with chronic pancreatitis. Cats may vomit periodically and owners often consider this to be 'normal' for their cat and not seek veterinary attention, or we may attribute the vomiting to the chronic pancreatitis. However, many of these cases may have concurrent IBD which may contribute to flare ups of pancreatitis. In these cases, management with an appropriate diet for their IBD (often a single source protein or hypoallergenic diet), +/- B12 supplementation and, if required, corticosteroids is necessary to manage the IBD, and may in these cases help with managing the chronic pancreatitis. Similarly, in cats with diabetes mellitus, whether diabetes mellitus makes pancreatitis worse or pancreatitis makes diabetes mellitus worse, we don't really know, and probably there is a bit of both. Therefore, good glycaemic control may be critical in reducing chronic pancreatic inflammation in diabetic cats.

I also think that nutritional management may be more important than previously thought although there is no evidence for this. This is addressed more in the next question.

I think that as we find out more about pancreatitis that it may become more important to try and specifically manage the chronic inflammation. I am becoming suspicious, purely anecdotally, that chronic pancreatitis may predispose to the development of pancreatic neoplasia, particularly as this happened in one of my own cats. How we most appropriately manage that chronic inflammation, and how we monitor response to any treatment, are also questions that as yet have no clear answers.

Vanessa Barrs: I most commonly diagnose chronic pancreatitis in cats with concurrent diabetes mellitus or concurrent inflammatory bowel disease (IBD) and less commonly in cats with concurrent cholangitis/cholangiohepatitis. Like Kath and Andrea I also favour combination triple therapy using oral transmucosal buprenorphine, maropitant and mirtazapine. I do not use tramadol in cats because of the dysphoria that many seem to develop. Analgesia appears to be effective in most cats with chronic pancreatitis treated with buprenorphine 0.02 mg/kg q 8 h given by the oral transmucosal route on an outpatient basis. Some cats can become dysphoric on that dose rate, in which case 0.01 mg/kg q 8 h is usually well tolerated.

Treatment of underlying IBD, if present, is an important part of therapy, and I agree with Andrea that enteropathies like IBD are probably under-diagnosed in cats with chronic pancreatitis. If the cat has underlying IBD and is not responding to dietary therapy alone, I will prescribe oral glucocorticoids (prednisolone) +/- cobalamin (Vitamin B 12) supplementation, if indicated. Although controversial, glucocorticoid therapy may have concurrent beneficial effects in chronic pancreatitis.

The 'chicken and egg phenomenon' regarding diabetes mellitus and pancreatitis is an interesting one. Most of us would be familiar with the concept of chronic pancreatitis causing islet cell loss due to fibrosis and atrophy. More recently a potential causal role of diabetes in feline pancreatitis has been proposed. In light of this, I agree with Andrea that good glycaemic control is important because of the potential to reduce chronic pancreatic inflammation.

Amy Lingard: Similarly, my approach to management of chronic pancreatitis is largely symptomatic, utilising oral transmucosal buprenorphine, mirtazapine and maropitant. Like Vanessa, I have also observed dysphoria in cats administered oral transmucosal buprenorphine at 0.02mg/kg. Differences in the buccal pH and swallowing of the drug by some cats might explain inconsistent bioavailability. Despite this, it remains my preferred option for analgesia.

Cats with pancreatitis may commonly have concurrent inflammatory bowel disease, so I often recommend parenteral cobalamin (vitamin B12) supplementation and feeding an appropriate diet, such as a highly digestible, novel protein or hydrolysed diet. In cats with chronic relapsing pancreatitis or concurrent IBD, I will often trial prednisolone 0.5-1mg/kg once daily orally. There are increasing reports of corticosteroid use for chronic pancreatitis, anecdotally with good results.

Though there is little evidence to support their use, I would consider trialling pancreatic enzyme supplementation and/or s-adenosylmethionine (Denosyl®)in cats that respond inadequately to the above management.

6. WHAT ARE YOUR VIEWS ON DIETARY MANAGEMENT BOTH IN ACUTE AND CHRONIC PANCREATITIS? HOW SOON DO YOU START FEEDING THEM, AND WITH WHAT?

Kath Briscoe: For cases of acute pancreatitis, I begin feeding as soon as vomiting is controlled. This is for multiple reasons. Firstly, withholding food may lead to weight loss which may precipitate the development of hepatic lipidosis in cats, which would make management a whole lot more complicated! Secondly, there is evidence that withholding food results in atrophy of the small intestinal villi, and alters the balance of small intestinal bacterial flora which may precipitate diarrhoea. So feeding as soon as is practical is of great importance, in my opinion. Obviously, I control the nausea and vomiting prior to feeding, as we don't want the cat to develop food aversion, and preferably I offer the foods that the cat likes to eat at home as the first foods offered. If the cat remains inappetent in hospital, my preference is to place an enteral feeding tube. Depending on how long the assisted feeding tube is likely to be in place, I use naso-oesophageal feeding tubes or oesophageal feeding tubes. Rarely do I find it necessary to place more long-term feeding tubes such as gastrostomy tubes. The cons of using the naso-oesophageal feeding tube is that only very liquid foods such as Ensure can be administered, and these are not ideal for feeding cats as they do not have the appropriate amino acid balance. Thus, in any patient that remains inappetent for more than a few days, and which is an appropriate candidate for a short anaesthetic, an oesophageal feeding tube is placed. Through this device I tend to feed a recovery diet such as Iams® (formerly Eukanuba) Maximum Calorie or Hills A/d®. Given that there is no evidence that high fat diets precipitate acute pancreatitis, I tend not to worry about the fat content of the diet. If the patient has intercurrent disease such as IBD, the diet I choose reflects the underlying disease.

For cats with chronic pancreatitis, we do not know which diet is most appropriate, so if there is an intercurrent disease for which dietary therapy is known to assist (e.g. chronic renal disease, diabetes mellitus), I tend to use that diet (in that case, Hills K/d® or similar, or Hills M/d® respectively). If there is no intercurrent disease, I recommend that the cat be fed the diet which it was previously accustomed to, with no specific dietary therapy instituted. Again, this is because it is uncertain whether diet bears any relation to the development of pancreatitis in cats.

Andrea Harvey: My approach to dietary management in acute pancreatitis is similar to Kath's. The only thing I would add is that when using naso-oesophageal tubes, I add whey protein (10g per day) to the Ensure. Liquid diets specifically formulated for cats are available in other countries (e.g. Royal Canin convalescence powder) but unfortunately not in Australia.

The only other thing that I have slightly different thoughts on to Kath is fat content of the diet. Kath is right that there is no evidence that high fat diets precipitate pancreatitis in cats, contrary to the situation in dogs and in people, and thus historically the approach in cats has been not to focus on the fat content of the diet. However, I have the impression that fat content can be important in some cases, although this is totally anecdotal. Again, this impression started with one of my own cats which developed pancreatitis after starting Hill's k/d™, a high fat diet. We can learn a lot from our own cats' illnesses! Subsequently, he seemed to get flare-ups with any slightly high fat foods, and I have since observed similar in other patients and, also more recently, heard top pancreatitis researchers advocating low fat diets for managing pancreatitis in cats too. So, my approach is that I get them eating whatever they will eat first, but try to stick to low fat foods like cooked chicken or tuna if the cat will eat them, and then try to wean them onto a commercial low fat diet. I figure that as long as they eat the diet it shouldn't do any harm, and might help in some cases. As there is nothing else that we are doing to specifically treat the pancreatitis, it makes me feel like I am doing something! This is my personal approach but as yet there is no evidence to support that.

When there is concurrent IBD, I would like to feed both single source protein or hypoallergenic diet, and low fat, however such a commercial diet doesn't exist! I tend therefore to go with whatever diet I feel best suited to managing the IBD, but during a flare-up of pancreatitis try to feed lower fat, e.g. if they are on a chicken and rice based diet, I would go with cooked chicken during a flare-up of pancreatitis.

Vanessa Barrs: We are all in agreement that early enteral nutritional support is a key part of treatment for cats with acute or chronic pancreatitis, and that NO-tubes are an excellent short-term solution for providing enteral support in critically ill patients, until they can be safely anaesthetized for the placement of oesophagostomy tubes. Andrea's observations about high-fat diets and flare-ups of acute pancreatitis are interesting, and although there is no evidence base, I will certainly be 'watching this space' for any developments in this area. We all know that cats are not small dogs, and this is especially true in regards to the anatomical differences in bile duct anatomy of cats and dogs that predispose cats to ascending biliary infections from the gut, BUT there do seem to be many similarities in the pathophysiological effects of acute pancreatitis in cats and dogs.

Amy Lingard: There are no studies to support diet selection for cats with pancreatitis. As mentioned, high-fat foods are not implicated in causing feline pancreatitis; though there is some suggestion that we should avoid feeding high-fat diets when treating these cats. I have to admit that initially I am not too concerned about what they eat, just so long as they are eating! I will offer a variety of options including dry food, wet food, cooked chicken and/or raw beef, based on the individual's preference. It is important not to overwhelm them with food, so I only offer small portions and replace them with fresh options frequently. The benefits of warming food, providing a flat dish and gentle nursing encouragement cannot be overestimated underestimated. Once patients are eating, I will usually try to introduce a highly digestible intestinal prescription diet.

For cats that remain inappetent for a few days, an enteral feeding tube should be placed. In stable patients, my preference is to place an oesophagostomy tube through which Hills A/D™ or Royal Canin recovery formula can be provided. I use appetite stimulants to help support caloric intake, decrease dependency on the feeding tube over time, and support the removal of feeding tubes.

In the longer term, given that many cats may have concurrent inflammatory bowel disease, my recommendation is to feed a highly digestible, novel protein or hydrolysed diet. New diets should be introduced gradually over a period of 1-3 weeks.

7. DO YOU USE ANTIBIOTIC TREATMENT IN CATS PANCREATITIS AND, IF SO, IN WHAT SITUATIONS?

Kath Briscoe: The use of antibiotics for cats with pancreatitis is controversial. Given that recent studies using technologies such as FISH (fluorescence in-situ hybridisation) have identified bacteria in the pancreata of cats with acute pancreatitis, I feel that there is justification for the use of antibiotics in this situation. Also, the anatomy of the cat is such that the pancreatic duct and common bile duct join prior to entry at a common point (the major duodenal papilla) in the duodenum. One theory behind the possible cause of acute pancreatitis in cats is that there is reflux of intestinal contents (and thus likely bacteria) up the pancreatic duct, causing activation of the pancreatic enzymes within the pancreatic tissue. This gives further justification of use of antibiotics in acute pancreatitis in cats. Having said this, I tend to use a broad spectrum but still 'first line use' antibiotic such as amoxycillin-clavulanate, and reserve more 'heavy hitting' antibiotics such as the fluoroquinolones for cases in which I have confirmed

bacterial infection (e.g. a case with confirmed concurrent acute neutrophilic cholangitis and a positive bile culture). I do not feel that antibiotics are appropriate for the management of chronic pancreatitis in cats.

Andrea Harvey: Again, I largely agree with Kath's comments. I tend to use antibiotics in cases of acute pancreatitis and particularly when there is evidence of, or suspicion of, concurrent neutrophilic cholangitis. Having said that, although bacteria have been found in the pancreas of cats with pancreatitis, the cause and effect relationship is still not known. I would just hate to miss treating a treatable cause, particularly given that this is the only potential aetiology that we can specifically treat. I also would usually use amoxicillin-clavulanate as a 'first line' in these cases, although I might change to marbofloxacin if the cat is really sick and not improving, particularly as it is gram negative bacteria that have been most commonly found in the feline pancreas. If I use a fluoroquinolone I always use marbofloxacin and not enrofloxacin due to the risks of retinal toxicity with enrofloxacin in cats.

In cats with suspected concurrent IBD I might also use metronidazole treatment, and whether or not this has any benefit in treating concurrent pancreatitis is unknown. I guess the other 'can of worms' to open is that some cats presenting with vomiting may also have Helicobacter spp infection. This is a whole controversial area of its own but just something else to consider as another potential concurrent disorder that we may see with pancreatitis, where antibiotics would be indicated. This is probably uncommon, but just something to have on our radars.

I also wouldn't routinely use antibiotics in cats with chronic pancreatitis. However, given the possibility that bacteria could be an inciting cause of inflammation, there are some cases of chronic relapsing pancreatitis that I would treat with say a 2 week course of antibiotics. Again, this isn't really evidence based, except that given some emerging evidence that bacteria may be an inciting cause of an overactive immune response leading to chronic inflammation, likely this is thought to occur in some chronic inflammatory liver diseases, so I would hate to miss a chance to treat an underlying aetiology. I don't like using antibiotics indiscriminately though and it is hard to know which cases may benefit, so I always try and carefully weigh up each case individually.

Vanessa Barrs: The use of antimicrobials in acute pancreatitis is indeed controversial. I try to avoid routine use unless there is a specific indication, for example concurrent bacterial cholangitis, or evidence of gastric ulceration (e.g. melaena). Having said this, in a patient with pyrexia and/or a left-shift neutrophilic leukocytosis or leukopenia, although this could be due to non-sterile inflammation from the pancreas, this is for me an indication for antimicrobial therapy. Haematogenous and transmural spread of *E. coli* from the colon to the liver and pancreas has been demonstrated in an experimental model of AP in cats (Widdison et al, 1994). Most *E. coli* isolated from our hospital are susceptible to amoxicillin-clavulanate, so I will use this or ticarcillin-clavulanate which can be given intravenously as first-line therapy. Similarly, *E. coli* and anaerobes are the most common bacteria isolated from cats with neutrophilic cholangitis/cholecystitis, so either of these antimicrobials are also appropriate for first-line therapy.

For cats with chronic pancreatitis, I do not prescribe antimicrobial therapy unless there are specific indications such as concurrent inflammatory liver disease.

Amy Lingard: I do not routinely use antimicrobials in cats with acute pancreatitis, unless there is a specific indication or clinical finding as suggested above. Another reason that I will avoid indiscriminate use of antibiotics is that they can exacerbate nausea and vomiting in cats.

I am aware that there are some studies using culture-independent methods suggesting that greater consideration needs to be given to the role that bacterial infection may play in feline pancreatitis.

For cats with chronic pancreatitis, I would not recommend antimicrobials. Though, in cats with concurrent inflammatory bowel disease I often use metronidazole 10mg/kg once or twice daily orally for its antimicrobial and potential immunomodulating effects.

8. DO YOU EVER USE CORTICOSTEROIDS FOR TREATING PANCREATITIS IN CATS AND, IF SO, IN WHAT SITUATIONS?

Andrea Harvey: I don't routinely use corticosteroids for treating pancreatitis but I certainly use them in some situations. I generally use corticosteroids in cats with suspected or proven concurrent IBD where clinical signs have not resolved with dietary and supportive management alone. IBD may have been proven on intestinal biopsies, but biopsies are not possible in all cases, and I will also suspect concurrent IBD in cats with a history of chronic vomiting and/or diarrhea and/or weight loss, and sometimes those with really severe diffuse ileus.

There are other situations that I do use corticosteroids in treating pancreatitis but those situations are hard to define! Pancreatitis is such a variable disease there are always lots of individual factors to weigh up. I guess I also consider using corticosteroids in any case of pancreatitis that is not improving with supportive therapy alone. Some of these older cats may have concurrent lymphocytic GI lymphoma rather than IBD, and in those cases some improvement may be seen with corticosteroids. Decision making can be difficult because at a time of acute pancreatitis and severe ileus, anaesthetizing for gut biopsies may create more complications, and so these type of cases I may trial on corticosteroids.

I will usually start off cautiously, maybe 0.5mg/kg daily and assess for response. In cats with acute pancreatitis I watch carefully to make sure they do not get worse with corticosteroids. I am always ready to stop them if there is worsening. If there is no worsening and perhaps some improvement but still not adequate improvement, then I might increase the dose, again carefully assessing for response. Some of these cases may still have concurrent IBD and so it is always hard to assess which disease the corticosteroids are having the biggest impact on. If fPL is significantly elevated then this can be very useful to monitor. If it significantly drops after starting corticosteroids then it gives you confidence that you are doing the right thing!

I might also trial corticosteroids in chronic relapsing pancreatitis where flare-ups are occurring frequently.

I generally avoid corticosteroids in cats with pancreatitis and diabetes mellitus although, ironically of course, if pancreatic inflammation is controlled then diabetic control may improve. However, I worry in most cases about development of diabetic ketoacidosis if glycaemic control is not being monitored very closely. There are some cases where if the owner is really switched on and is doing close home blood glucose monitoring, and are aware of the risks, then in this situation I might still try corticosteroids in a diabetic cat, but I would monitor very closely and cease if diabetic control was worsening rather than improving.

Kath Briscoe: I do not use corticosteroids routinely in cats with pancreatitis. I am aware that some advocate the use of corticosteroids for use in chronic pancreatitis, as is sometimes the case in humans with this disease, but I feel that we do not have enough information on the disease in cats yet to use this medication routinely. Like Andrea, there are some instances where I would recommend use of corticosteroids in a cat with pancreatitis. Specifically, if the cat had known or suspected underlying IBD or

low-grade alimentary lymphoma, if there was chronic relapsing pancreatitis with flare-ups occurring frequently, or if there is another disease for which corticosteroids are indicated. Again, I tend to start with lower doses and increase them only if I am not seeing the desired response. Rarely would I use immunosuppressive doses as a starting point. If the patient has concurrent diabetes mellitus, I would avoid use of corticosteroids.

Amy Lingard: Like Andrea and Kath, I would consider using corticosteroids in cats that are failing to respond to supportive management, those with confirmed or suspected inflammatory bowel disease, and in some chronic pancreatitis cases. In my experience, many of the cats that we see with chronic pancreatitis often have some degree of concurrent GI disease. I tend to use a slightly higher dose of prednisolone 1mg/kg once daily orally, and taper gradually once a clinical response is achieved. Anecdotally, most cats with concurrent IBD and pancreatitis respond well to prednisolone, though whether this is the pancreatic or intestinal disease (or both) that are responding is unknown. In cats with acute pancreatitis that are failing to respond to supportive therapy, I may administer a single injection of dexamethasone 0.15mg/kg subcutaneously. My rationale behind the use of corticosteroids in acute pancreatitis is relative adrenal insufficiency.

Vanessa Barrs: I use oral corticosteroids in cats with chronic pancreatitis and evidence of a concurrent enteropathy that have not responded to dietary therapy with a novel-protein single source carbohydrate diet, or where owners cannot feed such a diet to a hydrolysed protein diet. In cats with enteropathies and concurrent chronic pancreatitis, management of the enteropathy is often key to an overall improvement of the patient's health. Ideally, the enteropathy should be diagnosed histologically on intestinal biopsies, to enable the distinction between different subtypes of enteritis and also from low-grade alimentary lymphoma (LGAL) which is a very common enteropathy in cats over 10-years of age. Of course, biopsies are not always possible, and in these situations I will prescribe prednisolone at 1 mg/kg/24 h initially and monitor for a response. For cats over 10-years of age where LGAL is a major differential diagnosis for the enteropathy, I carefully monitor for resolution of vomiting and weight gain, and if that does not occur, I increase the dose of prednisolone (2 mg/kg/d) and add in chlorambucil 2 mg PO three times weekly. Cobalamin supplementation is also indicated in cats with distal small intestinal disease and evidence of deficiency.

Like Andrea, I also prescribe oral corticosteroids to cats with chronic relapsing pancreatitis and no evidence of concurrent enteropathy, and assess the response to therapy. I am very wary of using corticosteroids in cats with acute pancreatic (is this correct ?), and agree with Amy's comments that they may be warranted in very low doses for treatment of relative adrenal insufficiency.

9. DO YOU EVER USE NSAIDS IN CATS WITH PANCREATITIS AND, IF SO, IN WHAT SITUATIONS?

Andrea Harvey: I have never knowingly used NSAIDs in cats with pancreatitis and would really worry about GI side effects and GI ulceration. However, I can think of some cases with inappetence of unknown aetiology (which could be due to pancreatitis) where a colleague has started NSAIDs and the cat has consistently improved with this treatment. But I don't know what is being treated in that situation. I would always worry about prescribing NSAIDs to a cat that isn't eating, and I am always really strict about only using NSAIDs in cats that are eating, so I'm not sure how that would work. I am, however, also aware of a couple of medicine specialists that have used NSAIDs as a last resort in some cases of chronic relapsing pancreatitis in cats, with good response. So maybe there is a role, but I would be too cautious scared to use them at the moment, or to recommend them. You have to be so careful with NSAIDs with GI disease and inappetent cats in my opinion.

Kath Briscoe: No, I don't use NSAIDs in cats with pancreatitis. It is my thought that the risk of adverse effects from NSAIDs (GI ulceration and GI side effects, even acute renal failure) in an inappetent cat far outweighs the potential benefits. Having said that, if I had a patient that was eating well, had chronic pancreatitis and had a need for NSAIDs for another reason (e.g. osteoarthritis), I wouldn't necessarily withhold treatment, as long as the cat was eating well and the medication was given with food. Specifically, in that instance, I would use meloxicam and would use the lowest dose effective to control the patient's clinical signs.

Amy Lingard: If I am considering using an anti-inflammatory for pancreatitis, I am generally more likely to use a corticosteroid than a NSAID. However, I have used meloxicam in a number of cats with acute pancreatitis and concurrent bacterial cholangitis, in which there was persistent pyrexia and abdominal pain despite broad spectrum antimicrobials and opioids. It is critically important that hypotension and hypovolaemia have been corrected prior to administration of a NSAID. I tend to only use meloxicam 0.1mg/kg once subcutaneously or orally (note that this is lower than the registered injectable dose).

Vanessa Barrs: I steer clear of NSAIDs in patients with pancreatitis, and agree with Andrea and Kath that the potential adverse effects outweigh the potential benefits. If I want to use an anti-inflammatory drug I would use a corticosteroid.

10. ARE THERE ANY OTHER TREATMENTS THAT YOU HAVE USED OR THAT MAY BE USEFUL IN MANAGING PANCREATITIS?

Andrea Harvey: There aren't other treatments that I have frequently used. However, I do remember one particular case that was a diabetic cat with chronic frequently relapsing pancreatitis (clinical signs of inappetence, lethargy, vomiting and an extremely elevated fPL), and I suspected also IBD based on a history of chronic vomiting but no intestinal biopsies had been taken. Although bouts of pancreatitis responded to supportive treatment the owners were getting to the end of their tether with the frequency at which they were occurring. If he hadn't been diabetic I would have started corticosteroids, but I was worried about using them in this case. At the same time, I had just heard of a colleague in the US using cyclosporine in some cases of chronic pancreatitis, with anecdotally good success. After a lot of counseling with the owners on the pros/cons, risks etc, we opted to try cyclosporin in this case. Vomiting stopped almost immediately, fPL reduced from > 50ug/l to 20ug/l in one week, and the cats diabetic control improved substantially. If there is improvement with cyclosporin it would suggest an immune-mediated aetiology for pancreatitis, which has not been shown in cats. But of course this would be possible, when it is so closely related to IBD and inflammatory liver disease which is also thought to have an immune-mediated component. So, it may well be that immunosuppressive therapies have a role in some cases, and other drugs such as chlorambucil may have similar roles. Indeed, there are cases of lymphocytic GI lymphoma with concurrent pancreatitis that I have treated with prednisolone and chlorambucil with great response. But, of course, you never know if the treatment is actually improving pancreatic inflammation directly, or whether it is improving secondary to controlling the intestinal signs associated with the lymphoma. So there is definitely still so much that we simply don't know, but I think this is an exciting area, because it may be a way to more definitively treat the chronic inflammation and thereby reduce risk of neoplastic transformation, if that happens. With my own cat having had chronic pancreatitis and then a couple of years later developing pancreatic carcinoma, this is obviously an area that is close to my heart!

The other treatment worth a mention is supplementation with pancreatic enzyme. This is sometimes used in part of the management of people with chronic pancreatitis and said to reduce post-prandial pain, although there is little evidence for this even in people. I'm not aware of this being used very frequently in cats, and I have never really used this. However, again in my own cat I did try this! He really, convincingly to me, showed evidence of post-prandial pain, and I have never observed this in a cat before, but it may simply be that it is hard to observe in a hospital situation when cats' normal behaviours are altered, and that most owners are not observant enough to pick up these subtle behaviours. As a consequence of these observations with my cat, I tried pancreatic enzyme supplementation and subjectively thought it did help, but who knows really! It certainly won't do any harm so it is an option. In clients' cats however, one of the important things I focus on is not overwhelming them with too many medications, and it is very easy with pancreatitis where we don't really know the best treatments to suddenly have heaps of medications that you want to try! I always try to keep it as simple as possible and try to limit to around 3 medications at any one time if possible.

KB **Kath Briscoe:** There are no other medications that I would routinely use in cats with acute or chronic pancreatitis. Having said this, I have used cyclosporine in a couple of cats with suspected chronic pancreatitis. In both of these cases the fPLI was markedly elevated persistently and the cats' clinical signs were persistent despite use of antiemetics and appetite stimulants. In those cases, I hadn't wanted to use corticosteroids (one case had chronic FHV-1 infection and flared up with corticosteroids and the other was diabetic) and I had heard of colleagues using cyclosporine. In both of those cases, clinical improvement was seen, but the fPLI remained elevated, so I am not certain whether this was coincidental or due to the cyclosporine. I haven't tried using pancreatic enzyme supplements in cats. Like Andrea, I try to keep the number of medications we are using to a minimum, as we don't want the cats to 'rattle'!

AL **Amy Lingard:** I have used pancreatic enzyme supplementation in a number of cats with chronic pancreatitis and in some considered it to be beneficial. There are 2 cases that come to mind, in which there was an exceptional response. Neither cat had an fTLI performed, so it is possible that they had some degree of exocrine pancreatic insufficiency. The main drawback is that some cats will not eat well when pancreatic enzymes are added to their food.

I have used s-adenosylmethionine (SAMe) Denosyl® 90mg once daily orally in a few cats with pancreatitis. The main drawback is that SAMe must be administered as an intact tablet on an empty stomach at least 1 hour before feeding. Increased oxidative stress has been implicated as a potential factor in the pathogenesis of chronic pancreatitis. And it has been suggested that supplementation with an anti-oxidant may lead to a reduction in oxidative stress and reduce abdominal pain. Though, in most studies, a combination of anti-oxidants was required to relieve pain.

Cobalamin (B12) deficiency may occur in cats with chronic pancreatitis and concurrent intestinal disease. A deficiency of cobalamin can impair nutrient absorption. Whilst it is recommended that serum cobalamin levels be assessed, I often elect to simply trial parenteral cobalamin supplementation at 250µg/cat subcutaneously once weekly for 6 weeks, then once fortnightly for 6 weeks, then once monthly. An additional benefit to supplementation is that it may provide an appetite stimulant effect.

VB **Vanessa Barrs:** The only other therapy that I tend to use for pancreatitis in addition to those that have been discussed is plasma transfusion for cats with severe acute pancreatitis, as a source of albumin, clotting factors and protease inhibitors. In our hospital, plasma can only be sourced from a donor cat, so given that I am collecting a unit of 50 mL of whole blood, if the cat has concurrent anaemia I will give whole blood instead of just plasma alone. I have not used pancreatic enzyme supplements in cats with pancreatitis, and use cyclosporine for cats with IBD +/- concurrent pancreatitis where corticosteroid use has not been effective or is a significant risk factor for diabetes (e.g. Burmese or very obese cats). Given the high frequency of intestinal disease in cats with chronic pancreatitis, I agree with Amy that the possibility of cobalamin deficiency should be considered.

11. DO YOU FIND MEASURING SPEC FPL IS USEFUL AND DO YOU ADVOCATE USING THAT FOR MONITORING IN ANY SITUATIONS?

AL **Amy Lingard:** I routinely measure feline specific pancreatic lipase (Spec fPL) in all patients presenting with a history of being 'unwell', particularly those patients with non-specific clinical signs such as lethargy, inappetence, vomiting, or abdominal discomfort; and in all patients with suspected or confirmed primary GI disease, hepatic disease or diabetes mellitus.

Spec fPL is currently the most sensitive and specific serum marker available for the diagnosis of feline pancreatitis. In a study evaluating Spec fPL compared to histopathology, the overall sensitivity (ability to detect pancreatitis) was 67% (100% in cats with moderate to severe pancreatitis). The specificity (ability to rule out pancreatitis) had an overall specificity of 91%. In a more recent study, sensitivity and specificity were approximately 80% using a cutoff of 5.4ug/L.

Sensitivity is speculated to be lower for chronic pancreatitis than acute pancreatitis as histologic lesions typical for chronic pancreatitis, such as pancreatic fibrosis and atrophy, are not expected to be associated with release of pancreatic enzymes. Other conditions of the exocrine pancreas, such as pancreatic neoplasia, pancreatic abscesses and pancreatic pseudocysts, may be associated with elevated Spec fPL concentrations if concurrent pancreatic inflammation is present. With these limitations in mind, I would typically base a diagnosis of pancreatitis on an elevated Spec fPL concentration, in conjunction with consistent ultrasonographic findings, supportive clinicopathology results, and historical and physical examination findings.

Evaluation of serial measurements of serum fPL concentrations to monitor progression in individual animals remains controversial, as there is considerable inter-individual variability. For this reason, I do not rely on individual measurements to determine the severity of the pancreatitis. And only consider large increases or decreases (2-3x fold change) indicative of a change in the disease status, with smaller changes assumed more likely to reflect biologic variation. I typically repeat a follow-up Spec fPL within 2 weeks of the initial serum lipase assay to monitor response to therapy. However, I rely more heavily on the patient's clinical response (resolution of clinical signs) as an indication of response to therapy.

The SNAP fPL is a semi-quantitative, point-of-care assay based on the same methodology as the Spec fPL. Whilst there are no validation studies, the manufacturer (IDEXX laboratories) indicates that there is 92% agreement between the SNAP fPL and Spec FPL when values are within reference interval and 82% agreement when values exceed the reference interval. I use the SNAP fPL as a diagnostic rule out, and confirm abnormal results with a quantitative Spec fPL assay. Recently a newer catalytic lipase assay (DGGR) was validated for use in diagnosing feline pancreatitis. Initial studies indicate substantial

agreement between the 2 lipase assays, with the DGGR assay having the potential advantage of being a more cost effective alternative, which may prove beneficial for serial assessments.

VB **Vanessa Barrs:** I find the feline specific pancreatic lipase assay very useful for diagnosis of pancreatitis in combination with other clinical information including abdominal ultrasonography. I do not use it as a stand-alone test. I find both the SNAP fPL and Spec fPL assays useful. For an acutely unwell cat where pancreatitis is on the differential diagnosis list, the SNAP fPL assay is a useful cage-side test to help rule out pancreatitis from the differential diagnosis list. It's also useful in an emergency setting where other diagnostics such as abdominal ultrasound or Spec fPL may not be instantly available. An example that springs to mind is a patient that presented to our emergency clinic after-hours last week with acute collapse, hypothermia, hypotension and abdominal pain. A strongly positive SNAP fPL was supportive of a diagnosis of acute pancreatitis, as were the results of initial CBC, biochemistry and abdominal radiographs. Treatment was initiated for suspected acute pancreatitis, and this diagnosis was subsequently confirmed with consistent changes on ultrasonography and a Spec fPL of > 50 µg/L.

The DGGR-lipase (1,2-o-dialuryl-rac-glycero-3-glutaric acid-(6'-methylresorufin) assay that Amy referred to seems to be another promising serum biochemical test to aid in the diagnosis of feline pancreatitis. It is important to ask your lab by what method they are measuring lipase in serum biochemistry panels. The DGGR-lipase assay is a colorimetric assay in which the formation of methylresofurin, a blue chromophore, is measured after cleavage by serum lipase. The results of other biochemical assays for lipase determination, e.g. the 1,2-diglyceride assay do not appear to correlate well with pancreatitis in cats. We are planning on including the DGGR-lipase assay in the Veterinary Pathology Diagnostic Service serum biochemical profiles at the University of Sydney.

KB **Kath Briscoe:** Like Vanessa and Amy, I use fPL to help confirm the diagnosis of pancreatitis. In addition, if the specfPL is normal and the abdominal ultrasound shows no evidence of pancreatitis, even if clinical signs are consistent with pancreatitis, I would say that pancreatitis is effectively ruled out. I think it is really important to remember that in the case of pancreatitis, what is considered the gold standard is histopathology, but even that is not a 'perfect' test. Unless you remove the entire pancreas and perform histopathological evaluation on the entire pancreas, you can never say with 100% certainty that the cat doesn't have pancreatitis, so when we use ultrasound and the SNAP or Spec fPL we are using them as non-invasive tools to diagnose pancreatitis as best we can at the current time.

With respect to performing serial measurements of SpecfPL to monitor pancreatitis, I tend to do this on a case-by-case basis. For example, if I have a young cat that presents with vomiting, abdominal pain and has consistent clinicopathologic and ultrasonographic findings of pancreatitis, is treated for the disease with the care discussed in this article, and recovers well, I wouldn't necessarily repeat the fPL. However, if the patient wasn't recovering as I would expect, or if there is recurrence of signs, then I would repeat it. The other situation that I use an fPL is in the situation of chronic pancreatitis to get a baseline measurement. The particular case I am thinking about is an older cat who has repeated episodes of vomiting and inappetence. He came in for assessment during one of these episodes and his fPL was around 25ug/L, so it was consistent with a diagnosis of pancreatitis and he had ultrasonographic changes suggestive of the acute disease. On ultrasound, there were also changes suggestive of a more chronic process, so when he was well again I repeated his fPL and it was around 10ug/L. This level is still consistent with pancreatitis, but he didn't have any clinical signs of illness, so I now use this as a baseline for whether or not he is having an acute episode of pancreatitis or if it is likely something else contributing to his illness. There is no evidence in the literature to support the use of an fPL in this way, but it seems to work for this cat! As Amy said, in theory cats with chronic pancreatitis should have more fibrosis than inflammation resulting in release of lipase into the blood stream, so the utility of serial measurements in this regard is questionable.

AH **Andrea Harvey:** Like my colleagues I do find measuring Spec fPL useful, but usually use it in conjunction with ultrasound. I consider a high Spec fPL to be diagnostic for pancreatitis, however the main reason that I would not use this as a stand-alone diagnostic test is because of the frequency of concurrent disorders with pancreatitis, and without combining this with abdominal ultrasound, concurrent disorders may be missed. Having said this, in referral practice I obviously tend to see the more severe or recurrent cases, and if I had a case with a high Spec fPL and very mild clinical signs that responded well to symptomatic treatment, then I wouldn't say it was critical to do abdominal ultrasound in this scenario. However, if the cat had other clinical or laboratory findings suggestive that concurrent disease could be present, if the cat had severe or recurrent clinical signs, or was not responding well to symptomatic treatment, then abdominal ultrasound would be indicated. I don't routinely monitor Spec fPL in cats with pancreatitis, although I think it would be very interesting to! However, currently in most cases this would be difficult to justify. Where I do perform serial monitoring is in cases where the Spec fPL has been extremely high, and where I may be using a specific treatment either aimed at the pancreatitis or for a concurrent disease such as cholangitis, IBD or diabetes mellitus, using treatments such as antibiotics, dietary management, insulin, corticosteroids or cyclosporine. In this situation I find it can be useful to know what effect the treatment is having on fPL, and this could influence management in some situations, depending on whether fPL is reducing or increasing with treatment. A couple of examples of cases in where serial monitoring appeared to be useful was firstly the case example that I mentioned in Question 10, the difficult to control diabetic cat on the cyclosporine treatment. A second case was my own cat which had pancreatitis with, again, a very elevated Spec fPL > 50g/L but with presenting signs more suggestive of IBD (diarrhoea). He was not responding to symptomatic and dietary therapy, and although I did not take intestinal biopsies I suspected IBD and opted to start corticosteroids. He had a dramatic response to this, and of course it is hard to know if that improvement was due to improvement in pancreatitis, IBD or both. Monitoring fPL gave me an idea of what effect the corticosteroids were having on the pancreatitis, and this did drop with treatment, giving me confidence to continue the corticosteroids.

12. DO YOU FIND SERIAL ULTRASOUND EXAMINATIONS USEFUL IN MONITORING PANCREATITIS CASES?

AL **Amy Lingard:** I always perform abdominal ultrasonography as part of our diagnostic investigation in any cat where pancreatitis is considered a possible differential. However, I would uncommonly perform serial ultrasound examinations to monitor the progression of pancreatitis. Circumstances in which I would repeat abdominal ultrasonography include patients that acutely deteriorate, and in patients where there is a concern regarding the development of secondary complications, such as extra-hepatic bile duct obstruction or pancreatic abscess.

Ultrasonographic changes associated with pancreatitis include hypoechogenicity of the pancreas (or less commonly hyperechogenicity due to fibrosis), pancreatic enlargement, irregular pancreatic margins, hyperechoic peri-pancreatic mesentery and fat,

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and/or a corrugated duodenum. In my experience, peri-pancreatic fluid, pancreatic pseudocysts and pancreatic calcification are infrequently seen. There is no significant association between pancreatitis and pancreatic duct width, or the ratio of pancreatic duct width to pancreatic thickness; they are, however, significantly associated with age.

In some pancreatitis cases there will be no detectable sonographic abnormalities. Studies have shown abdominal ultrasonography has a sensitivity of 24% to 67% for detecting pancreatitis, which in real terms means that 33% to 76% of cats with pancreatitis have no sonographic changes detected or that the pancreas cannot be visualised. In my experience, abnormal ultrasonographic findings are reasonably specific for pancreatitis, meaning that a cat with compatible historical and clinical signs and pancreatic sonographic changes is very likely to be correctly diagnosed with pancreatitis.

The low reported sensitivities of ultrasonography probably partly reflect its limited usefulness for detection of chronic disease, which in many low-grade cases can only be detected by histologic examination. Certainly the sensitivity of ultrasonography has been shown to increase with increasing severity of pancreatitis as determined histopathologically. Interestingly, a recent study found that ultrasonographic changes were not associated with outcome (Stockhaus et al, JAVMA 2013). And another study found that the agreement between pancreatic ultrasonography and serum lipase assays was only fair (Oppliger et al, JAVMA 2014).

Vanessa Barrs: It's an interesting question – there is very little information published about the utility of serial ultrasonography in cats after diagnosis of pancreatitis. I personally do not do serial ultrasound examinations unless the cat is not responding clinically to management for pancreatitis or, as Amy has outlined above, if the cat has had a suspected relapse of pancreatitis. Although the sensitivity of ultrasonography is reported to be widely varied, I think, in general, we are getting 'better' at using ultrasonography to diagnose severe/acute pancreatitis. In a recent study by Williams and others in The Journal of Veterinary Internal Medicine, using elevated fPLI as the gold standard, ultrasonography was 84% sensitive in detecting pancreatitis. Specificity of ultrasonography was only 75% in that study, so I am mindful that if the clinical presentation doesn't fit the ultrasonographic diagnosis, I need to consider the possibility of a 'false positive' result, and continue investigating.

Andrea Harvey: I don't routinely use serial ultrasonography to monitor pancreatitis cases; however, I do utilize this if a cat is not improving with therapy as I would expect. The reason for this is several fold. Firstly, in a case of severe pancreatitis if an owner is getting close to giving up and difficult decisions need to be made, repeat ultrasonography can provide some guidance as, if there were significant ultrasonographic improvement, it would give me more confidence that the cat was going to improve and just needed more time to do so, whereas if there was worsening ultrasonographically, I would be more worried prognostically. Secondary, if there was any suspicion of pancreatic neoplasia, repeat ultrasonography can provide some guidance. I had this experience with one of my own cats that I thought had severe pancreatitis (he had had pancreatitis a couple of years before this episode), but was not improving, and on ultrasound the pancreas was very enlarged and getting worse with time and beginning to result in significant extra-hepatic biliary duct obstruction. These ultrasonographic changes eventually guided the decision for euthanasia and on post-mortem examination he was found to have a pancreatic carcinoma. The other situation that serial ultrasound can be useful for is in cats that have been known to have had pancreatitis recently, responded to treatment, but then had a relapse of clinical signs that could have been due to a flare-up of pancreatitis, a concurrent disease, or a combination of both. Reassessing ultrasonographic changes in these cases can sometimes help to guide therapy.

Kath Briscoe: The only time I use ultrasound for serial monitoring of cats with pancreatitis is if I am worried that they are getting worse despite my interventions. The case I am thinking of is a cat that, we think, had acute necrotizing pancreatitis with a large, hypoechoic pancreas on ultrasound, a markedly elevated SpecfPL and a small volume abdominal effusion at the time of initial evaluation. Also on ultrasound there was an area of the pancreas which we were concerned was hypovascular and necrotic. The cat just didn't do well initially, and so we repeated the ultrasound about 3 days after starting treatment to evaluate whether the pancreatic changes were significantly different from the initial evaluation. Our thinking here was that if the area we were concerned was necrotic had increased in size, or if there was evidence of progression of disease, then we might need to take the cat to surgery (something I would only do if REALLY necessary). In that cat we were able to see that the area we were previously concerned about now had evidence of vascular flow within it, and that the liver now had changes consistent with lipidosis, so rather than it being a worsening of the pancreatitis, it was the development of a complication of the pancreatitis that was resulting in the cat not improving as we would expect.

13. WHAT OTHER DISEASES DO YOU COMMONLY SEE WITH PANCREATITIS AND HOW DO THEY INFLUENCE YOUR MANAGEMENT?

Amy Lingard: A diagnosis of pancreatitis should always be considered in the potential context of a wider disease picture. It is important that a positive Spec fPL and/or consistent ultrasonographic findings not be considered an endpoint to the diagnostic evaluation, as many cats with pancreatitis (especially chronic pancreatitis) often have concurrent disease.

I often see cats presenting with concurrent IBD, less commonly with neutrophilic cholangitis, and sometimes with both conditions. Pancreatitis may also trigger hepatic lipidosis; whilst other diseases such as diabetes mellitus may be complicated by pancreatitis. In most situations it is unclear which disease occurs first and what role the initial disease plays in the pathogenesis of the other. Regardless, concurrent disease can influence the clinical progression of pancreatitis, and in some cases may alone be associated with an uncertain prognosis. Therefore, diagnosis and management of both pancreatitis and concomitant conditions are critical to a successful outcome.

In addition to supportive therapy for pancreatitis (discussed elsewhere in this article), for cats with concurrent IBD I would consider the addition of metronidazole, oral corticosteroids, parenteral cobalamin and an appropriate diet, such as a highly digestible, novel protein or hydrolysed diet. I tend to stage my approach to therapy, as many cats will respond to dietary manipulation alone. In those requiring therapeutic intervention, I would initially trial parenteral cobalamin and metronidazole, followed by prednisolone 1-2mg/kg once daily orally, tapering every 3-4 weeks once a response is achieved. In cats with concurrent diabetes mellitus, or those considered at higher risk of developing diabetes mellitus (e.g. Burmese, overweight), I may elect to use budesonide, a local acting corticosteroid; though some systemic absorption may still occur. I will sometimes use chlorambucil or cyclosporin to manage severe inflammatory bowel disease.

Antimicrobial therapy should be used in cats with concurrent neutrophilic cholangitis, as it is considered to have an infectious aetiology. Antibiotic selection should ideally be based on *in vitro* susceptibility testing. I typically recommend empirical therapy with amoxicillin-clavulanate (or ticarcillin-clavuanate) and metronidazole for 4-6 weeks, with the addition of a fluoroquinolone in more complicated cases. I frequently use ursodeoxycholic to promote bile flow and provide cyto-protection. Vitamin K supplementation may be indicated due to biliary obstruction which may impair the absorption of fat soluble vitamin K; and I would always administer vitamin K at least 24hrs prior to the collection of hepatic aspirates or biopsy. Anti-oxidants, such as S-adenosylmethionine (SAMe) may also be beneficial. Interestingly, in human medicine there is compelling experimental and clinical evidence that oxidative stress may also play an important role in chronic pancreatitis. Similarly for cats with concurrent hepatic lipidosis, I would institute ursodeoxycholic acid, vitamin K and anti-oxidants. In some cholangitis cases, surgical intervention may be required.

Cats with pancreatitis can become insulin resistant and develop transient or permanent diabetes mellitus. I commence most cats with mild hyperglycaemia (> 12 mmol/L) on a low dose of glargine (Lantus®) insulin (0.5-1IU/cat) to reverse glucose toxicity and potential beta cell damage. Insulin requirements can vary as a result of waxing and waning of the severity of the pancreatitis, so blood glucose levels need to be monitored closely.

Parenteral cobalamin may be indicated in some cats with chronic pancreatitis due to a lack of pancreatic intrinsic factor. Cats that develop exocrine pancreatic insufficiency secondary to chronic pancreatitis should receive pancreatic enzyme supplementation.

Vanessa Barrs: The 2 most common concurrent diseases that I encounter in cats with pancreatitis are diabetes mellitus and chronic enteropathies, especially lymphocytic-plasmacytic enteritis (IBD), and sometimes low-grade alimentary lymphoma, which I suspect is due to malignant transformation of pre-existing IBD in some cats. Like Amy, I diagnose inflammatory liver disease less commonly than enteropathies in cats with pancreatitis. Clues for the presence of concurrent neutrophilic cholangitis/cholecystitis on ultrasonography include gall-bladder wall thickness of > 1 mm combined with the presence of echogenic material in the gall-bladder and dilation/tortuosity of the common bile duct. Culture of bile will confirm a diagnosis, but ultrasound-guided cholecystocentesis should be performed with caution by a skilled operator.

My approach to the treatment of concurrent IBD and inflammatory liver disease is similar to Amy's. As discussed elsewhere, provision of enteral nutrition (NO or oesophagostomy tube) is important in anorectic patients or where there is concurrent hepatic lipidosis. I do see a fair number of Burmese cats with pancreatitis and IBD. Because of their genetic predisposition to developing diabetes mellitus (DM), I also avoid corticosteroids in these patients. Given that systemic absorption still occurs, I tend not to use budesonide. Cyclosporine is my first-line therapy in these patients, but I only use it if IBD is confirmed histologically on gut biopsies. Cyclosporine can work well as sole therapy in these patients, and I have not seen complications from severe immunosuppression, such as toxoplasmosis, when it is used as sole therapy and not given concurrently with prednisolone. To reduce the risk of complications from immunosuppression, if I use cyclosporine I always perform trough levels 2 weeks after starting therapy to ensure that the cyclosporine levels are not too high, as can occur in some cats (1 mL EDTA blood sample collected just before the next dose of cyclosporine is due, aiming for a trough level of 250 – 500 ng/mL).

In cats with acute pancreatitis, the development of systemic inflammatory response syndrome (SIRS) and multi-organ dysfunctions syndrome (MODs) is common. In these cases pleural and abdominal effusions commonly develop, and stabilization needs to include supportive therapies such as pleural space drainage, nasal oxygen etc.

Kath Briscoe: Like Vanessa and Amy, IBD and neutrophilic cholangitis are frequently associated with pancreatitis, with IBD occurring far more frequently than neutrophilic cholangitis. My first line of treatment for cats with IBD is dietary therapy, with or without prednisolone, and if that doesn't control the signs then I will usually add in cyclosporine or chlorambucil. If I have a cat that I don't want to use prednisolone in, then I usually use cyclosporine as my first line therapy. As already discussed, for neutrophilic cholangitis, treatment with antibiotics (ideally based on culture and sensitivity) is indicated. The other common disease is hepatic lipidosis, and to discuss the management of this we really would need a whole issue! In short, enteral nutrition is key, as well as managing the complications associated with hepatic lipidosis such as vitamin K deficiency causing coagulopathies, vomiting, taurine deficiency etc. Amy has already discussed the use of UDCA and SAMe, which are also indicated.

The times that I see pancreatitis associated with diabetes mellitus are when the patient presents in DKA, at the time the patient is initially diagnosed with diabetes mellitus (the owner presents the patient for assessment of the vomiting rather than the PU/PD, weight loss and polyphagia), in acute deterioration of the diabetic patient, and in transient diabetics. As Amy has already touched on, the key to these cases is to reverse the insulin-resistance induced by pancreatitis and manage the hyperglycemia with exogenous insulin. Typically, I would use insulin glargine or detemir, but in severe cases, I would use regular crystalline insulin, especially for DKA patients.

The other conditions which I frequently see associated with pancreatitis, though not directly linked, are 'old cat' diseases, like cardiac disease (e.g. hypertrophic cardiomyopathy) and chronic kidney disease. The reason that I think it is important to discuss these here is that it can sometimes change our management a little. For example, if you have a patient with known cardiac disease and pancreatitis, you may want to be a bit careful with IV fluid therapy so as to avoid fluid-overloading them; for a patient with chronic kidney disease and pancreatitis, you might be more likely to start an anti-ulcer drug such as omeprazole, as they have 2 concurrent diseases which predispose to GI ulceration. Also, if a cat with CKD develops pancreatitis, they are likely to get dehydrated more rapidly as they can't concentrate their urine, so they may be more critical when they present. I think it is really important, especially when treating older cats, to remember that more often than not we have to manage multiple diseases at once and 'juggle' our treatment.

Andrea Harvey: I fully agree with all of my colleagues' comments on this question and don't have much to add to their comments. The only other aspect that I will raise is dietary management. If you believe that a moderately low fat diet may be important in pancreatitis, it becomes a challenge to know which is the most appropriate diet to use when a cat has concurrent IBD and you may want to use a single source protein or hydrolysed protein diet, which aren't low fat diets, or in a cat with concurrent diabetes mellitus where you want to use a high protein low carbohydrate diet, which also isn't low fat. I don't have any answers here but it is just something to think about, and of course we don't know whether low fat diets are important in cats with pancreatitis or not. In these situations I try to tailor dietary management to the individual according to their clinical response.

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PERSPECTIVE 113

AUSTRALIA'S INVOLVEMENT IN LIVE ANIMAL EXPORTS IMPROVES ANIMAL WELFARE STANDARDS & STANDARD OF LIVING IN IMPORTING COUNTRIES

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Download Comments on Perspective 107 here

In a recent article in *Control and Therapy,* Perspective No. 107 (Issue 275, June 2014), emotive language and dated images unrelated to current live animal export practices were presented in an effort to carry an argument that live animal export from Australia is unethical. One of the most shocking images was of a man surrounded by recently beheaded carcasses with a long blade raised above his head, the handle grasped in both hands, obviously about to attempt to severe the neck of a buffalo calf. The caption stated 'Sadistic slaughter following starvation and neglect'. This image is actually from the *Himalayan Times*, where the original caption stated 'Hindu devotee slaughters a buffalo as an offering to the Hindu goddess Gadhimai in Bariyapur village, Bara district, some 70 km south of Kathmandu, on November 24, 2009. Up to a million Hindu devotees gathered November 24 in a village in Nepal to witness the slaughter of hundreds of thousands of animals in a mass sacrifice that has drawn widespread criticism. Worshippers travelled long distances, many coming from neighbouring India, to attend the 2-day festival, which honours the Hindu goddess of power and takes place once every 5 years in southern Nepal. *AFP*. 24 November, 2009.' Australia does not export live animals to Nepal, and whilst this image and the festival it depicts are unacceptable to those of us engaged in improving animal welfare, it is irrelevant to the issue of live exports from Australia, except that it emphasises the importance of Australia's engagement with other countries to work collaboratively to improve animal welfare.

My direct involvement in live export is limited to the export of cattle to Indonesia so this paper will be largely limited to that particular market, which is, however, our largest live cattle market (Dearth et al., 2014; ABARES, 2011). This discussion will address the issues raised by Perspective No. 107 and respond with an objective, factual assessment of: the importance of live cattle export to the viability of the cattle industry and cattle welfare in northern Australia; the limited current alternative market pathways to live animal market destinations for northern producers; the importance of live cattle imports to employment and the efficient use of by-product feedstuffs in destination markets and their reliance on wet markets; and

Figure 1. The Gadhimai Hindu festival in Nepal – a long way from the live export of Australian livestock.

the improvements achieved in animal welfare in these markets directly because of Australia's involvement in them.

The live cattle trade remains critical to the viability of the northern Australian cattle industry, and is an important contributor to Australia's export earnings. The importance of export earnings is unrecognised in gross measures of economic activity such as gross domestic product (GDP), and only export dollars maintain and improve the Australian mean standard of living – internal economic activity only recirculates the existing pool of funds. During each year of the three year period 2009/10 to 2011/12 inclusive, live exports of all cattle, sheep, goats and buffalo were worth $1 billion, which decreased by approximately 20% in 2012/13 to $790 million due to import restrictions in Indonesia, and interruptions to the supply of sheep to some Middle East destinations (Deards et al., 2014).

In 2013 $258 million of live cattle were exported from the Northern Territory, with Indonesia taking live cattle to the value of $203 million (Deards, et al., 2014). During that year $164 million of live cattle were exported from northern Western Australia, of which Indonesia took cattle to the value of $71 million (Deards, et al., 2014). During the 2013/14 financial year, total live cattle exports exceeded $1 billion for the first time (Beef Central, 2014). Whilst these are substantial inflows of export revenue to Australia, they only represent a small proportion of the total value of the large Australian beef industry (7% of total cattle turnoff in 2013; Deards et al., 2014). However, the northern Australian beef industry is highly reliant on live exports due to the transport distances and costs involved with selling cattle to southern abattoirs, grass fatteners or feedlots. Further, high grade *Bos indicus* cattle are most suited to the top end grazing country due to their resistance to ticks (Piper et al., 2010; Piper et al., 2008) and heat (Rahul Behl Jyotsna Behl Joshi, 2010; Carvalho, 1995; Khub Singh Bhattacharyya, 1991; Ledger, 1959), and their ability to better utilise a higher fibre diet compared with *Bos taurus* cattle (Essig, 1995). *Bos indicus* cattle have lower mean meat quality for a given body weight compared with *Bos taurus* cattle (Gonzalez et al., 2014; Schutt et al., 2009) and they therefore sell at a discount into southern markets, but, *Bos indicus* cattle are ideally suited to the hot, humid conditions of Indonesia. The reliance of the northern beef industry on live exports to Indonesia was clearly illustrated by the effects of the suspension of the live cattle trade to Indonesia on June 8th, 2011. The trade suspension resulted in the retention of increased numbers of cattle on northern stations due to the lack of alternative viable markets (Keogh, 2013). The retention of most animals from an entire calf drop was followed by a failed wet season in 2012/13, which forced producers in many instances to shoot cattle debilitated by hunger (Anon., smh.com.au, 2011). Marketing of cattle in these instances would have involved transport costs in excess of the value of the cattle at their southern destinations. Thus, the sudden suspension of the live cattle trade to Indonesia on animal welfare grounds created a new and extensive animal welfare problem. The unintended consequence of malnutrition created largely by the live export suspension applied to hundreds of thousands of cattle across the top end of Australia.

The financial effects of the suspension of the Indonesian live cattle export trade on the viability of northern Australian cattle enterprises were still being felt in 2013 (Anon., news.com.au, 2013), and they also filtered through to the southern Australian beef industry, through the sale of some cattle from the southern regions of the northern beef industry into southern abattoirs. This coincided with increased sales of cattle from the drought affected regions of central and western Queensland, resulting in a substantial depression of cattle prices not only in the north, but also in southern Australia (Keogh, 2013).

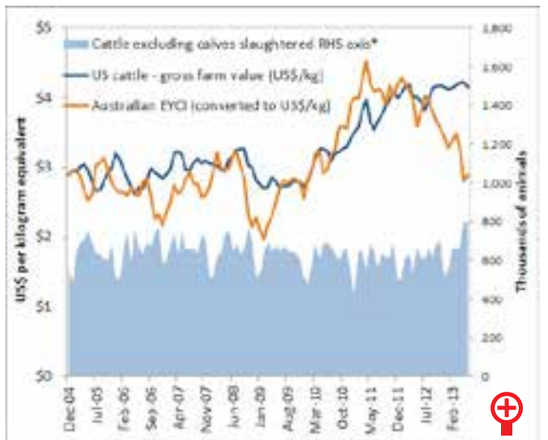

Figure 2. The relationship between Australian and United States cattle prices from the end of 2004 to mid 2013 (Indonesian live export trade suspended June 2011 and 2012/13 was a failed wet season; source, Keogh, 2013).

The longer term effects of cessation of live cattle exports out of northern Australia in terms of the loss of value of cattle due to the imposition of additional costs and reduced marketing options were estimated by Clarke et al. (2007) to be a decrease of 59 cents/kg in the Northern Territory and 50 cents/kg in northern West Australia. Expressed as proportions of original value, these losses in value approximately represent a 33% decrease in the Northern Territory and a 31% decrease in northern West Australia. It logically follows that many northern beef businesses would be rapidly bankrupt by such a severe cut in output price, accompanied by ever increasing input prices. Further, as illustrated by Keogh (2013), cessation of live cattle exports out of northern Australia would depress cattle prices throughout the entire Australian beef industry, both north and south.

Northern Australia is the ideal nursery for breeding young cattle to supply the feedlots of Indonesia in terms of location, climate, and the productivity of the land. The distances to southern outlets compared with the distance of the live export trip to Indonesia is not markedly different for the regions of the top end districts of the NT, and considerably shorter for the north Qld regions in Cape York. However, the travel distances from the Kimberley and Pilbara regions to Indonesia are markedly shorter than southern Australian destinations. Further, the cost of rolling land transport within Australia is markedly more expensive than the cost of floating transport by ship (approximately 4x to 10x; Torian, 2012; Cambridge Systematics Inc., 1995) and these greater land transport costs make the southern movement of cattle inviable under most market conditions.

The climate of the top end of Australia is similar to that of Indonesia and the *Bos indicus* cattle that are suited to northern Australia are also suited to the hot, humid conditions of Indonesia. Fortuitously, the lean carcase of the *Bos indicus* breeds matches the very low carcase fat requirements of the Indonesian market, with traditional dishes based on diced, slow cooked beef, or meat balls (Bakso balls). Conversely, high grade *Bos indicus* cattle do not meet the high meat quality requirements of the domestic Australian market, or those of many of the countries to which we export boxed beef, as effectively as the *Bos taurus* breeds that are well adapted to southern Australia.

The highly seasonal rainfall and relatively low fertility of much of northern Australia precludes the use of most of this country for agriculture other than rangelands grazing. Rumen fermentation is an elegant system for turning human indigestible forages into high quality protein. Further, calves grown to the target weight for export can then turn a variety of human inedible roughages and by-products in Indonesia into additional kilograms of high quality protein. It seems inappropriate for those of us from countries with plentiful, affordable protein, to deny people from countries with dietary protein deficiency the opportunity to convert by-products into high quality protein. The Indonesian feedlot sector not only utilises by-products, it also employs large numbers of people both directly, and indirectly (Leigh, 2013; FAO, 2007) – such as opportunistic sales of hand cut elephant grass harvested from road sides and waste areas.

Thus, breeding young *Bos indicus* cattle on the rangelands of northern Australia and exporting them to Indonesia integrates perfectly with the employment dense, value adding Indonesian feedlot sector. This marketing arrangement is of immense value to both Australia and Indonesia.

Table 1. Distances to cattle market destinations from northern Australia.

Cattle Station Location	Port and distance to port, km	Distance to Jakarta from port, km	Total distance to Jakarta, km	Distance to major abattoir - Brisbane, km (variance from live export trip)	Distance to feedlots - Darling Downs, km (variance from live export trip)
Douglas-Daly	Darwin 223	2720	2943	3370 (+427)	3163 (+220)
Victoria River District	Darwin 601	2720	3321	3395 (+74)	3186 (+135)
Cape York	Karumba 70	3896	3966	2085 (-1881)	1877 (-2089)
Kimberley	Wyndham 100	2539	2639	3703 (+1064)	3497 (+858)
Pilbara	Port Hedland 250	2017	2267	5206 (+2939)	4999 (+2732)

The general effectiveness of research and development investments by Livecorp and Meat and Livestock Australia in reducing mortality rates during shipping to export destinations is illustrated by the mandatory mortality reporting represented in the figure below. Mortality rates of grazing animals on pasture in Australia are generally within the range of 1% to 2%, but can be higher depending on the season, location and scale of the grazing enterprise. Consistently achieving voyage mortality rates below the target range for livestock on pasture in Australia is obviously a sound result.

Figure 3. Livestock export mortality rates 1996 to 2013 (taken from Livecorp, 2014).

With reference specifically to Indonesia, most consignments of cattle either maintain their body weight, or gain weight during the voyage (Thompson, 2000). Clearly, cattle that are stressed by poor animal welfare have reduced feed and water intake, and mobilise body tissue, and therefore lose body weight. Thus, low mortality rates, and maintenance or gain in body weight clearly indicate a high standard of animal welfare.

At present, the supply of electricity in Indonesia is inadequate in terms of the distribution network and consistency for the general population to have widespread access to reliable refrigeration (Leigh, 2013), and there is a lack of cold chain infrastructure (Deards, 2014). In addition, refrigerators represent a substantial investment in relative terms, and there is also cultural resistance to moving away from the purchase of fresh meat through wet markets for same day consumption. As the standard of living of Indonesians improves, local assembly and increased sales of refrigerators suggest this could change (Euromonitor, 2014), but it is fanciful to suggest now that chilled or frozen boxed beef is an alternative means of supplying beef to the majority of Indonesians. Accordingly, most beef from imported Australian cattle is marketed through traditional wet markets (Figure 4).

Figure 4. Distribution of beef from Australian live cattle imports in Indonesia (source Meat and Livestock Australia and Livecorp, 2011, reported in ABARES, 2014).

Using the objective animal welfare standards outlined by the Office Internationale Epizooties (OIE), the review panel, of which I was a member, that evaluated animal welfare of Australian cattle throughout the supply chain in Indonesia during March 2010, observed that animal welfare was generally good, but recommended continued investment in increasing the use of non-lethal stunning at slaughter (Caple et al., 2010). There have been improvements in animal welfare since this review, but readers who are interested in the detailed assessment of animal welfare of cattle in Indonesia during transport from the docks to the feedlots, at the feedlots, during transport from the feedlots to the abattoirs, and at the abattoirs should refer to this Livecorp/MLA report (Independent study into animal welfare conditions for cattle in Indonesia from point of arrival from Australia to slaughter, May 2010). Since the release of that report, and the intervening suspension of live exports, there has been a marked increase in the use of pre-slaughter stunning. Prior to the introduction of the Exporter Supply Chain Assurance System in August 2011 (ESCAS; Australian Government Dept of Agriculture, 2014), approximately 15% of Australian cattle slaughtered in Indonesian abattoirs were stunned, but this has increased to approximately 85% (Anon., Livecorp, 2014). Data on the use of stunning in Indonesia in the slaughter of cattle not

imported from Australia could not be found, but embedding the practice in the slaughter procedures of a given abattoir must inevitably result in its use with all cattle processed through that chain. This is a clear example of improvement in animal welfare standards for all cattle slaughtered in Indonesia, regardless of their origins, that has arisen because of Australia's collaborative work with a cooperative Indonesia. The achievement is all the more noteworthy when we consider that the cattle referred to as 'Australian cattle' by the media and animal activists have actually been purchased by Indonesian companies, and reside in Indonesia, and are therefore only Australian in origin (Fozdar and Spittles, 2011). Further, the Australian live export industry continues to invest in improvements in animal welfare in importing countries and it is inevitable that these benefits will accrue to local cattle and cattle imported from other countries over time. For a more comprehensive review of the improvements in animal welfare that have occurred as a consequence of the adoption of ESCAS by importing countries, see 'Submission by the Australian Livestock Industry to the Review of the Exporter Supply Chain Assurance System (ESCAS), July 2014'.

Figure 5. Indonesian carriers delivering cattle from port to feedlots. The drivers have a strong incentive to ensure the cattle are well cared for in transit since they must pay for any losses.

Legislation proposed by the RSPCA to make the immediate reporting of animal cruelty mandatory is a positive step towards improving animal welfare at the expense of the fund-raising strategies currently employed by some animal activist groups. Previously, the withholding of evidence of animal cruelty, in some cases for several months, has indicated that such activist groups are more interested in timing the release of such evidence to maximise its impact, and therefore its fund-raising capacity, rather than addressing animal cruelty as quickly as possible. It is unlikely to be a coincidence that of the three animal activist websites I checked in the course of writing this article, all had conspicuous 'Donate' buttons on their home pages.

It is logical for those of us genuinely committed to improving the welfare of animals from all over the world, not only those with Australian passports, to support the enhancement of animal welfare outcomes in destination countries through continued collaborative investment of funds and expertise. We should be guided by facts and not be forced to neglect the welfare of animals overseas through the emotional manipulation of groups fundamentally opposed to all forms of animal agriculture. The most effective strategy is to be vigilant and engaged with live export destination countries.

Figure 6. Cattle in an Indonesian feedlot bedded on sawdust which is changed every 3 to 4 days, with the residue utilised in compost. Note the byproduct based ration with palm kernel extract and casava prominent.

Figure 7. The welfare of cattle in Indonesian feedlots is of a high standard – cattle from Australia in Indonesian feedlots become very calm.

GREAT RESOURCE LINK SENT IN BY MARTIN WHITEHEAD, UK

E. mailto:martincnvets@gmail.com

CLINICAL REASONING TOOLKIT

https://improvediagnosis.site-ym.com/?ClinicalReasoning

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REPLY TO PERSPECTIVE NO. 113

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Dear Editor

Thanks for the opportunity to respond to Dr Paul Cusack's paper. I commend Dr Cusack (Director of Australian Livestock Production Services) for his effort however I stand by my article and note that there was little in my essay (Perspective 107 June 2014 Issue 275) that he could refute. He referenced the use of a photo (Figure 1, Perspective 107) that I believe appropriately typifies the attitude towards animals in middle eastern and other countries of the world, the likes of which we would never witness in Australia, and the equivalent of that seen in Karachi (2012) and most recently at the Muslim Feast of the Sacrifice (Eid al-Adha; October 2014). I found the photo shocking and Dr Cusack did agree and added ***'...it emphasises the importance of Australia's engagement with other countries to work collaboratively to improve welfare'***. The caption is correct as the emaciated animals were being sadistically slaughtered (beheaded). The brutal slaying (October 2014) of a Gaza bull below (Figure A[1]) is no less shocking. Australia exports cattle to Gaza.

Figure A

Dr Cusack commented my images were 'dated' (2003) and unrelated to current live animal practices. Dr Cusack may not be aware that many of the ships engaged in this long haul trade are mostly over 20 years old and very little has changed. Many of the disease processes still exist as they did at the start of the live export trade decades ago. The Salmonella/inanition complex in export sheep and heat stress in exported sheep and cattle are prime examples. Machinery breakdowns continue to occur regardless of better back-up systems. Ventilation and adequate air changes, and many other aspects of the whole seagoing venture, are always at risk of failure.

The MV 'Bader III' (Figure B[2]) was built in 1977 and has had 6 name changes. 4,179 sheep died (with most deaths attributed to hyperthermia) aboard the 'Bader III' in August 2013 whilst in the Persian Gulf on a 33 day voyage from Australia. Other examples of old ships are the MV 'Maysora' built in 1988 that has had 3 name changes, and the MV 'Barkly Pearl' built in 1993. The 'Barkly Pearl' was the focus of attention after sailing to Mauritius in October 2012 with a consignment of slaughter cattle including female cattle that had been certified not pregnant but a number of which started giving birth onboard and others subsequently found pregnant on arrival. It is illegal to slaughter pregnant cows in Mauritius. 65 of the consignment died mysteriously ashore with the importer alleging foul play, and those that survived to the point of slaughter were roped and hoisted prior to having their throats cut alive.

Figure B

The 'Barky Pearl' was also the subject of a South African documentary in 2012 with video footage that a South African judge reviewed in 2013 as demonstrating that 'cruelty' is prevalent and that has to be resolved in the future'.

Figures C[3] and D[4] are reasonably recent photographs of cattle on a long haul, again demonstrating the immense problems these animals face when exposed to long sea voyages.

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Figure C

Dr Cusack's paper reveals a pecuniary interest and attempts to provide economic persuasion to justify live export to Indonesia. No amount of academic economic argument can annul the cruelty many animals experience at sea and up to the points of slaughter. Furthermore, after having found many instances of irregularity in live export data, reliance on any biostatistics related to this trade must be viewed with caution. If individual Australians choose to invest in a high risk trade that involves the cruellest exploitation of live animals with no dependable protective measures; a trade that has no history of conviction for non-compliance, but does have a history of extremely poor governance, then the industry must expect criticism.

Figure D

The records show Dr Cusack being a co-author of a Final Report: Independent study into animal welfare conditions for cattle in Indonesia from point of arrival from Australia to slaughter dated May 2010: For Public Release (Prepared for: Meat & Livestock Australia and LiveCorp). The report stated *'… 29 cattle were observed slaughtered from 11 abattoirs.* Numerous issues with slaughter were detailed in the report and these same issues were still evident in 2011 in the footage used for the Four Corners program, A Bloody Business. MLA and exporter LiveCorp were aware in 2005 of the potential backlash that such footage would provoke, with a report recommending that, as an imperative it have ''a document prepared and a simple and accurate media response in the event of an overseas or Australian media report on slaughter practices in Indonesia'".

With increasing shipments of live cattle to Vietnam and a lucrative future for the same trade back into Indonesia, in the past 3 years MLA figures show cattle exports to Vietnam skyrocketed from 1,500 head initially to 131,000 currently. On 28th June 2013 a government report confirmed Australian cattle sent to Vietnam being repeatedly struck on the head with a sledge hammer. The report states *'In many of the videos the cattle are visibly distressed. The cattle can see slaughter and dressing of other animals, are restrained with ropes, are tied to the floor, hit with sticks and are seen to slip, trip and fall. The only restraint employed is manual restraint using ropes. Cattle are struck on the head with a sledge hammer in an attempt to make them unconscious. Many of the animals are struck several times before collapsing. Some of the animals are tripped and tied to a metal frame attached to the floor before the attempt at stunning is applied. Many of the animals are struck several times before collapsing.'* The investigation found that the cattle shown in the videos were slaughtered in a manner not consistent with World Organisation for Animal Health (OIE) – see www.oie.int/about-us/our-members/member-countries/ recommendations. The investigation concluded these cattle were likely to be some of the cattle that went missing from the Wellard supply chain. Wellard also identified 94 cattle sent to an abattoir not listed in that supply chain in advance of the department's approval.

There is no guarantee Australian animals will not be removed from authorised export supply chains, and there is no guarantee exported animals will receive humane treatment at any point of the supply chain (Figure E[5]).

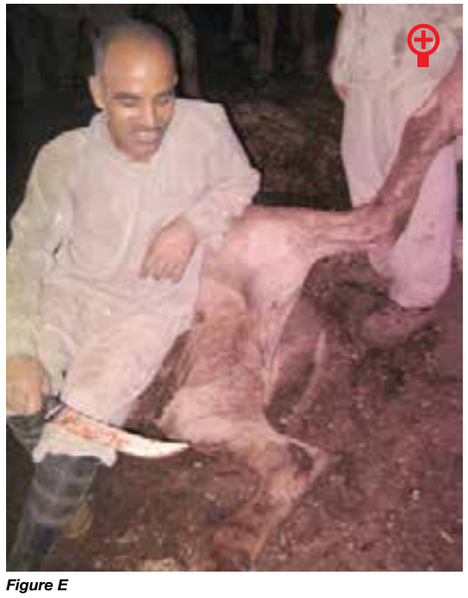

Figure E

These photos and thousands of others including those inserted in Perspective 107 very much relate to current live animal export practices and unequivocally demonstrate live animal export is indeed unethical.

In countries traditionally reliant on the live export trade, rapid growth in personal incomes, urbanisation and access to refrigeration has also contributed to an increase in demand for meat exports. While preferences for live or meat exports vary from country to country, there are more and more markets where live exports can be readily substituted for chilled or frozen meat exports. Australians are largely unaware that lamb carcasses for religious festivals are now also routinely flown to the Middle East. Even the argument that religious requirements give rise to the need for import of live animals is questionable.

What is clear is that overall the demand for meat exports has been consistently rising and is set to continue. For example, imports of sheep meat to the Middle East increased by more than 150% between 1990 and 2013 and overall meat exports from Australia are currently worth 9 times that of live exports. In China, demand for imported beef and sheep meat has been growing strongly due to rapid population growth, rising incomes, changing diets and increasing urbanisation, with Australian beef already accounting for 53% of beef imports and sheep meat imports growing rapidly. In these circumstances, why would this government even consider exporting processing jobs and opportunities by opening up a trade in live animals for slaughter to China?

The generally accepted average, natural attrition rate on Australian sheep farms is 2% to 5% per annum. This loss is mostly due to endoparasites, fly strike, drought, bush fires, floods and the ingestion of toxic plants. On long hauls at sea, the death rate is considered acceptable if < 2% per voyage. The causes of death are not the same as that seen on the farms. On a random sample voyage to the ME 14.1% of the total number of dead sheep died of starvation (inanition), 20.5% died from enteritis (Salmonellosis, Colibacillosis), 54.3% died of pneumonia and 1.3% died from suffocation and 2.1% from trauma. Overall, the main killers are Salmonellosis / Inanition Complex, pneumonia and hyperthermia. Sheep that die in this manner do suffer from their export-related diseases and die at a higher rate than on farm. If there were only 10 voyages per year and a 1.0% mortality rate per voyage then this would amount to 10.0% per annum. In some years the number of voyages has been 2 per month.

If any of the aforementioned is considered 'emotive language' then I make no apology. I, and many of my veterinary colleagues, will continue to use our scientific knowledge and skills for the benefit of society through the protection of animal health and relief of animal suffering in keeping with the principles of veterinary medical ethics.

Respectfully

Capt (Dr) Peter Kerkenezov
Veterinary Surgeon & Master Mariner
Ballina NSW 2478
30th October 2014

[1, 2, 3, 4 & 5] Photos of known origin extracted from the public domain

REPLY TO PERSPECTIVE 113

C&T NO. 5419

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Dr Cusack's contribution is welcomed as his knowledge, expertise and experience in this area are significant. However, as noted by Dr Cusack, the focus of most of his response is the northern cattle industry and, in particular, trade to Indonesia. In this debate, it is necessary to clearly differentiate between bovine and ovine welfare issues and even more importantly, the issues of those animals transported by long haul ship voyages compared to those transported by air or short-haul sea voyages. The majority of Australian cattle are exported to countries in South East Asia by short-haul ship transport. The majority of Australian sheep and in fact, the majority of Australian animals (in terms of absolute numbers) are not transported short haul to Indonesia, but via long haul sea voyages to the Middle East and beyond. Veterinarians on long-haul voyages are employed by the exporters. Veterinarians are not required at all on short-haul voyages. Given the lack of independent veterinary assessment of long haul voyages and lack of any veterinary assessment of routine short haul voyages, Dr Kerkenezov's contribution is particularly relevant, given his maritime and veterinary experience of long and short-haul animal transport by sea.

VALE would like to make the following brief observations in response to Dr Cusack's article:

1. **Northern Australian cattle industry**

   Dr Cusack's assertion that the northern cattle industry must involve live export to survive is entirely correct. The live export industry was a major factor in the demise of the northern Australian abattoirs. This lack of abattoirs made the industry completely reliant on the live export trade and exposed to its vagaries. Indonesia's own stated objective before the trade suspension was to become self-sufficient in beef (regardless or not of whether that was, or is, achievable) and they have also more recently signaled their intention to start importing live cattle from zones of Brazil declared free of foot and mouth disease. The Indonesian market is thus not secure and stable, a fact recognised by the northern cattle industry and highlighted by the recent rapid expansion of the live export cattle and buffalo trade to other SE Asian countries, especially Vietnam (overall welfare status unknown but reportedly 100% stunned slaughter (B Jones, RSPCA Australia, pers comm)).

   The lack of abattoirs also results in significant problems within northern Australia itself. In some areas of the Kimberley for example, it is reported that 1000 cattle are mustered for every 50 that are exported, a 5% turnoff (Joint Select Committee on Northern Australia 2014). The remaining cattle largely have no commercial value (NCV) (Joint Select Committee on Northern Australia 2014). In WA at least, this situation has implications for pastoral and environmental degradation of the rangelands (Novelly and Thomas 2013, Joint Select Committee on Northern Australia

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2014) and also for animal welfare (cattle of NCV are worth less than the fuel and the bullets that may be required to shoot them).

2. **Meat and Livestock Australia's report on animal welfare in Indonesia, 2010**

As Dr Cusack has pointed out, he was a member of the independent group detailed to assess animal welfare in Indonesia in 2010 (Caple et al 2010). Despite the fact that the group and the report made the conclusion that 'animal welfare was generally good', it detailed the slaughter of 29 animals in 11 abattoirs and found:

- there were problems with the Mark I boxes (and more so with their copies), and 17% of animals that went down actually gained their feet on release
- up to 18 incisions were inflicted in slaughter with an average of 4
- interference with eyes and tail twisting practices were noted immediately prior to slaughter once the animal was restrained and cast
- there was an average of 3.5 head lifts in casting prior to slaughter and head lifts were observed to pose a significant risk to animal welfare
- the severity of the falls, classified as mild, moderate or marked was found on average to be moderate
- that tossing buckets of water/hosing water over cast cattle before slaughter occurred
- excessive sensory overload prior to slaughter was observed at busy abattoirs where the opening of the restraint box led to sudden exposure of the cattle to activity on the slaughter floor

It was the slaughter descriptions provided in this report, which flagged the fact that despite over 20 years of Australian involvement in Indonesia, animal welfare had not been sufficiently prioritised by the industry. In addition, the report noted that 'Stunning was observed to deliver the single biggest animal welfare benefit and the general adoption of stunning in the slaughter of Australian cattle in Indonesia should be an aspirational goal.'

The 2011 trade suspension, regrettable as the fallout undoubtedly was, resulted in an increase of stunned slaughter from 15% to 85% in two years i.e. something that could have easily (and painlessly to pastoralists), been achieved over the previous 20 years had there been any real commitment to animal welfare in the live export industry. That stunned slaughter was still regarded as an "aspirational goal" by Dr Cusack and his team in the 2010 report does not reflect well on the industry's commitment to improving animal welfare in importing countries. The "aspirational goal" became reality in less than two years once the animal welfare spotlight was turned up and the trade suspension occurred. It should never have needed this. In addition, to accuse the animal welfare groups of withholding their footage for a few months when the industry had been well aware of these issues for over 20 years is somewhat hypocritical.

3. **Animal welfare at sea**

Dr Cusack rightly points out that the mortality involved in cattle voyages generally, but in short haul specifically, are low with acceptable (but arbitrary) limits of <0.5% for short haul voyages and <1% for long haul. VALE notes that "acceptable" for cattle is not translated to sheep and "acceptable" mortality for sheep is double that of cattle (<2%), presumably dictated by what is possible rather than what is desirable.

However, of concern is that Dr Cusack then compares these voyage mortalities that usually occur over 7-30 days with annual mortality figures for Australian grazing animals. Comparing an average consignment mortality rate for voyages of variable time periods (e.g. 7-30 days) with an on-farm annual mortality rate (365 day period) is problematic. It is not even clear whether the Department of Agriculture calculates their annual average voyage mortality rate as a computed rate (number of deaths/number of sheep) for each trip with an average derived by summing all rates and dividing by number of trips or a weighted average (number of total deaths/number of total sheep of all trips) (Norris and Norman 2012). What can be said is that if the mortality rate that occurs on a voyage of 7-30 days is even a tenth of the mortality rate that occurs on an average farm over 365 days, then more animals die/7-30 day period on board ship than would be expected on that farm for the same given time period. The average voyage mortality for sheep in 2012 was reported to be 0.81% with an average voyage duration to discharge of 22 days (Norris and Norman 2013).

Regarding weight gain onboard as an indicator of good welfare on ships, the reference cited by Dr Cusack (Thomson 2000) does not report that weight gain necessarily occurs. It discusses that some cattle delivered to export depots are clinically dehydrated (e.g. 10% dehydration with bodyweight losses of 12% or more) and that it would be ideal to keep these bodyweight losses below 10%. It reports that "Most exporters hope for a measured weight gain during the shipping phase, which as previously explained is more reflective of gut fill and hydration than a true weight gain" then details the shipping factors that can impinge upon this outcome, including "the true density of the cattle in volumetric terms" and states that "Cattle on ships are relatively crowded". It is important to note that weight gain alone does not indicate that animal welfare is of a high standard (Foster and Overall 2014) and that Dr Thomson, a senior livestock export veterinary officer at the time of his article, did not make this claim, merely commenting that weight gain (rehydration and gut fill) is a better measurement of welfare outcome than mortality rates when the rates are low (Thomson 2000). VALE notes that this reference from 2000, cited in evidence of good shipboard animal welfare, is older than the colour shipboard photographs provided by Dr Kerkenezov and criticised by Dr Cusack as being outdated (2003).

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1. Greene CE, Schultz RD. Immunoprophylaxis and immunotherapy. In: Greene, CE, 3rd Infectious diseases of the dog and cat. Philadelphia: WB Saunders Co, 2006; 1069–1119.

* Vaccine strains of Rhinotracheitis and Calicivirus

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