

C&T

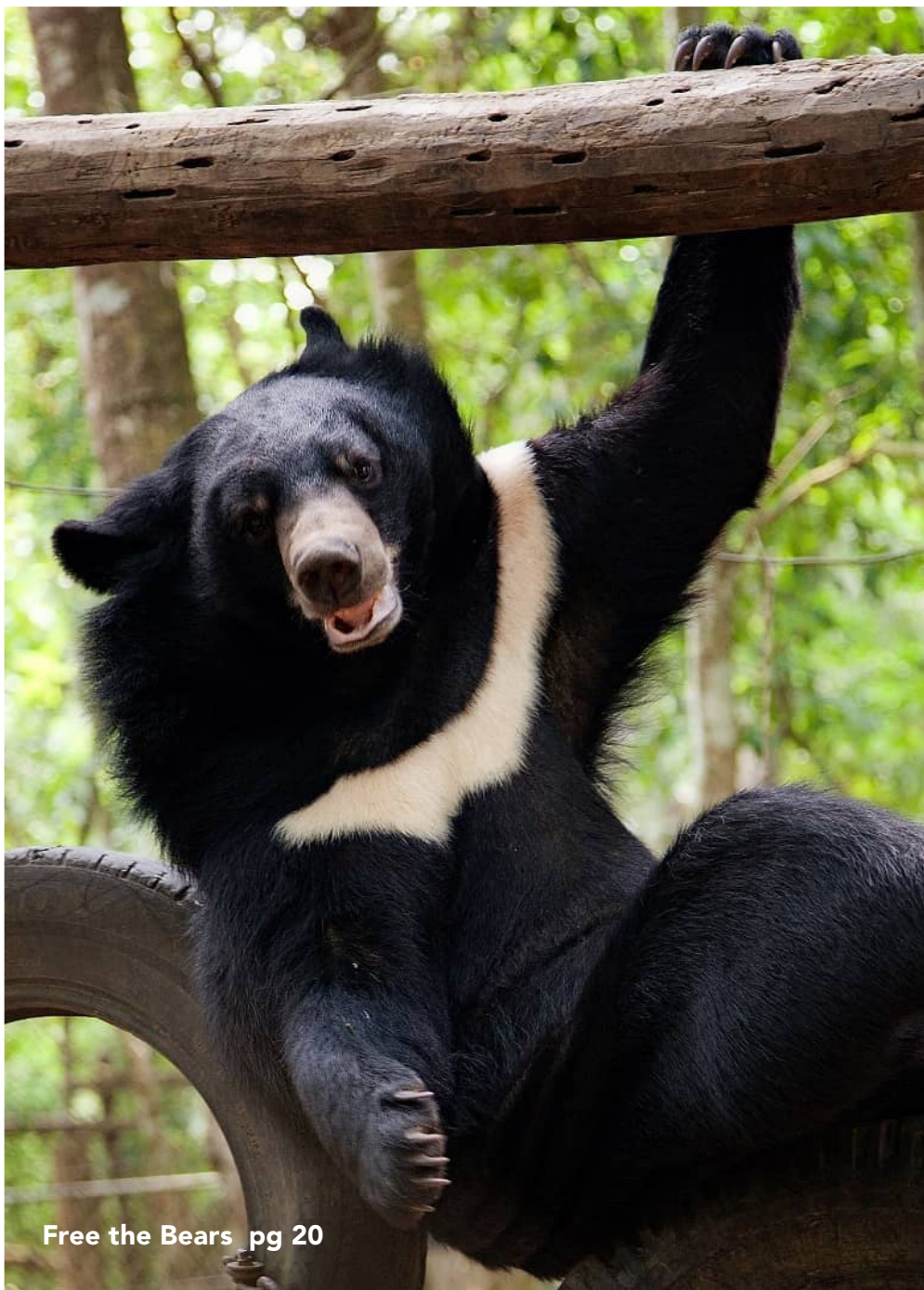


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Control & Therapy Series

Issue 302 | March 2021



Free the Bears pg 20



Radial
Agenesis
pg 5



Inconsistencies in the
Pinkeye (IBK) Story (so far)
pg 45



When a Nasal
Foreign Body is
the Sliver of the
Full Scope pg 17



Dr Claire Adams

Online now



Dr Henry Jones

I hear you guys switched to a mucosal vaccine?



Dr Claire Adams

Yes, we're using Bronchi-Shield 🐾 Oral



Dr Henry Jones

What prompted the change?



Dr Claire Adams

We all agreed the benefit of increased mucosal immunity to protect against respiratory disease was worth it!



BTW, mucosal vaccines are also strongly recommended by WSAVA for high risk environments such as shelters¹ 🏠

Dr Henry Jones

We considered it but it's a hassle to change protocols



Dr Claire Adams

We got the rep in, they sorted out the vaccination protocol and trained our staff. The new protocol worked out to be more cost effective too! 👍



Dr Henry Jones

We're also not sure if our clients would like it... 🤔



Dr Claire Adams

Hmmm...if the flu vaccine came out as an oral mucosal form, how many people would still stick with injections?



Dr Henry Jones



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Issue 302 | March 2021

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'I enjoy reading the C&T more than any other veterinary publication.'

Terry King
Veterinary Specialist Services, QLD

Thank you to all contributors

The C&T Series thrives due to your generosity. If you're reading this and have been contemplating sending us an article, please don't hesitate.

The C&T is not a peer reviewed journal. We are keen on publishing short pithy practical articles (a simple paragraph is fine) that our readers can immediately relate to and utilise. And the English and grammar do not have to be perfect—our editors will assist with that.

Join in—write up that interesting case

C&T authors agree that it is extremely satisfying to see their articles in print and know they are contributing to veterinary knowledge and animal welfare.

Winners

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Prizes

Major Winner: a year's free CVE Membership

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FROM THE DEPUTY DIRECTOR



In thinking of how to compose this editorial, I reviewed what Hugh had written in the last edition of C&T in December 2020. He reflected on the bushfires and pandemic, the trials of the year that was. As I write, many people in

the community have now been impacted by severe floods. If resilience was a tradable commodity, stock value would have to be sky high.

I've listened to interviews with those in flood-affected areas and the term that is repeated over and over is 'community'. People banding together, supporting each other and being empathic and responsive to the needs of others.

Ultimately, this is what the C&T is founded on – a community forum for discussion and sharing. It works because people are willing to share their experiences, failures and successes for the benefit of others and ultimately to enhance the welfare of animals in our care.

Carol Esson's story, 'Free the Bears', page 20 is an incredible example of the power of community. Her insight into the work of the Free the Bears charity is inspiring and moving – international and local people united by a common purpose. It's a great example of the diverse range of opportunities and experiences this profession can offer.

Although Carol's story was awarded the 'Best Visuals' prize, the pictures I couldn't look away from were those accompanying Pete Coleshaw's contribution, 'When a Nasal Foreign Body is a Sliver of the Full Scope', page 17. What a gastric treasure-trove was unearthed! Pete sums up nicely his key pieces of advice following this experience – potentially improving the outcomes of another clinician's patient.

So, a heartfelt thank you to all our contributors, in this issue and the issue 301 before it, for your generosity. For taking the time to share your knowledge and insights to help others and ultimately improve animal welfare. We owe you a debt of gratitude.

Simone Maher



Avian Anaesthesia TimeOnline

6 April-2 May 2021

'Dr Bob Doneley is one of the most helpful, resourceful and efficient teachers I have known - he not only provided excellent course content that was applicable to all vets, but provided excellent videos which are great for those who benefit more from visual aids, like me. Very happy and worth every cent.'

Sabrina Killius, Salisbury
Highway Veterinary Clinic

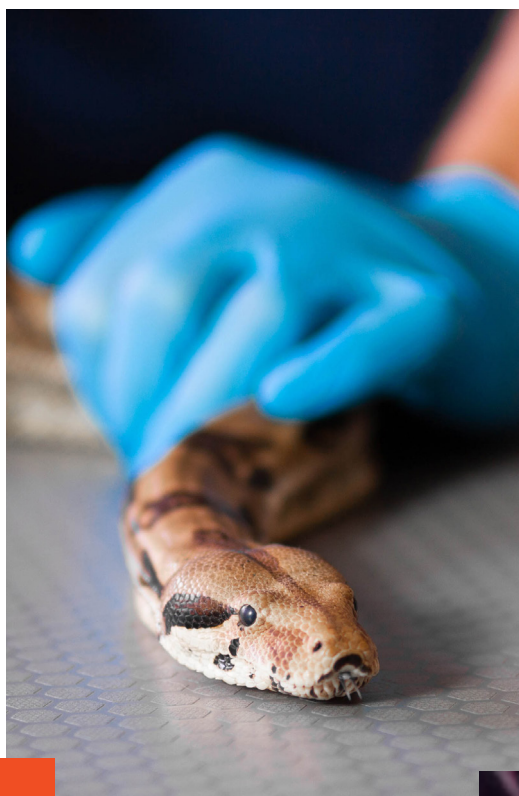


Equine Endocrine Disease TimeOnline

29 March-9 May 2021

'A great summary of complex conditions; it is important to remain up to date with current understandings re EMS and PPID as these are syndromes we encounter so often in general practice; the ambulatory clinician can make a big difference to equine welfare in both the prevention and management of associated laminitis - a big "killer" of horses.'

Clare Williams-Paterson,
Kyabram Veterinary Equine Services



Reptile Medicine TimeOnline

22 March-18 April 2021

'The Reptile Medicine TimeOnline was extremely informative and enjoyable. It has given me a much needed insight and confidence to talk more "reptile like" to the owners of any presenting reptile patients.'

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Brooke Schampers,
Animal Emergency Service



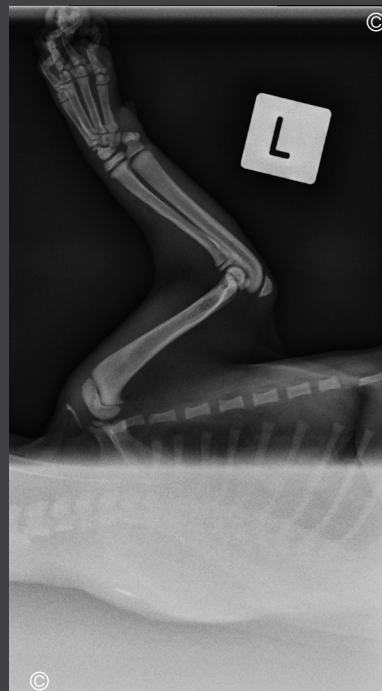
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WHAT'S YOUR DIAGNOSIS?

C&T No. 5869, Issue 301, Dec 2020

Dom is a male neutered 11 week-old-kitten DSH kitten who has some difficulty jumping but otherwise behaves like a normal kitten.

What is your diagnosis and how would you confirm the diagnosis and manage the case?



CONGRATULATIONS TO THE WINNERS!

See the next page for the winning names and replies. Each winner has won a CVE\$100 credit voucher.

Photos courtesy of Jenny Storaker.

REPLIES

ANSWER 1

Marilyn Simon
Albert Animal Hospital
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My diagnosis for little Dom is hemimelia, specifically feline radial aplasia where he has failed to form a right radius.

The diagnosis appears to be confirmed on the two views of the right forelimb where there is no radius to be seen and the ulna is comparatively short and deformed.

I would recommend getting an opinion from an orthopaedic specialist ASAP to see if they can work their magic (not actual magic, but possible reconstructive surgery), a physiotherapist to help with strength and function, pain relief if indicated, consideration of amputation of right forelimb if affecting quality of life and keeping him as an indoor cat to keep him safe.

This little guy is already desexed which is good, we don't want him to produce more kittens with this condition. And of course love and affection.

ANSWER 2

Vicky Wade
Ringwood Vet Hospital and Emergency
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This kitten incredibly appears to be missing his right radius! I've never seen such a thing but apparently it is a described condition called radial agenesis. I would certainly extend surveillance radiographs to include the femurs and assess whether they are also involved and hypoplastic. I feel as though the radiographs are pretty definitive in this case but a CT scan could always be performed to assess the bones in more detail. The limb has a varus deviation and long-term may benefit from a surgical opinion for ulnar-carpal arthrodesis or conservative supportive management, including disease modifying anti-arthritis drugs, weight management, exercise management and dietary therapy. If symptoms become severe enough, amputation could be a consideration.

Small

RADIAL AGENESIS

Anne Quain

BA BSc(Vet) BVSc MVS GradCertEdStud
MANZCVS (Animal Welfare) Dip ECAWBM
(AWSEL)

drannequain@gmail.com

C&T No. 5874

Dom is suffering from radial hemimelia or radial agenesis. Radial hemimelia or radial agenesis refers to partial (radial hypoplasia) or complete absence of the radius (Pisoni *et al.*, 2012, O'Brien *et al.*, 2002). Dom has a case of unilateral radial agenesis, with a missing right radius, a deformed right humeroulnar joint, a deformed right ulnocarpal joint and marked thickening of the right ulna. The right first digit is absent.

Prevalence

Radial agenesis is uncommon, but it is the most common hemimelia reported in dogs and cats (O'Brien *et al.*, 2002, Winterbotham *et al.*, 1985, Bezhentseva *et al.*, 2018).

Aetiology

The aetiology remains unknown, although the condition is thought to be inherited. This has been exploited by a small number of breeders seeking to create 'twisty cats', 'squittens' or 'kangaroo cats': <https://en.wikipedia.org/wiki/Squitten>

Hemimelia may also be secondary to teratogen-induced insult or injury to the neural crest *in utero*, associated with inflammation, infection, malnutrition or other material disease, vaccination, exposure to radiation, or toxins including drugs (O'Brien *et al.*, 2002). Another hypothesis is that the condition occurs due to vascular defects, leading to abnormal vasculogenesis which impacts bone growth (Pisoni *et al.*, 2012).

Clinical signs

Because the radius is the key weight-bearing bone of the forelimb, its absence tends to lead to dramatic deformity of the limb—particularly a varus deviation of the metacarpus, flexor constriction and muscle atrophy, and subsequently an abnormal gait (Pisoni *et al.*, 2012, O'Brien *et al.*, 2002, Bezhentseva *et al.*, 2018). In unilaterally affected cats like Dom, there may be obvious asymmetry and a moderate to marked difference in muscle mass between the limbs (Winterbotham *et al.*, 1985).

When sitting, cats with bilateral radial agenesis look a bit like a person sitting with their arms crossed in front of them. Severely affected cats may walk with the entire forearm in contact with the floor (Pisoni *et al.*, 2012), leading to thickening of the skin in this area (Winterbotham *et al.*, 1985).

Physical examination may reveal missing first digits (as in Dom's case), or polydactyly. It may be possible to palpate elbow joint subluxation. The range of motion of the carpi and elbows may be markedly reduced.

Unless secondary complications such as fractures are present, limb palpation is not usually painful (Winterbotham *et al.*, 1985, Pisoni *et al.*, 2012).

Because of abnormal load bearing, kittens like Dom may be prone to developing severe degenerative joint disease in the future (O'Brien *et al.*, 2002) and potentially skin abrasion of the forearms due to contact with the ground (Pisoni *et al.*, 2012).

Definitive diagnosis

Diagnosis is confirmed via radiographs, with partial or complete absence of the radius or radii (see Figures 1a-d). Concurrent abnormalities include malformations of the carpal joint, including complete or partial absence of the radiocarpal bone and first digit, varus deformity of the ulnocarpal joint and metacarpal or metatarsal fusion (synostosis) (Pisoni *et al.*, 2012).

Affected cats acquire further abnormalities due to abnormal load bearing and absence of normal support structures in the limb. These include a thickened, curved ulna due to increased load bearing, a varus deviation of the paw due to absence of medial support structures and contraction of extensor tendons, and humeroulnar subluxation due to the absence of the radial head (O'Brien *et al.*, 2002). Dom's right ulna is already markedly thickened and is unlikely to function normally. Radiographs are also important to screen for fractures, ankylosis and dislocations.

It is important to take radiographs of the limbs, thorax and spine, as affected cats may have other abnormalities including additional ribs and vertebrae, as well as metacarpal and metatarsal synostosis (Pisoni *et al.*, 2012) and cardiomegaly (Bezhenstseva *et al.*, 2018).

Management

Historically, the treatment for unilateral radial agenesis has been amputation or splinting (Bezhenstseva *et al.*, 2018). At the age of 11 weeks, Dom is relatively young but no amount of splinting will return this limb to normal. Early diagnosis and

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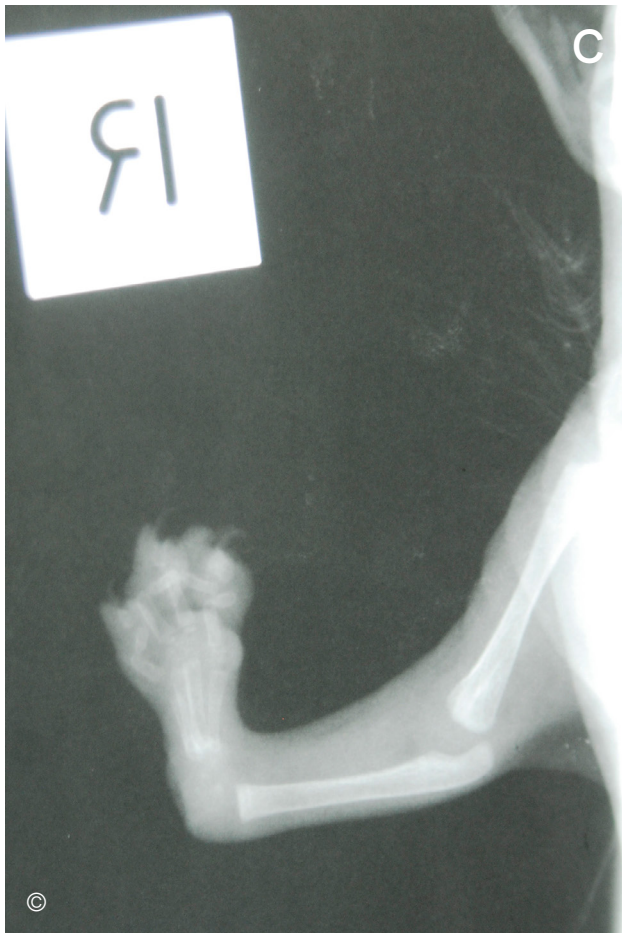


Figure 1. (A) Kitten with radial agenesis and syndactyly (B) Close-up of the right paw demonstrating syndactyly. (C) Craniocaudal radiograph of the right forelimb, note absence of radius. (D) Craniocaudal radiograph of the left forelimb



Figure 2. (A) Craniocaudal view of the right forelimb, note absence of radius. (B) Lateral view of the right forelimb. (C) Craniocaudal view of the left forelimb, note absence of radius. (D) Lateral view of the left forelimb.



Figure 3. Adult cat (approximately 8 years old) with bilateral radial agenesis, treated with benign neglect.

conservative treatment (e.g. splinting) may reduce flexural contracture which leads to varus deviation, muscle atrophy and pathologic fracture, although splinting is not without the risk of complications (Pisoni *et al.*, 2012). In this case, amputation of the limb would be a reasonable option.

In bilateral cases, euthanasia may be considered due to the extent of the deformity. In dogs a range of surgical approaches have been trialed, including reconstruction of the radius using a rib autograft stabilised with Kirshner wire fixation; distraction osteogenesis for lengthening the forelimb, potentially combined with ulnocarpal arthrodesis; combined humeroulnar and cranial ulnocarpal arthrodesis and dorsolateral ulnocarpal arthrodesis (Bezhtentseva *et al.*, 2018).

Bezhtentseva and colleagues performed staged, bilateral pancarpal arthrodeses in an eight-month old cat, six weeks apart. The cat made a good recovery. Almost five years later, the authors felt there was a 'good to excellent functional result', with all implants remaining stable and evidence of continued bone remodelling on radiographs. The cat did have some (approx. 15 degrees) internal rotation of the right forelimb manus and a shorter stride with some lameness.

They speculated that this could be due to the severity of the initial deformity and associated flexure contracture, asymmetry of the limb length, iatrogenic tendon shortening (this was performed during surgery on the right fore to address

unexpected extensor tendon laxity, but was not deemed necessary on the left fore); persistent soft tissue discrepancies or undetected pain or discomfort. The cat was pain-scored using a visual analogue scale, but only at long term follow up, and it is not known whether a validated pain scoring instrument was used.

Caroline O'Brien, Richard Malik and Robert Nicol diagnosed an 8-month-old female entire Devon Rex with bilateral agenesis in 2001 (O'Brien *et al.*, 2002) (Figures 2a-d). At the time the owner, the late Sybil Drummond, elected not to treat the cat, who lived exclusively indoors in a multi-cat household on carpeted floors. To reduce the risk of trauma, the owner placed carpet on hard surfaces (including chairs and tabletops) that the cats would jump on. I examined the cat on several occasions in 2009, in her home (Figure 3). While her gait was markedly abnormal—she walked and ran with her forearms contacting the floor—she ambulated at the same pace as other cats in the household. The owner reported that the cat's quality of life was excellent. At the time however, I did not perform any pain-scoring. Repeat radiographs revealed marked degenerative changes in the elbow and carpal joints (Figure 4a and 4b). The cat died at home in May 2014.

Given the potential for significant gait impairment, it is prudent to keep affected cats indoors (with outdoor access supervised, if permitted), and take steps to minimise trauma. It is likely that affected cats would be at increased risk of developing severe degenerative joint disease.

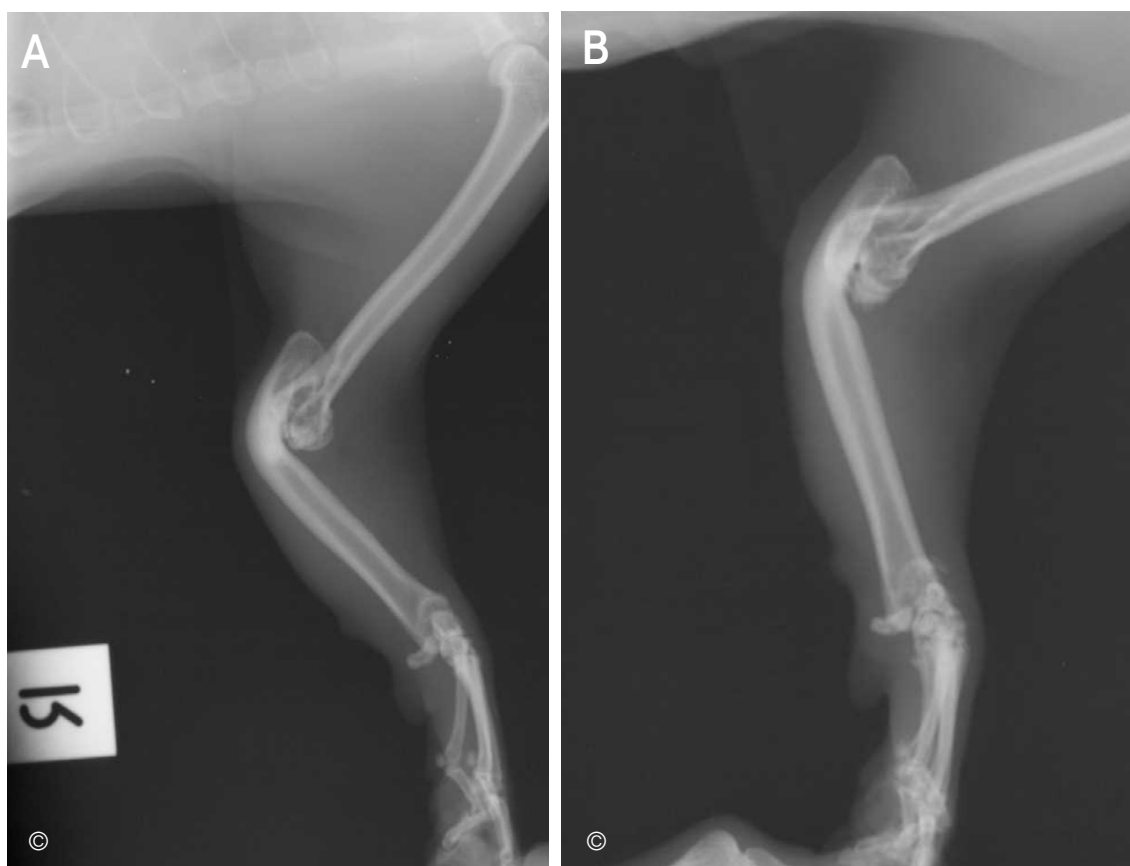
There is no published information about sequential pain-scoring of cats affected by this condition. Nonetheless, the Feline Musculoskeletal Pain Index is a validated pain-scoring tool designed by Duncan Lascelles and colleagues at the North Carolina State University's College of Veterinary Medicine to diagnose and monitor pain arising from chronic musculoskeletal disorders in cats. Owners and veterinarians can register an account for free at <https://painfreecats.org/> and complete multiple questionnaires.

This is useful for establishing a baseline for Dom, as well as monitoring progress and assessing the efficacy of different treatments if they are used. The website also provides useful information to owners about the impact of pain in cats, and potential management strategies.

Because this condition is potentially inherited, affected cats including Dom should not be used for breeding (Pisoni *et al.*, 2012) and any client expressing a desire to do so should be actively discouraged on animal welfare grounds.

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SOLVING THE PROBLEM OF OWNER COMPLIANCE WHEN TREATING CANINE OTITIS EXTERNA

The successful treatment of canine otitis externa traditionally relied on long courses of multi-dose medication administered by owners to their dogs at home. However, long courses of home treatment can make compliance and accurate dosing difficult to achieve.^{1,2} A survey found that most dog owners find twice daily administration for up to 14 days difficult, or even impossible.^{1,3}

Another study revealed that up to 80% of owners give the incorrect dose when administering ear products to their dog.⁴ This becomes more challenging when dogs become 'ear shy' or stressed, which is often further exacerbated by repeated administration throughout the course of treatment. Owners may also feel guilty that their pet is distressed, and cease treatment too soon.

86% of owners prioritise reducing stress for their dog when treating ear infections.¹

Furthermore, even owners that believe they can administer ear medication may not always want to. 72% of dog owners would prefer a single dose otitis externa treatment administered by the vet, including those that don't mind administering treatment to their dog at home.¹



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References:

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Small

USE OF INTERVENTIONAL PAIN MANAGEMENT FOR TREATMENT OF OSTEOARTHRITIS

Donna White

The Sydney Animal Pain Clinic

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C&T No. 5875

Interventional Pain Management

Interventional pain medicine can be used for the management of acute and chronic pain. Interventional pain procedures involve the administration of drugs locally around sensory nerves to reduce, or abolish, pain signals within those nerves. The most basic or well-known may be dental nerve blocks. Here vets use local anaesthetic drugs and their knowledge of dental anatomy to completely abolish pain sensation to the dental arcades prior to tooth removal. These skills can be extended to provide analgesia to the nerves supplying painful areas of the body, such as a sore shoulder, elbow, hip, stifle etc. Pain management through use of an integrated approach in pain clinics is a well-established treatment model in human medicine. The use of pain clinics is a new concept in veterinary medicine, which will likely become more commonplace.

Case Example – Alice and her osteoarthritic shoulder

Signalment and History

Alice is a 10-year-old female spayed Shih Tzu x Maltese crossbred. She was adopted by her owners as a puppy and lives with one other dog.

Alice initially presented to her general practitioner for intermittent lameness in her left forelimb, about two years ago. She was diagnosed with left shoulder osteoarthritis via physical examination and radiographs. Her owners could not identify any previous injuries to this area that may have



Figure 1. Alice

predisposed this joint to osteoarthritis. Alice was prescribed meloxicam orally for seven days and then as required, which she reportedly responded well to. Within six months, Alice was requiring meloxicam every day to maintain good mobility in her left forelimb. As time progressed Alice's lameness progressed and she became reluctant to go on walks. Her referring vet then added gabapentin to Alice's medication regime. Alice again seemed to respond well to the addition of this medication with improved mobility and demeanour. Physical examination and screening bloods were otherwise unremarkable. Alice was referred to the veterinary pain clinic for further evaluation of her pain management plan.

Medication Options

Alice's owners are unable to directly tablet her. All medications are given in food. While this can work very well for medications such as meloxicam and gabapentin, other chronic pain medications such as amantadine and amitriptyline are much less palatable. As such, we were limited in what medications we could now add in.

Interventional Pain Management Options

Interventional pain management procedures for shoulder osteoarthritis include nerve blocks and intra-articular treatments.

The nerves supplying the shoulder joint arise from the brachial plexus. Interventional pain procedures aimed at the brachial plexus may aid pain

management by reducing the pain signals within these nerves. These nerves include both motor and sensory nerves. The use of local anaesthetics (both short acting such as bupivacaine, and long acting such as liposomal bupivacaine) are best avoided in this situation as they will inhibit motor function. Other analgesic drugs such as long-acting steroids can be used. It is believed their effectiveness is due to dampening of the pain signals in the sensory nerves

Intra-articular medications include Platelet-Rich Plasma (PRP), hyaluronic acid, botulinum toxin (Botox) and steroids. There is no strong evidence for any one of these treatments in dogs with osteoarthritis, although individual animals can have surprisingly good, and prolonged, responses to one or more of these treatments.

Alice's owners elected to treat her with brachial plexus nerve block. This was performed under sedation and ultrasound guidance, with the administration of long-acting corticosteroids and an opioid. Alice responded very well to the interventional treatment. Within one week

of treatment Alice's owner noted a significant improvement in her forelimb gait, and furthermore an improvement in her demeanour. They were able to wean down her meloxicam treatment. The analgesic effects of the nerve block treatment are expected to last about 8 weeks, although there is some individual variation, with some animals receiving effective analgesia for up to twice as long as this.

The Sydney Animal Pain Clinic operates a mobile pain clinic specialising in Interventional Pain Management. It was started as the passion project of two Veterinary Anaesthetists with a love of pain management and a desire to offer the same pain clinic services to animals that are available to people: Dr Fernando Martinez-Taboada and Dr Donna White. Most cases seen at the clinic are chronic pain cases; osteoarthritis being number one and neuropathic and oncological cases comprising a good proportion. Nerve blocks are the primary treatment performed, with intraarticular treatments and nerve ablations also available as indicated.

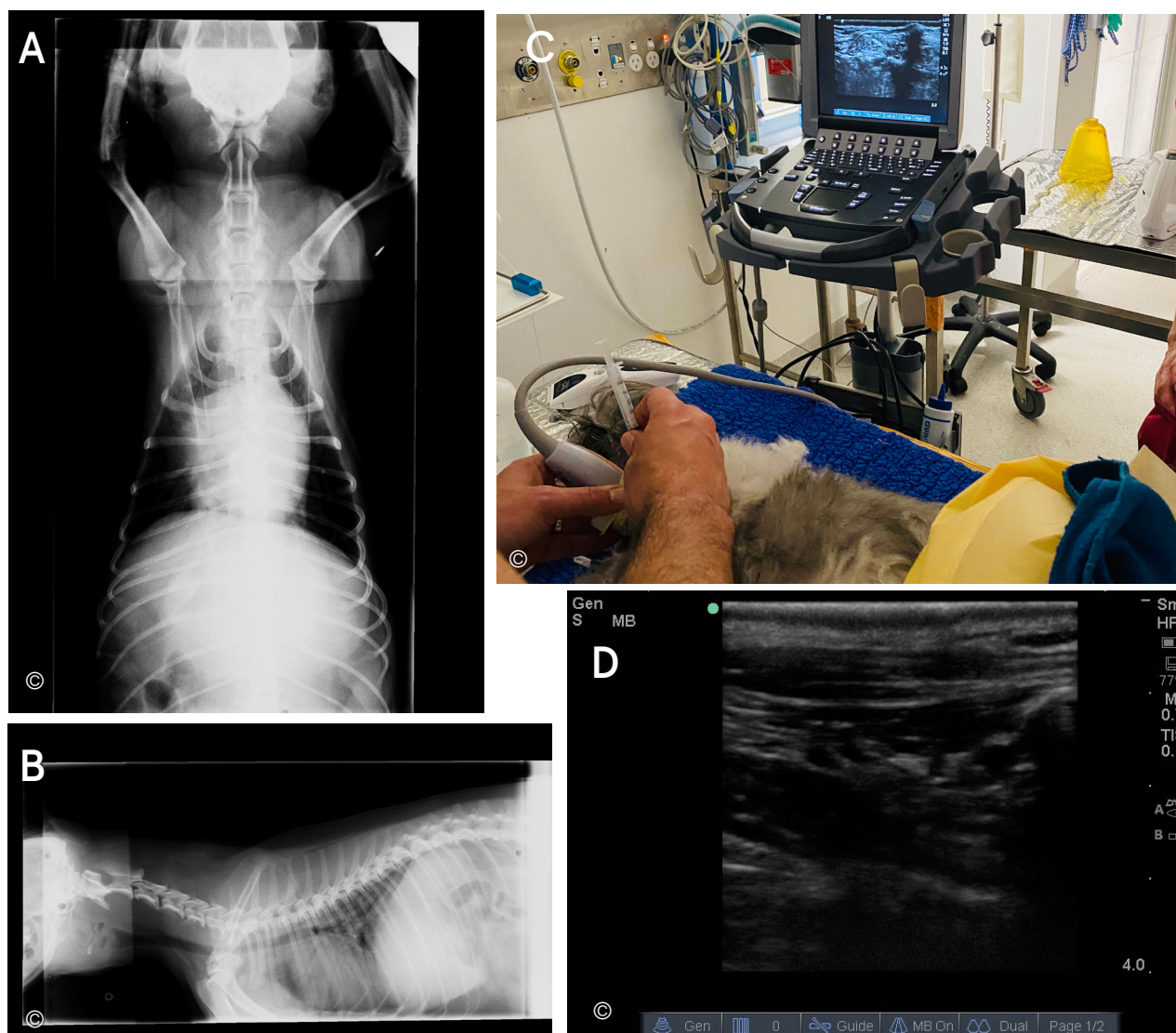


Figure 2. (A & B) Referring vet radiographs showing shoulder OA (C) Alice getting nerve block (D) Ultrasound image of brachial plexus

Small

TWO NASAL FOREIGN BODIES

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These two cases occurred four weeks apart, both on a Friday afternoon...

Case report 1

History

A three-year-old male neutered Golden Retriever presented for evaluation of acute unilateral epistaxis of the left nostril and sneezing episodes after running through some bushes. The patient was otherwise well with no pre-existing medical conditions and had not had any recent surgery. There was no other evidence suggestive of an underlying coagulation disorder and the dog had not had any known exposure to rat bait. The dog had been consistently dosed with three-monthly prophylaxis for fleas, ticks and internal parasites (Bravecto® and Milbemax®). No previous drug, food or environmental allergies had been recorded in this patient.

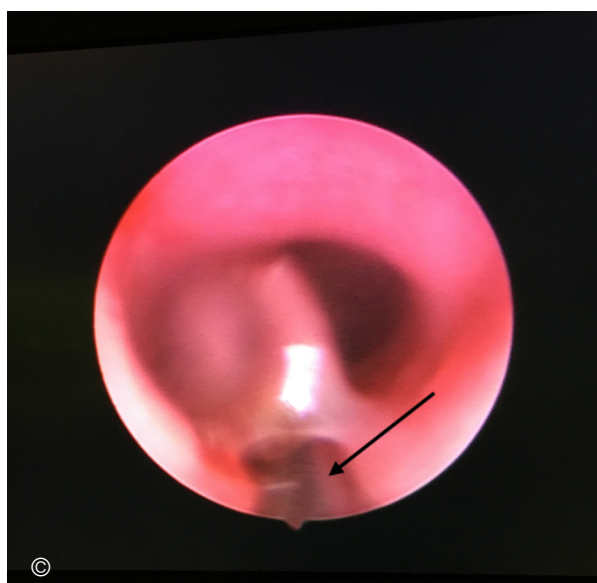


Figure 1. View through a rigid endoscope. The arrow highlights the foreign body (FB)

Physical examination

On physical examination, the patient was bright, alert and responsive, albeit nervous and panting. The mucous membranes were pink and moist, capillary refill time less than 2 seconds and body condition score 2.5/5. Cardiothoracic auscultation was normal, with heart rate of 120 beats per minute. The body temperature was elevated at 39.8°C. The dog did sneeze a few times during examination but there was no evidence of epistaxis or other forms of nasal discharge. There was no visible external nasal trauma and no pain on manipulation of the nares or on opening of the mouth.

Recommendations were made to proceed with endoscopy should clinical signs persist in this patient. Over the next 24 hours, the dog seemed comfortable at rest; however, exercise or eating brought on sneezing episodes with mucoid nasal discharge.

Endoscopy

The following day, the patient was admitted into hospital for further investigation. The dog was panting, and mucous membranes were pink, moist and capillary refill time less than 2 seconds. The heart rate was 108 beats per minute, temperature 38.6°C. Pre-anaesthetic blood work was declined. The dog was pre-medicated with low doses of acepromazine (0.02mg/kg) and methadone (0.2mg/kg) given intramuscularly into lumbar epaxial muscle. An intravenous catheter was placed. The patient was induced with alfaxalone given slowly to effect (2mg/kg diluted 50:50 with saline) and intubated routinely with a 10mm cuffed endotracheal tube. Anaesthesia was maintained with isoflurane. Intravenous Hartmann's fluids were run at surgical rates throughout the anaesthetic. Monitoring included capnography, pulse oximetry, oesophageal temperature, blood pressure (oscillometric) and forced warm air circulator and fluid line warmer for thermal support. A rigid rhinoscope was advanced into the left naris from where a foreign body was observed (Figure 1). Biopsy forceps were advanced through the rigid scope which was used to grasp the foreign body. A 10cm stick was retrieved. A small amount of epistaxis ensued but did not persist into recovery. The dog was extubated and recovered routinely from anaesthetic.



Figure 2. Grasping the offending FB – a L0000NG stick (Photo courtesy of Anne Quain)



Figure 3. Happy patient and two proud veterinarians!



Figure 4. SUCCESS—the hard to dislodge stick



Figures 5. Rigid endoscope

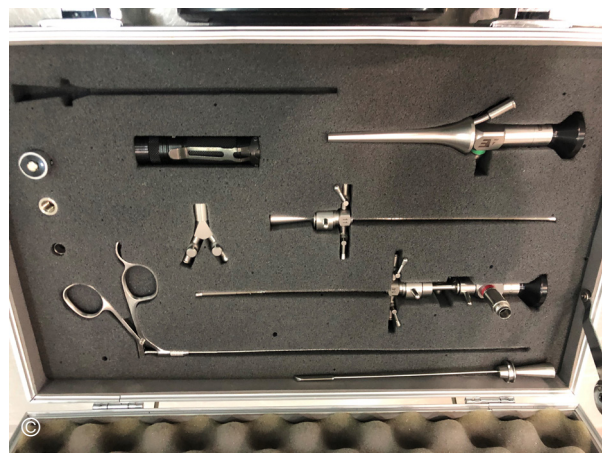


Figure 6. Rigid endoscope and kit

Case report 2

History

A two-year-old male neutered Labrador presented for evaluation of unilateral epistaxis from the right naris and violent sneezing episodes, at least once a day, for the past week. The patient was otherwise well with no pre-existing medical conditions and had not had any recent surgery. There was no other evidence suggestive of an underlying coagulation disorder and the dog had not had any known exposure to rat bait. The dog had been consistently dosed with three-monthly prophylaxis for fleas, ticks, and internal parasites (Bravecto® and Milbemax®). No previous drug, food or environmental allergies had been recorded in this patient.

Physical examination

On physical examination the patient was bright, alert, and responsive. Vitals, including cardiothoracic auscultation and rectal temperature were within normal limits. There were no abnormalities on examination of the nares, oral cavity or pharyngeal regions. **During the examination, after palpation of the soft palate, the dog did a large sneeze that resulted in a stick (2cm in length) being expelled from the nasal cavity.** The decision was made to send the dog home and monitor in the hope that the problem was resolved.

Over the next 24 hours, the dog continued to sneeze and the recommendation was made to proceed with endoscopy.

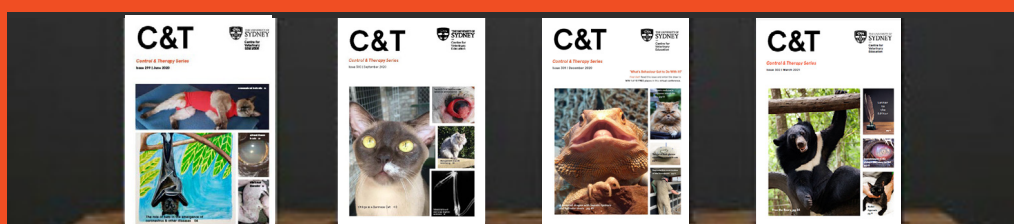
Endoscopy

Pre-anaesthetic blood work was within normal limits. The dog was pre-medicated with low doses of acepromazine (0.02mg/kg) and methadone (0.3mg/kg) given intramuscularly into lumbar epaxial muscle. An intravenous catheter was

placed. The patient was induced with alfaxalone given slowly to effect (2mg/kg diluted 50:50 with saline) and intubated routinely with a 10mm cuffed endotracheal tube. Anaesthesia was maintained with isoflurane. Intravenous Hartmann's fluids were run at surgical rates throughout the anaesthetic. Monitoring included capnography, pulse oximetry, oesophageal temperature, non-invasive blood pressure (oscillometric) and forced warm air circulator and fluid line warmer for thermal support.

A rigid rhinoscope was advanced into the right naris. A stick was visualised wedged ventrally, approximately 2.5cm into the nasal cavity. Biopsy forceps were passed through the biopsy channel; however, purchase was not able to be gained on the stick as it was wedged too tightly, and the jaws were not wide enough for the width of the stick. In a similar manner, a flexible cystoscope was passed into the naris and biopsy forceps passed through the biopsy channel; but these were also unsuccessful in grasping the stick. **Alligator forceps were then used to run alongside the cystoscope.** The cystoscope was used to visualise

and guide the alligator forceps to grasp the stick. Unfortunately, the stick could still not be grasped due to it being wedged firmly in the cavity. **The flexible cystoscope was then passed through the oral cavity and retroflexed up behind the soft palate into the nasopharynx. The choanae were visualised and saline flushed firmly through the scope and into the right naris from where it meets the choanae.** This process was repeated three times. Afterwards, the flexible scope was used again to enter the right naris rostrally and this time the stick was sufficiently dislodged and was able to be retrieved through the right naris using the soft flexible scope and biopsy forceps. The stick was 5cm in length and removed in its entirety. The stick was malodorous and some mucopurulent discharge was present at the distal margin. The dog was extubated and recovered smoothly from anaesthetic. The patient was sent home with antibiotics (amoxicillin clavulanic acid) for 5 days. Figure 4 shows the two fragments put together (one from what was expelled in the consultation and the remaining 5cm that was retrieved using rhinoscopy).



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Small

WHEN A NASAL FOREIGN BODY IS THE SLIVER OF THE FULL SCOPE

Pete Coleshaw

Jaffa's Health Centre for Cats

Salisbury UK

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C&T No. 5877

2020 was our 'year of the snot' (older readers may be familiar with Al Stewart's late 70's classic 'year of the cat' song).

Let me elaborate; we had a client start up a rescue centre in spring—which had the boost of every local established cat-welfare organisation dipping out of any cat welfare due to COVID-19. So suddenly, we were presented with loads of colony cats presenting with lots of diseases, many of which I had not seen in 30 years—coccidiosis, tritrichomoniasis, aggressive calici ulcers, rampant herpes, sneezes, snots, and watery eyes. Famciclovir sales went through the roof.

Enter Coco, a 5-month-old kitten from an infected household—epiphora, sneezing—the usual. After two courses of famciclovir and courses each of doxycycline, azithromycin and finally amoxycylav, Coco was still sneezing, **with a tenacious left-sided mucopurulent discharge.**

This had me confused—I have seen more than enough chronic snuffles, but never unilateral disease.

A nasal flush (with seemingly free passage of saline both sides) gave temporary relief, but our geriatric paediatric bronchoscope had expired and had not been replaced, so further investigation was not possible. The cat was uninsured and our visiting expert endoscopist is beyond the financial resources of the owner.

Consultation with those with greater knowledge suggested nasal polyps, choanal atresia, or simply blockage with inspissated pus.

A new scope was acquired on approval, a 4mm paediatric bronchoscope with 180/130-degree flexion. With the cat in dorsal recumbency, sloping head down, throat packed with a cotton

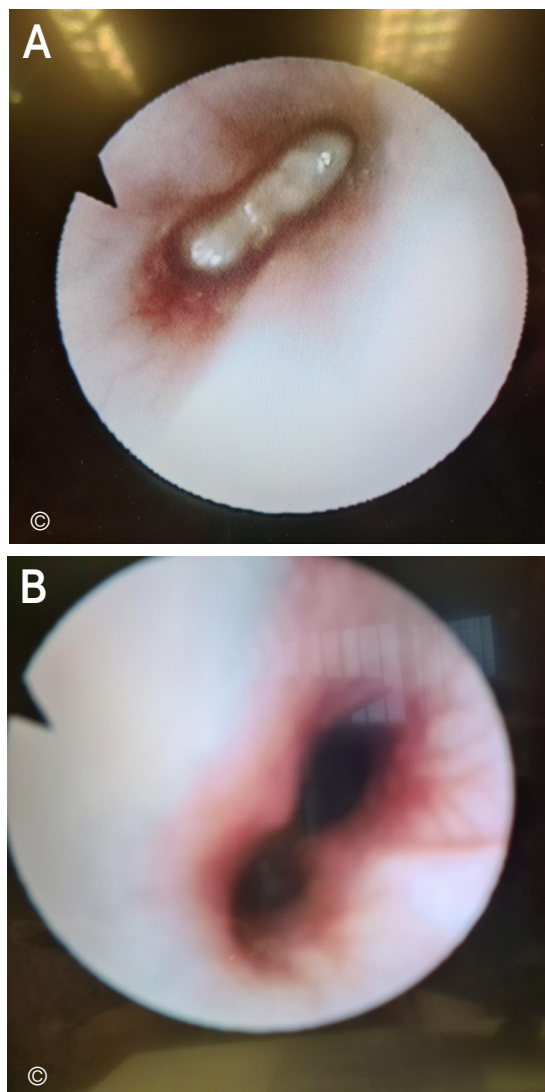


Figure 1. Nasopharynx before (A) and after (B) flushing. Note the sponge occluding the choanae in (A).

wool ball and cuffed endotracheal tube in place, retrograde caudal rhinoscopy was performed. A whitish structure was immediately seen occluding both nasal passages. An attempt was made to use grasping forceps to sample/retrieve the structure but proved impossible with the grabber in place—not sure it is designed to bend through a tight 180°, but it survived unbroken.

However, we had seen our pathology and we could not stop until it was shifted by flushing or by 'pull-through' of a piece of gauze. Low-pressure jetting had failed. Increased pressure from a 20mL syringe gave a more forceful and **successful flush of a small piece of sponge.** It later transpired that the kitten had a fetish for sponges much in the way others have for hair bands. And that was where I was initially going to end this vetective story. However, there is more!

When flushing the nares, we thought we saw water running down the inside of the anaesthetic tubing, despite all our preventive measures. So, we quickly radiographed the chest, extubated, scoped the trachea, and confirmed we were being paranoid.



Figure 2. Our scope is a 4mmx680mm long bronchoscope to which we attach an inexpensive hard-wired camera head-though Bluetooth ones are now available – <https://fireflyglobal.com/de1250-wireless-endoscope-camera/> A light source with suction and ‘blow’ is also required, and if starting out with a tight budget then second-hand units can be acquired at sensible money. A laptop can then be used for display and recording purposes.

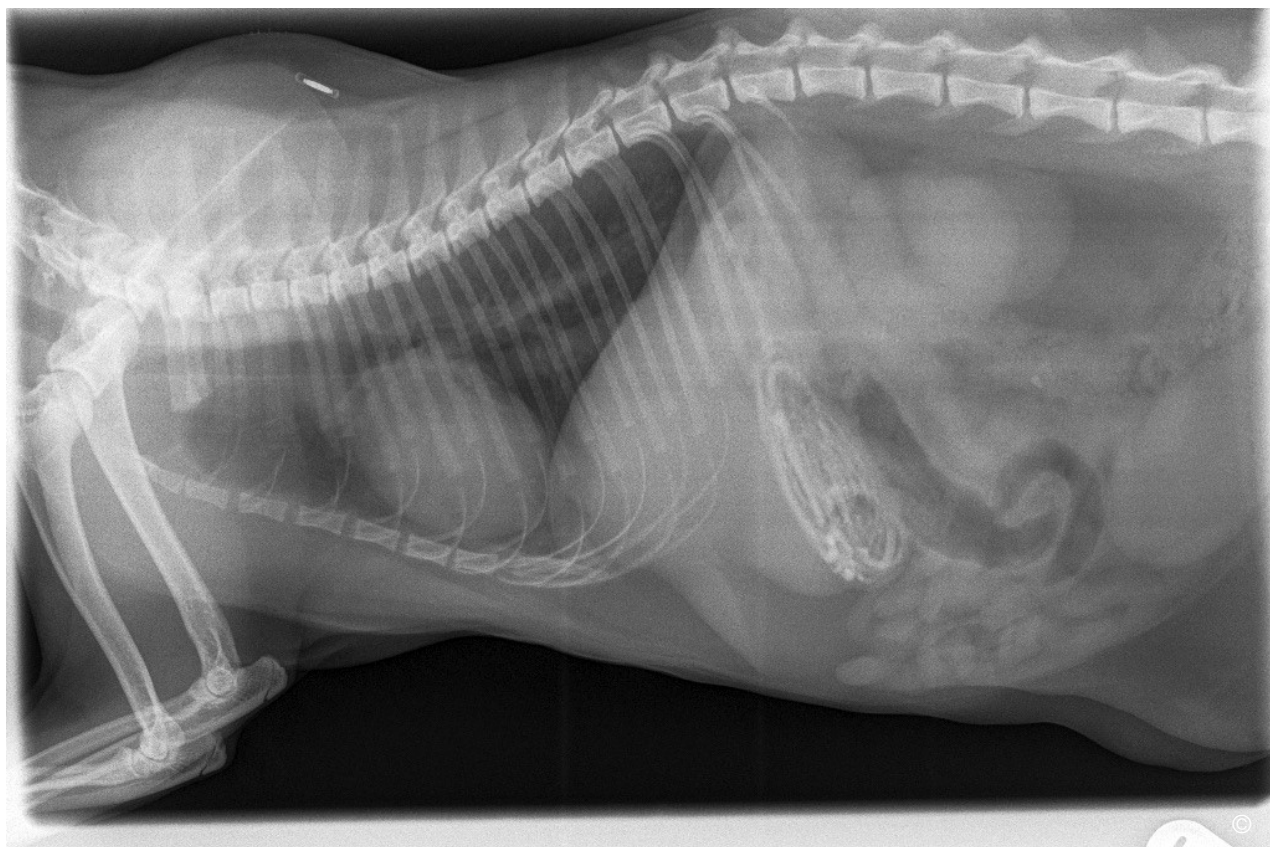


Figure 3. Lateral radiograph showing foreign bodies in the stomach



Figures 4. A & B. Objects retrieved at gastrotomy

Two days later when putting the radiographs into the client notes we noticed the full cat X-ray! Could this be a lost dental swab with its cotton tail? Could we really be so lax? We tried to retrieve by endoscopy, but our grabber was never going to extract the tangled ball of foreign substances extracted later at gastrotomy. Hair bands, elastic bands—and hey—a piece of sponge!



So, what are the take home messages from this story?

1. It can be cheaper to buy a 2nd hand endoscope which you can use for 10 years, rather than get a visiting specialist to do a very simple scope.
2. Always consider a lesion in the nasopharynx when confronted with refractory nasal discharge in the cat, especially if it's asymmetrical.
3. Remember that the most common reason material gets to the nasopharynx is via vomiting, with some material going up ABOVE the soft palate, to lodge in the nasopharynx.

As well as the FBs seen here, we are aware of tablets, fish bones, grass seeds, blades of grass (classic!) that can be lodged in this location. And do not forget about choanal atresia, nasopharyngeal polyps, posterior nasal cryptococcosis and caudal nasal lymphoma!

Wildlife

FREE THE BEARS

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PhD

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C&T No. 5878

Background

Free the Bears is an NGO created in 1995 by Perth grandmother Mary Hutton when she learnt of the plight of Moon Bears and Sun Bears that were subject to bile farming and illegal wildlife trade in SE Asia. Since 1995 five sanctuaries have been formed—one in Cambodia, two in Vietnam and two in Laos and partnerships formed with groups in three other countries (India, Indonesia and Thailand) resulting in over 950 bears having been rescued and rehabilitated.



Left: Matt Hunt, CEO, Free the Bears

Matt Hunt has been the CEO of the Sanctuaries since 2007. Matt has done and is continuing to do an amazing job, running, building and improving the sanctuaries and their facilities while rescuing a continual stream of neglected and abused wildlife.

Volunteers Program

I went over to Laos in December 2019 as part of an Australian Volunteers Program, that partners with local government bodies. I was the Veterinary support officer for the Laos Free the Bears sanctuary located in Luang Prabang, a beautiful town situated on the banks of the Mekong River. My role was to help with the continuation of setting up the wildlife hospital, (which was well underway due to the hard work of my predecessor), mentor the newly graduated Laos veterinarian, and provide veterinary care for the 70 bears, 42 Macaques, three red pandas and several other species that were housed at the sanctuary.

Two Sanctuaries in Luang Prabang province, Laos

The original sanctuary, established in 2003, is set near Kuang Si waterfall, one of Laos' premiere tourist destinations. As the number of rescues grew, space at the sanctuary became



Kuang Si waterfall

insufficient and it was soon necessary to expand. Work commenced on development of a second facility, the Luang Prabang Wildlife sanctuary in 2017. Covering more than 25 hectares, around eight km as the crow flies from the first, the new sanctuary offers more space for creating larger enclosures for the bears, housing for other species commonly encountered in the illegal wildlife trade and a dedicated wildlife hospital. Currently there are four bear houses and a cub nursery with a fifth bear house being started soon. A BBC documentary will soon be aired 'Bears about the House' telling the story of two rescued cubs..... but that's all I'm going to say; you have to watch for yourselves.

Local and International People Making a Difference

My passions have always been studying wildlife disease and conservation. We live in a beautiful and amazing world that unfortunately humanity seems intent on—not appreciating and looking after—but destroying. I went to Laos in the hope that I could in some way contribute in helping these stunning and endearing animals. It was an amazing and humbling experience; I am so glad I could be part of it, even for a short time before the onset of COVID-19. I take my hat off to the people I met and worked with—a dedicated group of international and national people, drawn together in their quest to save not only the bears from awful, tortured lives, housed in small cages for the archaic milking of bile but the many other species that pass



General health check on newly acquired male bear 'Henry'.

through the illegal wildlife trade, housed in appalling conditions or killed for body parts.

Introduction & Assessment of New Arrivals

All new animals to the sanctuary undergo 30 days in quarantine before being introduced to the other animals. Unless requiring a general anaesthetic upon arrival for assessment and treatment of obvious injuries, most new animals undergo a general health check during the quarantine period.

The bears are darted in their den with a mixture of medetomidine and Zoletil and are then transported to the surgery for their health assessment. The animals are intubated and maintained on isoflurane during the procedure. Body measurements are taken and body condition assessed. Routine checks of eyes, ears and teeth are performed. Quite often the bears have dental issues from being fed inappropriate diets and chewing on bars of cages. Ultrasound assessment of internal organs, especially



Lab area in the Wildlife hospital

the gall bladder in case of bile extraction, are also performed. Radiographs of limbs and chest are taken if needed to ascertain joint issues and lung health, with tuberculosis being a risk in bears who have had previous close contact with people.

Blood samples are taken, smears assessed, and serum is collected and stored for future studies. Hair samples are also taken for DNA analyses.

Why Moon Bears are exploited

Moon Bear bile has high levels of ursodeoxycholic acid, making their bile a prized ingredient in traditional medicine. This bile acid, known to have some non-essential medicinal properties, is now being made synthetically, so there is no reason at all for it to be extracted from bears. However, despite this fact, this horrendous tradition continues in some parts of Asia.

In summary

Overall, it was an amazing experience. Working on conservation projects such as these is truly rewarding. I would encourage everyone travelling through Laos, Vietnam or Cambodia to visit at least one of these sanctuaries, see the bears and support the work these amazing people do.



Carol performing surgery on a Moon Bear at the Tak Kuang Si Sanctuary Clinic



Viewing area outside one of the bear houses. The bears love to climb and sunbake on the platforms. Food items and treats such as seeds, nuts and peanut butter are hidden around the enclosure to encourage the bears to move around and help alleviate boredom to prevent stereotypic behaviours from occurring. Many of the bears that are rescued have already developed behavioural idiosyncrasies from years of being confined in small cages or chained. Once these are ingrained, they are very difficult to stop.



Two rescued bear cubs arrived just before I left; a male and female. They were in quarantine but have since been moved to the cub nursery. Extremely cute, you just want to give them a cuddle but they still have sharp teeth and claws.



Moon Bears or Asiatic Black Bears—*Ursus thibetanus* have been exploited for decades by humans for their bile, fur and other body parts



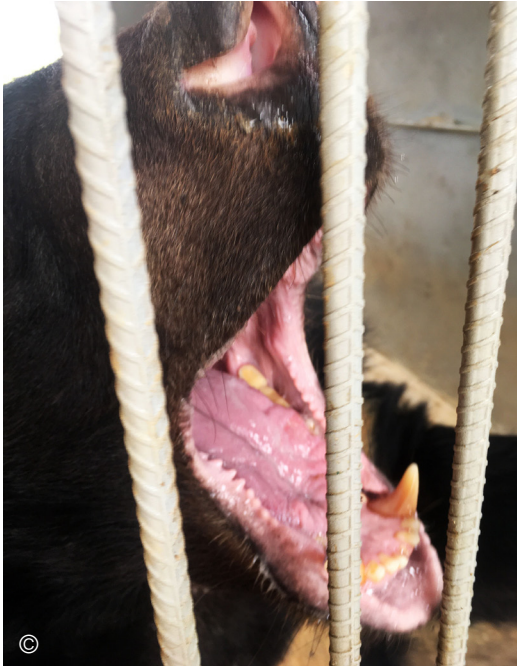
Akong, Carol, Lar (Animal Manager) and Meng the Laos Vet



At Kuangsi sanctuary—Meng and Nikki (Education officer)



Meng with an anaesthetised Moon Bear undergoing a general health check



Bears are taught to open their mouths so that their teeth can be inspected.



Abdomen has been shaved for ultrasound investigation



'Sarah' sitting outside her den. She and her brother Henry were donated when they became too big to be kept by their family. Henry came in at a whopping 240kg; he is now down to a svelte 180kg.



Crested goshawk *Accipiter trivirgatus* surrendered by a local person



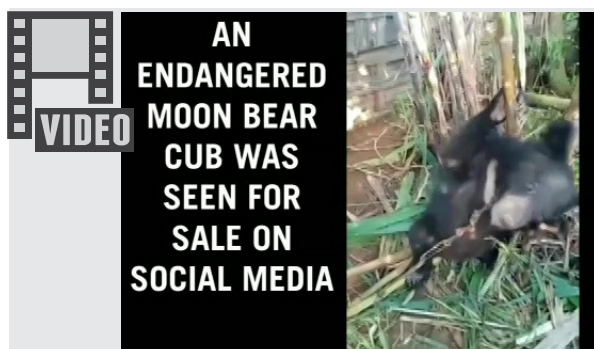
Leopard cat kittens *Prionailurus bengalensis* were rescued from illegal wildlife trade. There were three initially but unfortunately one succumbed to the heavy parasite burden, emaciation and dehydration they came in with. The remaining two males are growing rapidly with plans to release them once a safe release site is established.



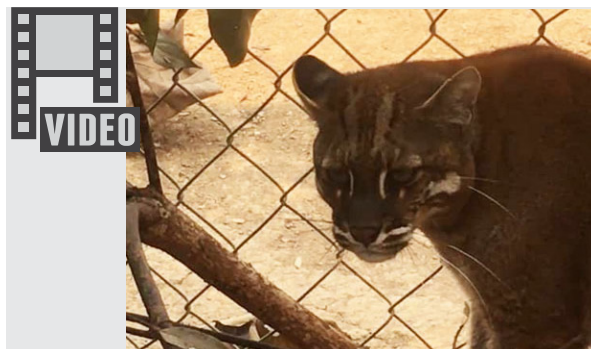
'Belle'— A baby Assames Macaque



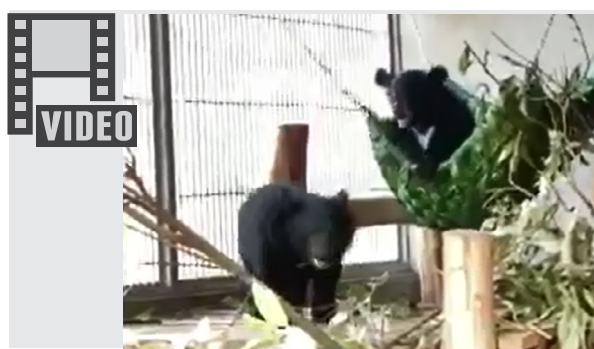
Keeper weighing one of the Stump-tailed macaques *Macaca arctoides*. Animals are weighed monthly to ensure they are maintaining their weight. Many of the monkeys were either rescued or surrendered so they too have behavioural issues. It is a constant challenge to give the monkeys plenty of environmental enrichment and stimulation to prevent over-grooming and other repetitive behaviours and fighting.



This bear was rescued on the 3rd of March 2021 after the bear was seen for sale on social media



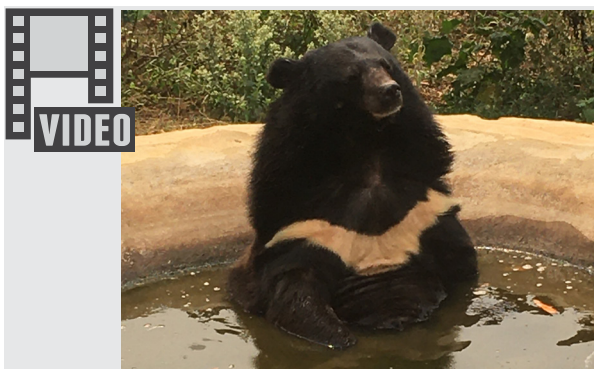
Golden cat *Catopuma temminckii*



Bears playing



Bears playing at Kuang Si



Civet Mai having a quiet dip in his pool



Juvenile Bamboo rat *Cannomys badius*. Three were rescued from the local market and brought in by a member of the public. Unfortunately one didn't survive. The remaining two were nursed back to health and released on the sanctuary grounds.



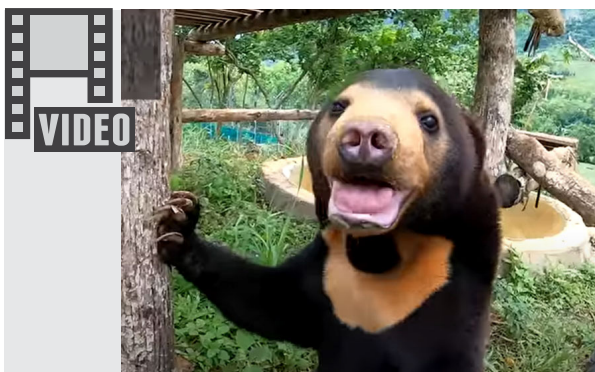
Left: Slow loris *Nycticebus bengalensis*, was rescued only a few days after I arrived in Laos. As you can see in the photo, the animal's left eye was unviable. My first surgery at the sanctuary was an eye enucleation on a species I had only seen pictures of! One of the interesting features of this—as the name would suggest—slow moving animal is that it is the only venomous mammal. It has a venom gland on the inside of its elbow and when threatened it raises its arms above its head and licks the gland. The effects of a bite range from pain to anaphylaxis.

Control & Therapy Series - Issue 302 March 2021

Right: Post-surgery. The wound went on to heal well and the loris was placed in a large aviary with food placed around it in different places every night. Camera traps were used to monitor the slow loris to see if it was having trouble finding food with only one eye, the aim being to see if the loris would be suitable for release back into the wild.



Filming Baby Sun Bears is NOT easy! | Bears About The House | BBC Earth



Orphan Sun Bear gets a new chance at life | Bears About The House | BBC Earth



Centre for Veterinary Education | Est. 1965

Saving Bears in the wildlife trade across Asia | Bears About The House | BBC Earth



Feeding 39 Hungry Moon Bears Takes A Military-Style Mission! | BBC Earth

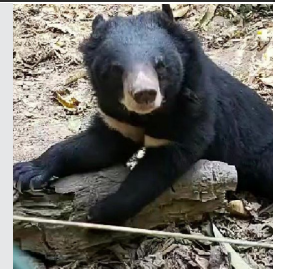


14 Reasons Sun Bears Are Your New Favourite Animal | Bears About The House | BBC Earth



Like to volunteer with Free the Bears? – [READ MORE HERE](#)

For more information and an application form, please [email us here](#).



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HOW DO YOU PROCESS YOUR SURGICAL INSTRUMENTS?

Are there room for savings?



Is your current process sustainable?



Can processes be improved?



Have you ever really looked at the way your practice processes its sterile equipment?

Do you know what the financial, environmental or labour impacts are of these processes?

Your clinic is likely sterilising Surgical Instruments and implants using at least one of the following methods;

- single-use blue wraps
- single-use paper or sealable plastic pouches
- re-usable cloth drapes
- re-usable rigid sterilisation containers

Single-use wraps are inexpensive and commonly available. However, they are designed to be used once and then discarded. They are easily perforated, therefore compromising sterility.

Single-use paper or sealable plastic pouches are also inexpensive and commonly available. They serve the purpose of allowing small quantities of instruments to be contained and sterilised, but are also discarded after each use and are also easily perforated.

Like many single-use products in the clinic, blue wrap and sealable pouches will contribute to a significant percentage of a practice's waste that will end up as land fill. Australians produce approximately 21.6 billion tonnes of land fill each year;¹ however, there are other options to help reduce the waste and the impact it has on our environment.

Re-usable cloth drapes are relatively expensive. Although they are disposed of infrequently, they need to be washed and dried after each use. They can still be perforated and hold contaminated waste in the fibres. Consider the water, detergent, electricity and nursing time required to clean them.



[View Primeline ProContainer](#)

Reusable rigid sterilisation containers are typically made of aluminium. While they require a larger upfront cost, there's no comparison in lifespan as rigid containers can last for more than a decade. If you calculate the processing cost per kit, a practice can see savings as soon as year 2 of ownership, simply by comparing to the cost of consumables that would have been used instead.

For more information on Aesculap reusable container systems, visit

- bbraun.com.au/en/products-and-therapies/product-catalog/sterile-goods-management/sterile-supply/sterile-containers.html

or email us at vetcare.au@bbraun.com for personalised assistance.

B | BRAUN
SHARING EXPERTISE

General

AN INNOVATOR IN FELINE MEDICINE

Gary D. Norsworthy

DVM, DABVP (Feline)

Alamo Feline Health Center

San Antonio, Texas, USA

2020 Texas VMA Distinguished Career
Achievement Award Recipient

C&T No. 5879



Figure 1. Gary next to his DR radiology monitor

Gary Norsworthy is an inspirational veterinarian. Although he has been in exclusive feline practice from many years, the lessons from his life could provide a template for the careers of inspiring young veterinarians just as distinctive and impressive as Tom Hungerford's 'goanna track' manifesto. My take-outs from his life story—part of which is provided in the article that follows are that:

1. You don't need to come from money to make a successful career
2. Hard work is terrific, but to have success, you need a unique vision—often to 'follow the road less travelled' as in the Robert Frost poem
3. Never stop wanting to innovate and learn
4. Choose your partner carefully and look after him or her
5. Be proud of where you came from

Gary doesn't enjoy overseas travel (Sydney is an awful long way from Texas)—but he spent a week with Australian veterinarians in 2012), and gave a unique series of talks not only about feline medicine but how to run an incredibly efficient and lucrative private veterinary practice. We hope you enjoy the article that follows.

—Richard Malik, Veterinary Editor

When Gary D. Norsworthy, declared he wanted to specialise in cats in the late 1960s, many of his classmates at Texas A&M University College of Veterinary Medicine (TAMU-CVM) thought he was crazy. Cats weren't embraced as a family pet, and many people possessed a 'goldfish mentality' toward them; if a cat fell ill, people would just get another one because they were free.

This didn't stop him from pursuing this field.

'I was just attracted to cats,' he said. 'They were clean. They were not noisy like barking dogs. They were affectionate, but they were not blubbery-kind-of affectionate. That just resonated with me.'

His passion for cats and his decision to pursue veterinary medicine stemmed from the same experience. He worked at a small animal clinic between his second and third year of undergraduate studies at Abilene Christian University (ACU) in Abilene, Texas. Upon returning to ACU in the fall, he completed his pre-vet prerequisites and applied to veterinary school at TAMU-CVM. He was not accepted. The selection committee recommended he attend Texas A&M University because of its larger student population. Not being a quitter, he did just that.

The summer before he enrolled at TAMU in 1968, he and his wife Linda of 52 years got married.

'She has been with me through the ups and downs of my career, of parenthood, and of life in general,' he said. 'She is the wind beneath my wings.'

He and Linda arrived at College Station as newlyweds, and young Norsworthy vigorously studied during his first year. He applied a second time and was accepted. When he graduated from TAMU-CVM, he wanted to work at a feline-only practice, not realizing there were only three in the nation. He applied to two and only heard from one, The Cat Clinic of Seattle.

The Cat Clinic of Seattle

'I was definitely the best applicant because I was the only applicant,' he said. 'Nobody else would take the job.' He easily landed the role, and they moved to Seattle in 1972. He and Linda had never traveled anywhere and didn't grow up in wealthy families. *'When we told our parents we were going to Seattle, we might as well have said, "we're moving to Russia"'* he said. *'It was like another world, another country. They were all in shock.'*

They took advantage of Seattle's outdoor amenities, exploring the mountains, walking through waterfalls, skiing, and riding on ferry boats.

While he enjoyed the city, he also faced the sobering facts about cats. They were vaccinated for only two diseases: panleukopenia and rabies, the latter of which was not mandated by law in the majority of states. The feline leukemia virus (FeLV) had just been discovered, but veterinarians knew or understood little about the condition. His clinic made strides and was one of the first to know of an IFA test and use it.

Dr. Norsworthy's colleague and mentor Bill Freitag, DVM, was frank with him.

'Dr. Freitag told me "There's a whole lot of things we don't know about cats"', Dr. Norsworthy recalled. *'There are a lot of diseases that haven't been discovered, and a lot of the stuff we think we know is not right. We're always keeping our eyes open for new ways to do things because some of the things we're doing aren't working.'*

Dr. Freitag's statement reminded Dr. Norsworthy of a quote by one of his favorite veterinary professors: *'We have bad news, and we have worse news. The bad news is half of what we're teaching you is wrong. The worst news is we don't know which half it is.'* These discouraging statements coupled with Dr. Norsworthy's passion for cats drove him to play a role in the discovery process and spread his knowledge with the veterinary community, beginning in 1977 with his contributions to *Feline Practice Journal* about FeLV's diverse presentations.

Alamo Feline Health Center

After spending two years in Seattle, he returned to Texas in 1974 with this newfound perspective of cats. He became co-owner of Acres North Animal Hospital, a small animal practice in San Antonio, with Don Miller, DVM. In 1995, Dr. Norsworthy became a charter Diplomate of the American Board of Veterinary Practitioners in the feline category. Five years later, after 25 years of working together, Dr. Miller retired and Dr. Norsworthy opened Alamo

Feline Health Center, the first (and currently only) feline-exclusive practice in south Texas.

Dr. Norsworthy and his two full-time associates treat approximately 200 cats per week. One of the benefits of owning a feline-exclusive practice and using electronic medical records for over two decades is his ability to hone in on his research and discovery process. When he conducts studies and publishes articles, he pulls data from 100 to 500 cats. This is significant relative to most other small animal studies and reflects his impressive caseload.



Figure 2. Dr. Norsworthy owns the only CT scanner in a feline-only practice in North America

Vomiting cats are not normal cats

One of Dr. Norsworthy's major contributions to the feline species is his breakthrough research in chronic small bowel disease (CSBD). Prior to his research, chronic vomiting in cats was considered normal, and CSBD was largely not diagnosed or misdiagnosed. His research findings, which reported on 300 cases, were published in *JAVMA*, and showed that chronically vomiting cats have significant small bowel disease.

'The two papers in JAVMA have opened a whole lot of eyes to the fact that these vomiting cats are not normal cats,' he said. *'The most satisfying thing I've done in my whole career is to show that chronic disease of the small intestine causes chronic vomiting in the vast majority of vomiting cats.'*

Other advancements include proving that ACE-inhibitors are safe for cats with kidney disease (Lavallee J.O., Norsworthy G.D., Chew D.J. *Safety of Benazepril in 400 Azotemic and 110 Non-Azotemic Client-Owned Cats (2001-2012). (2017) J Amer Anim Hosp Assoc. 53(2), 119-127*) and that tight- and traditional-control approaches for cats with diabetes are no better than loose-control. In fact, loose control provides several advantages to the cats and their owners. His 2019 research paper on

this topic (Restine LM, Norsworthy GD, Kass PH. Loose-control of diabetes mellitus with protamine zinc insulin in cats: 185 cases (2005-2015). *Canadian Veter J.* 2019;60(4):399-404) which boasts a sample size of 185 and an 11-year length of study, is the only published paper on loose-control of feline diabetes. These are just two of countless feline diseases and conditions he has played a role in advancing.

With the mindset that there is still much to be discovered about cats, Dr. Norsworthy's medical research continues. He is in the middle of four studies and seeks better treatments for two diseases: chronic rhinitis and lymphoplasmacytic stomatitis.

'I've said for many years that I can't retire until I figure out a way to conquer stomatitis, and I think I'm getting close—close to conquering stomatitis but not retiring,' he said.

In the meantime, Dr. Norsworthy has shared his knowledge with the veterinary community, serving as a major author of 7 books, including *Feline Practice*, one of the first definitive textbooks of feline medicine, and *The Feline Patient: Essentials of Diagnosis and Treatment*, a worldwide reference that is in its fifth edition and has been translated into Spanish, Portuguese, Japanese, Italian, Chinese and Korean. He has contributed more than 50 peer-reviewed publications to journals on a plethora of topics in feline medicine, such as hyperthyroidism, chronic small bowel disease, blood collecting and diabetes, just to name a few.

Over the past 15 years, his expertise has attracted numerous national and international veterinary students to serve as externs at his clinic, where they gain access to cutting-edge diagnostic and treatment equipment and experience in delivering quality feline medicine, managing a feline practice and conducting research in feline medicine. In recognition of his work with students, he is an Adjunct Professor at two U.S. veterinary colleges.

He also has hosted veterinarians from across the globe, helping advance standards of veterinary care in Brazil, China, Finland, France, Mexico and Slovakia, among other countries. Dr. Norsworthy doesn't just teach at his clinic, as he has presented more than 600 lectures at veterinary associations and conventions in the U.S., Canada, Australia and Brazil.

Dr. Norsworthy believes his strength is that he understands what works and doesn't work in private practice. His goal is to provide practitioners with a proven technique that they can implement into their practice when they return to work on Monday.

He earned the 1992 Companion Animal Practitioner of the Year Award, the 1997 Outstanding Alumnus Award of the companion animal medicine category, the 1998 Clinical Referral and Consultation Award and the 2009 Medical Specialty Practitioner of the Year Award.

**This article was adapted from an article first published in 'The Texas Veterinarian'.*

Note: Gary has been a prolific and generous contributor to the C&T Series over the years, contributing 18 C&Ts since 2008.

For example, read here:



eBook download
C&T NO. 5451 - 'Honest Doc, he was normal yesterday...',
Issue 278 March 2015



eBook download
C&T NO. 5364 - Those frustrating vomiting cats
Issue 273 December 2013



eBook download
C&T NO. 5311 - A blade of grass causing respiratory distress and epistaxis in a cat
Issue 271 June 2013



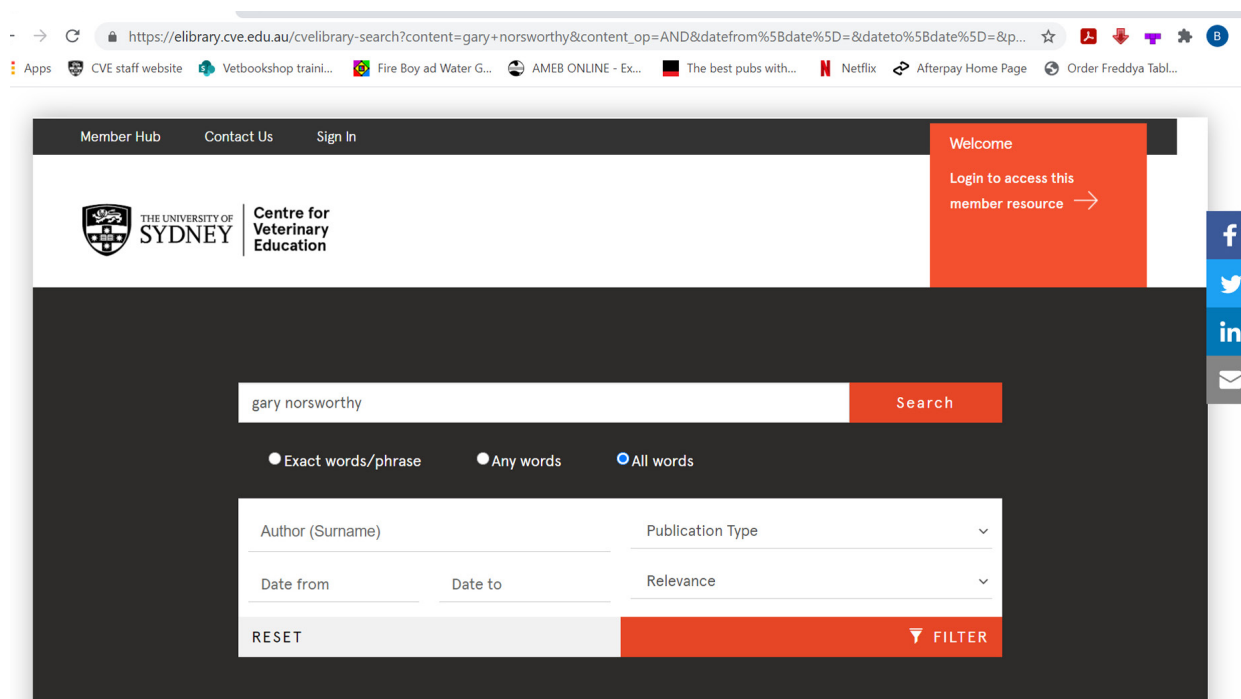
Figure 3. Gary using a surgical laser in the oral cavity

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Open-Access Resources – FREE to everyone

Mental Wellbeing Hub

Watch this heart-felt and at times excruciatingly honest presentation. Dr Yumiko Kadota worked as a plastic surgery registrar until she found herself burnt out and unable to negotiate safer working hours at the hospital. Eventually she resigned in June 2018 and became hospitalised after continuing to decline in physical and mental health. She now works as an academic in anatomy and advocates for wellbeing amongst health professionals. Her book was launched on 11 March 2021.

Replies & Comments

TWO SIDES TO EVERY STORY

Reply to C&T No. 5867

5th Commandment of vet med: thou shalt never force an animal to fit a blood test result

Pete Coleshaw

Jaffa's Health Centre for Cats

Salisbury, United Kingdom

jaffa@jaffavets.com

C&T No. 5880



eBook download – C&T NO. 5867

5th Commandment of vet med: thou shalt never force an animal to fit a blood test result

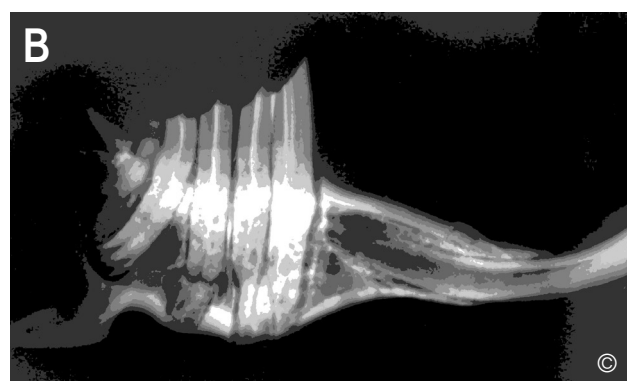
Editor's Note: Thank you to Pete and Aine for the huge effort involved in producing this C&T article. Highly motivated vets and keen and generous contributors to the C&T Series, they take the time and effort to write up their cases, experiences and opinions to share with their colleagues.

Although they disagree on many points, they have supplied considered and informed arguments about a topic that is controversial and often polarising.

Thank you on behalf of our members and readers. Both have won a CVE\$100 voucher.

I write in response to C&T No. 5867 regarding a dog who sadly died of bowel-perforation after consuming a sawn piece of raw bone.

Let me start by introducing myself and providing my take on 'raw feeding'. Forty years ago, I was a (UK) mixed farm/equine/small animal vet who was horrified to visit a particular farm that threw its calving losses onto the yard for the dogs to consume. It was against everything I had been taught—and yet funnily enough those dogs seemed to thrive on their diet. Over the years, they never came in for repair of the bone-induced injuries I had been taught to expect.



Figures 1. A & B. Effects of a non-biologically appropriate diet on the dentition of rabbits

In the days of 'bunny-muesli', I learned that 90% of rabbit diseases are diet related—a fact that has become generally accepted across the rabbit-vet fraternity in the interim, with a commensurate move away from the biologically-inappropriate diets such as rabbit cubes and the like (I understand that the same concept has now been accepted on the equine front—feed fibrevores as fibrevores and do not stuff them with hard feeds full of corn and oats and expect good gut health!)

I then asked myself whether the same applied to our pet carnivores and concluded that this was indeed the case, reinforced by reading the words of wisdom from your own Australian raw meaty pioneer, Tom Lonsdale—everything made perfect sense. At the time, I was a new partner in my practice, with big loans, so I had previously been a promoter of kibble and cereal-based dog and cat-foods, attracted by the profitability of food sales. Then I 'saw the light' (at least in my eyes). I now have over 20 years of raw-feeding experience under my belt, both from my own pets, and also from all my clients' cats and dogs, many hundreds of which I have converted to a more ancestral diet.

So, back to the article. Pet death is very emotive—for both the pet owner and for us vets. When tragedy happens, it is very easy to point blame without considering the bigger picture—and when bones are

involved, the cause and effect is so immediate that very strong feelings are aroused. However, whilst most nutrition-related disease is insidious, it can be every bit as lethal as an improperly chosen bone. Is it any less of a tragedy when a cat dies from acute on chronic renal disease due to ureteroliths resulting from feeding kibble?

I therefore approach this topic with extensive experience of both sides of the story.

As veterinary surgeons, we are actually animal scientists. Therefore, we should look at all the evidence in an unbiased manner and be prepared to have our views influenced by the 'facts'—independent 'facts' and not just those generated by vested interests in either of the (polar-opposite) camps.

One of my personal tenets is that any discussion of risk is entirely meaningless without some evaluation of the risk. How often have you read the statement that 'feeding raw food to dogs can endanger the life of your dog, yourself, and your children'? Scary stuff.

But let us look at the stats from America's Centre for Disease Control, (CDC) considering the number of people who fall seriously ill from *Salmonella* where their pets are also *Salmonella* positive (from whatever cause). We find that over 7 years, with population of 300 million people—so that is 2,100 million human years, there were just 7 incidents! You have a greater chance of being struck by lightning (by a factor of 2).



Figure 3. Sawn, cooked 'chop bone'

It is clear that the dog in C&T No. 5867 died as a result of feeding **sawn** bone – something to be generally discouraged. This is not a natural, out-of-the-wild risk. Certain sawn bones are exceptionally dangerous—lamb and pork chop bones (vertebrae) are my main hate, even when fed raw. However, I would (and do) give my dog a rack of raw lamb rib-bones any day without fear, with soft cartilage cushions on all the ends. Having said that, sharp splinters are not the problem—as carnivores crush and shatter bone into digestible pieces that will not survive gastric acid. It is spiky pieces in unnatural shapes (e.g., vertebral bodies with their various processes) that can be swallowed whole that are the problem.

Any food that you feed your pet will have a risk of a fatal food-induced disease or injury. Brachycephalics can choke on kibble, formulations can be incorrectly constituted, novel or contaminated ingredients can be included with unknown consequences, and the list goes on. The most recent scary examples are the unexplained megaesophagus cases in Latvia and Australia, presumably due to autonomic neuropathy from feeding premium kibble to dogs. Despite extensive investigations the putative neurotoxin has not been identified despite using the best internists as consultants. One should not condemn and avoid a way of feeding based upon an adverse reaction, even if fatal. I once investigated the death of 80 lambs due to monensin toxicity—someone had keyed in a decimal point incorrectly, overdosing by 1000 times. How often does this happen with ingredients that are less lethal than monensin?

'The second aim of this essay is to highlight the need for vets to give full disclosure advice when they recommend raw meat and bones'

I agree totally—and we should apply this to processed diets too. However, do 'mainstream' vets ever 'give full disclosure' of the potential harmful effects of the diets they recommend to clients? Do they have any real knowledge of the potential 'side effects' processed diets can cause, and the

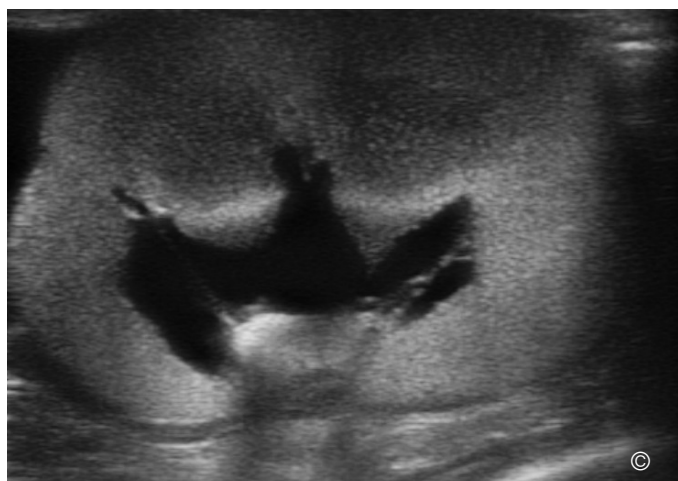


Figure 2. Hydronephrosis resulting from oxalate urolithiasis causing ureteral obstruction

frequency with which they occur? I have yet to come across a vet that advises about the risks of kibble when selling the product. Is that through lack of knowledge? Or lack of time. Or bias from the desire to make a lucrative sale?

I personally do not believe that any responsible vet should make any dietary recommendations of any description without knowing, nor discussing the current diet, its benefits, deficiencies, and potential shortcomings and the same factors for the proposed new diet.

We 'raw feeders' tend to be very impassioned about the subject and have generally studied it at length and are usually aware of adverse issues with both processed and raw foods. I will come back to the risks of raw and how these can be mitigated, but first let us take a dispassionate perspective on mainstream kibble and be aware just how dangerous it can be.

Here are a few references to recent incidents:

- i. [Excessive sulphite preservatives in 'pet mince' and food rolls resulting in life-threatening thiamine deficiency in cats and dogs – Read here](#)
- ii. [Kidney disease in dogs \(Fanconi like syndrome\) in dogs especially small dogs fed chicken jerky treats sourced from China – Read here](#)
- iii. [Demyelinating brain and spinal cord disease fed irradiated cat food sourced from Canada \(the irradiation was the trigger\) – Read here](#)
- iv. [Hypervitaminosis D resulting in life-threatening hypercalcaemia in several brands of canned cat food – Read here](#)
- v. [The presence of indospicine in dog food made from camel meat resulted in liver failure in dogs in Western Australia – Read here](#)
- vi. [Megaoesophagus in dogs fed Advanced Dermcare food made by Mars in Australia – Read here](#)

So, it is clear that serious and ultimately fatal processed-food-induced disease is not a rarity. Precious little can be done by owners to avoid most of these problems, though most are blissfully unaware of the inherent risks.

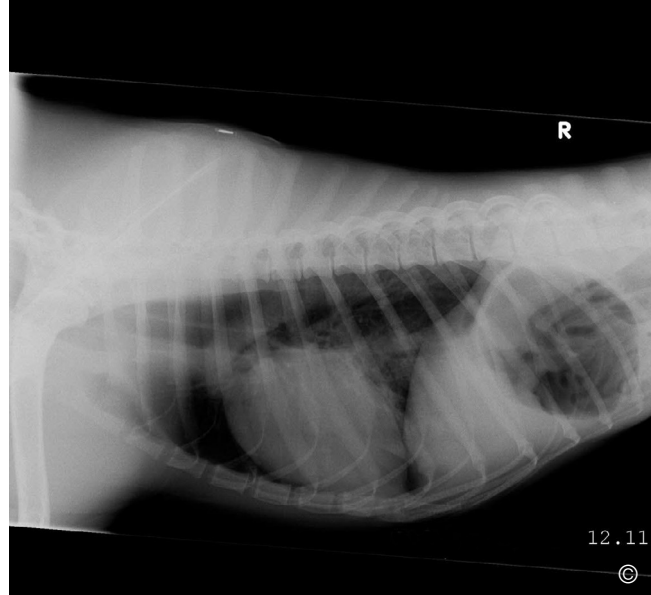


Figure 4. Canine megaesophagus

As a promoter of the prey-model biologically appropriate carnivore diet for over two decades, let me explain my experiences of both potential and real issues of this way of feeding; experiences which are shared by most of us who take the 'natural' approach.

The aim is not to feed a natural diet, as feeding live prey is hardly socially or ethically acceptable. But cats and dogs have evolved to eat prey, bones, and all. Selective breeding for phenotypic traits has not altered their fundamental biology, and the key to this way of feeding is simply to mimic 'wild' as closely as possible.

In the wild, prey-species are neither sawn nor cooked, and our domestic carnivores are not challenged by feeding meaty bones of an appropriate size and nature.

Occasional dental fractures result—often the tips of the cheek teeth in cats, and the lateral aspect of the upper carnassial, usually in dogs. However, I reckon I have extracted 200 times more teeth due to pyorrheic periodontal disease than I have seen carnassial slab fractures. And when these slab fractures occur the teeth can usually be saved by root-canal therapy, unlike teeth that are severely affected by periodontal disease.



Figure 5. A & B. Endodontic treatment of carnassial slab fractures. (Photos courtesy of Dr Alexander J Smithson BVM&S)

This dietary-induced dental disease CAN be repaired!—as opposed to the sewer mouth! How often do GP vets see dogs with stinky 'sewer-mouths' where many of the teeth can be extracted by a simple finger-pull? These mouths are not just distasteful—they can have serious toxæmic effects on the whole body (multiple body systems) and create major welfare issues. I have yet to see this oral presentation in a raw meaty bone-fed dog. When I raised the issue of raw bones versus cooked on a major international forum, it was clear that most of our colleagues were unaware that cooked bones are a different product, and no-one seemed to ask 'the question' when bone-issues were experienced. More alarming to me was the view of one vet that he did not really care, as he was not going to recommend raw. Is this the attitude of enlightened professionals? Who was it who said that science does not really care what you think?

So general recommendations are that bones are of a physiological size (e.g., rabbit/chicken for a cat) and for small carcasses, disarticulation is the best way of dividing a carcass. As we have said, sawing can lead to very un-natural multi-spiked pieces almost designed to get stuck—chop-bones are a perfect example. And on the subject of slab fractures, avoid

beef marrow bones—a popular choice perhaps, but the dense cortical bone of the femur is not the most appropriate bone for even the largest dog.

Choking on raw is not a common experience. I would avoid meaty bones for brachycephalics, but I would personally never have a brachycephalic mutant anyway. Plenty of pets have choked on kibble. Oh, and do not feed necks—too much thyroxine!!—but a risk so easily avoided once you know.

'Contrary to Aine's assertion, in the UK most vets who recommend raw are very keen to discuss risk mitigation, especially in a veterinary world so hostile to this way of feeding. As a founder member of the worlds' first raw-feeding veterinary group, the UK Raw Feeding Veterinary Society (RFVS), I confirm that our main (and stated) aim is the education of pet owners, and of vets who choose to engage with the science of nutrition.'

I personally feel the biggest danger for pet owners is the general lack of engagement of the profession with those owners who choose to feed alternatives to highly processed convenience foods. So often, these clients dare not declare the nature of their pets' diet through fear of being chastised or worse, and they therefore do not get informed appropriate advice from their vet.

In the UK, many corporates and vet schools are now treating raw-fed pets as potential bacterial time-bombs, insisting on barrier nursing them when an in-patient. And yet we know that a considerable proportion of the cats and dogs we see and hospitalise every day (maybe 10–25%?) are carrying the same bugs that are so feared, and they are totally asymptomatic. The same clients and vets do not worry if their cats catch and eat small prey animals and birds which also contain these bacteria. If carriage of these 'potential-pathogens' is so dangerous, should we not be barrier nursing every in-patient? Indeed, should we have them in our houses sharing our lives?

When humans do contract pet-associated bacterial diseases, does anyone ever ask, or report, the hygiene standards of the human patient? In a recent UK case of Shiga-toxin, the owner was sharing his toothbrush with his dog!! We accept chicken carcasses in the home knowing that 50% are contaminated with *Campylobacter* spp, knowing that we can successfully mitigate most of the risks. It is no different with raw feeding. But it needs client education – preferably by someone who has had extensive experience of feeding raw.

So, Aine, I do not think all our pets will ever be safe. Life can never be so certain.

But if we are to give dietary advice, we should firstly know the real risks—and have an idea of their frequency. Over the last 20 years of raw-feeding I have had one serious raw-related incident—oesophageal obstruction, but the cat had a concurrent life-threatening acute coagulopathy, so I suspect other factors were at play. I have seen 4 cases of renal/ureteric stones requiring investigation for subcutaneous ureteral bypass (SUB) surgery in the last 12 months alone and have treated maybe a dozen diabetics over the same timescale.

Surely it is not logical to condemn a manner of feeding because of the rare occurrence of adverse incidents. If we did, both we and our pets would have starved ourselves to extinction by now. So, let us bring on the science and look at the real evidence. Let us educate ourselves to offer dietary alternatives from a position of genuine knowledge.

So, I make my plea—can we please discuss this topic in an objective manner. Raw is not a universal panacea, but neither is kibble a totally safe, risk-free way of feeding our pet carnivores.



Figure 6. A & B. 'Sewer mouth'

REPLY: NOT ALWAYS.

Aine Seavers
Oak Flats Vet Clinic
58A Central Ave
Oak Flats NSW 2529
reception@oakflats.com.au

C&T No. 5881

I wasn't going to reply initially.

My article was the reporting of a factual event. There are no 'two sides to it'. It happened as clinically recorded. This case history was not something that needed rebuttal.

But then I read:

'At the time, I was a new partner... with big loans, so... a promoter of kibble, attracted by the profitability of food sales. Then I 'saw the light' (at least in my eyes).'

And I thought: 'Here we go again; the subliminal dig that my decisions, and those of other non Raw Meaty Bones (RMB) diet vets, must be driven by profit not prevention and hence I/we are "blind to the light"'.

It gets a bit tired, where this old chestnut-of the self-congratulating reformed 'sinner' reflecting their own old sins back on to the opposing party character, raises its head.

Ask anyone, but especially ask any NSW sales Reps who have ever dealt with my clinic: All will rapidly and urgently advise against ever mentioning profit as a means to get my attention or my support for their product.

Neither I nor my opinion have ever been for sale, so let's bury that insinuation right now. If the vets feeding raw do so because they once committed the sin of Money-Over-Medicine, then that is their burden to bear. That was never my sin nor that of many of my group. I have spent the last 30 years defending my integrity and my colleagues against those vets in the RMB following who chose to use this tactic—it is sad I still have to keep doing it. Reading on, I was then puzzled by some of the references the author supplied to support his argument. Most Australian vets will be aware of my involvement from Day 1 in raising awareness of the diseases reported in your supplied references. So here goes my response.

'therefore approach this topic with extensive experience of both sides of the story.'

I wrote the article from the perspective of having extensive experience in the mid-80s, prior to Premium pet food access. I have seen the effects of the raw/cooked/tinned food and bones, the raw food movement of the early 1990s, and non-premium, woeful commercial diets. I have worked in the UK with pets affected by their appalling nutrition, whose diseases disappeared once they were moved onto super-premium foods like Hill's. My rescued pets went on Hill's diets and no longer were the chronically ill patients surrendered by their original owners who didn't believe diet not drugs were the answer to their pets' illness.

Many decades later I continue to heal with diet, releasing many pets from constant drug therapy and in some cases avoidance of chemotherapy, just by using diet.

My extensive highly profitable, orthopaedic puppy disease surgery list wilted away as Hill's puppy food made so many nutritional bone diseases disappear.

For many years now, I have received regular thanks from veterinary surgeons around the world for changing how they practice veterinary medicine so as to better resolve several chronic conditions especially otitis, anal sacculitis and dermal mast cell tumours. These vets report happier clients, healthy pets and a lower level of stress and anxiety in the vet's own day.

This week I euthanased a 13-year-old Weimaraner. He came to me at 6-years-old, with owners in desperation because of his chronic illness. I used only diet to heal him. He was never ill again. The owner's thanks included this profound comment: *'Thank you for your customised nuanced approach that used food (Hill's) to free our pet up to live a wonderful long life drug free pain free life.'*

'Pet death is very emotive—for both the pet owner and for us vets. When tragedy happens, it is very easy to point blame without considering the bigger picture—and when bones are involved, the cause and effect is so immediate that very strong feelings are aroused. However, whilst most nutrition-related disease is insidious, it can be every bit as lethal as an improperly chosen bone. Is it any less of a tragedy when a cat dies from acute on chronic renal disease due to ureteroliths resulting from feeding kibble?'

Equally as distressing is when a dog dies from a ruptured bladder due to the huge meals of bones that was its diet. Equally distressing to see cats with disease from cheap bad dry food about which—you can rest assured—I will alert and warn my clients in full when the issue is raised.

However, let's not detract from the issue here: my perfectly healthy patient in the article died because her diet was changed from Hill's dry food to include raw bones.

Let's own that and not try to dilute the horror by pointing the finger at other disease issues.

'As veterinary surgeons, we are actually animal scientists. Therefore, we should look at all the evidence in an unbiased manner and be prepared to have our views influenced by the 'facts'—independent 'facts' and not just those generated by vested interests in either of the (polar-opposite) camps.'

As a vet and scientist, I can assure you I always look at any issue 360 degrees and fully inform myself of all the facts before making any decision. I might only be a general practitioner in a small clinic in remote Australia but that doesn't mean either my reading or indeed my own research would be sub-par. Compared to some, my 17-peer reviewed published papers, the majority as primary author and maybe only just 100+ more publications via C&T and The Veterinarian might not amount to much, but to most vets, it does suggest I am more than capable of already having a fully rounded, up-to-date and sound science-based opinion.

'One of my personal tenets is that any discussion of risk is entirely meaningless without some evaluation of the risk. How often have you read the statement that 'feeding raw food to dogs can endanger the life of your dog, yourself, and your children'?

Scary stuff.

But let us look at the stats from America's Centre for Disease Control, (CDC) considering the number of people who fall seriously ill from Salmonella where their pets are proven to be Salmonella positive (from whatever cause). We find that over 7 years, with population of 300 million people—so that is 2,100 million human years, there were just 7 incidents! You have a greater chance of being struck by lightning (by a factor of 2).'

The data from the UK and Canada is more informative so I have supplied many references you might like to read. These veterinary based papers are

more relevant and expand not just on Salmonella but other infectious agents—it seems this risk is a risk I would like to avoid for my clients.

'It is clear that the dog in C&T No. 5867 died as a result of feeding sawn bone—something to be generally discouraged.'

ALAS: SAWN bones are almost the norm here in Australia.

There are promotional videos of the raw diet vets and their staff using these saws to cut carcasses for sale.

Raw meat is for sale out the back of some RMD vet clinics. One such set-up up looks to all intents and purposes to be a meat processing plant. Aside from huge amounts of beef/deer/meat, at least an additional '2 tonnes of chook carcasses a month/600 kg of quail carcasses monthly' was cited by a RMB vet as what he sold directly to owners. In my opinion, the sheer mass of pet food being supplied through that clinic alone makes them a de facto pet food producer and ispo facto vets who make profit from selling pet food. That is a lot of income from pet food.

Yet when I make a paltry profit from a bag of dry food (and lose the profit I could have made if I had not advised the food thus allowing these pets get some pretty nasty and expensive diseases I could prevent)—then somehow I am the 'corrupt/unenlightened profiteer'. Such a double standard!

'One should not condemn and avoid a way of feeding based upon an adverse reaction, even if fatal?'

I agree

- But when it is more than one...
- But the supporters of that food (RMB should admit that there are adverse reactions
- But when they do not do as I do when there is a problem with any diet—raw or cooked—which is to move heaven and earth to alert the public and work with all parties to have the offending food removed—then I have to accuse some of double standards.

'The second aim of this essay is to highlight the need for vets to give full disclosure advice when they recommend raw meat and bones (quote from C&T No. 5867)

I agree totally—and we should apply this to processed diets too. However, do 'mainstream' vets ever 'give full disclosure' of the potential harmful

effects of the diets they recommend to clients? Do they have any real knowledge of the potential 'side effects' processed diets can cause, and the frequency with which they occur? I have yet to come across a vet that advises about the risks of kibble when selling the product. Is that through lack of knowledge? Or lack of time. Or bias from the desire to make a lucrative sale?'

Again—another inference that non RMB vets are drive by desire for sales over a desire to create a customised solution. These inferences need to stop.

I alert my clients to ALL health concerns.

We have our own Vet clinic App. Using it, I immediately and routinely send out health alerts for ALL foods, be it raw, cooked, dry, tinned, Black Hawke, Jerky treats etc, even if they are not foods we recommend. When there was a Hill's issue and a Royal Canin issue, the alert was sent out just as fast. We follow up with Facebook and Website alerts and with regular updates as new information becomes available. Several of my colleagues/competitors will link to my page to help share the alerts which is how it should be.

My clients love that we have their backs 24/7.

How many raw food vets shared the alert on the work on Acute Polyradiculoneuritis (APN)?

I didn't find any RMB persons alerting the clients to this risk. Hopefully for client and pet safety alone, evidence can be provided to the contrary.

However, I did find comments on a general vet forum from a RMB vet deriding the findings and calling the University work part of an 'utterly corrupt, "research" contaminated with bogus belief systems and their findings unreliable'.

Sadly, I did find a Facebook discussion on a RMB vet page where one of the RMB vet clients, reported very respectfully back that—whilst their vet was dismissing the link—their own pet had contracted said condition by feeding food advised by the vet. Let's just say the response that client got back from that vet was less than stellar.

I am accused of being blinded by profit and by lack of knowledge and of 'selective' alerts. Now you know the reality is so different. I would suggest it would be better for you to investigate the track record of the RMB lobby effort re alerts on their advice, rather than you continuing to suggest mine and others are sub-par.

Your reply does not mention APN; did this issue even reach the UK RMB advocates' ears? If not, I take comfort that now you are aware you will alert your members.

You are the first RMB vet I have known to alert regarding the link with diet-induced hyperthyroidism and chicken necks. I didn't initially see any silver lining in my dead dog case report. If now it becomes the means by which UK vets now know about APN then some good has come from such tragedy.

'I personally do not believe that any responsible vet should make any dietary recommendations of any description without knowing, nor discussing the current diet, its benefits, deficiencies, and potential shortcomings and the same factors for the proposed new diet.'

We 'raw feeders' tend to be very impassioned about the subject and have generally studied it at length and are usually aware of adverse issues with both processed and raw foods.'

Your inference that the non-raw feeder is not the above is misinformed and offensive.

Again—read how I and so many other vets approach these issues and become enlightened here that your group does not have the monopoly on these desired traits

'I will come back to the risks of raw and how these can be mitigated, but first let us take a dispassionate perspective on mainstream kibble and be aware just how dangerous it can be.'

Here are a few references to recent incidents:

- i. ***Excessive sulphite preservatives in 'pet mince' and food rolls resulting in life-threatening thiamine deficiency in cats and dogs*** [Read here](#)

The rawness of the pet mince is the issue here—hence why unscrupulous sellers poison it with the sulphites. This is an example of *RAW food being a problem*—not commercial dry food.

Not sure why you would use this to support your approach as it reflects on what you advise, not what I advise to be fed.

I have copies of these alerts all over my clinic walls.

Food rolls; the name is an oxymoron. The majority of these rolls are junk not food and not something I ever approved of.

- ii. ***'Kidney disease in dogs (Fanconi like syndrome) in dogs especially small dogs fed chicken jerky treats sourced from China.*** [Read here](#)

Are you aware that I and another Vet Grahame Baker wrote the C&T alert that started the ball rolling and eventuating in the chicken jerky product being removed by all supermarkets from all supermarket shelves? [Read here: Warning - Potential new threat to pets!](#)

We sounded the alarm in the September 2008 C&T within hours of realising the link.

I then spent 4 months of my life, many hours a day, unpaid, chasing down information, collating data, being warned off because the lawyers would come for me and take everything if I raised too much of a stink. Despite the pressure, I persisted and with a great group of other vets joining us, we had the product exposed on national TV and had the product withdrawn. I include some extra references you might like to read as an example of how preventative medicine must be at the heart of all we do.

- iii. ***'Demyelinating brain and spinal cord disease fed irradiated cat food sourced from Canada (the irradiation was the trigger).*** [Read here](#)

If you would like to see the private correspondence from several of the authors of this paper to me acknowledging my support and encouragement and liaison ability from Day 1, then happy to share.

The issue occurred because this was a boutique food appealing to the *RAW food market*. It was not cooked as normal foods are. In fairness though, it was the irradiation by AQUIS not the food *per se*, raw or semi-raw, that caused the problem.

I have included a copy of the original Orijen press release so you can familiarise yourself with what actually happened. [Read here](#)

Regardless, it is an example of how I put prevention first for all animals, even ones that are not my clients or from a food I would NEVER have recommended.

- iv. ***'Hypervitaminosis D resulting in life-threatening hypercalcaemia in several brands of canned cat food'*** [Read here](#)

I intensely dislike tinned food with a vengeance—an eco-unfriendly way to transport 90% water around the country.

- v. ***'The presence of indospicine in dog food made from camel meat resulted in liver failure in dogs in Western Australia'***
[Read here](#)

Are you aware of the work we did in one of the best-read C&T publications *'Toxic Tales from around the World'*—of which I was the originator and Guest Editor? Given this was a uniquely Australian problem, we got it out through the C&T to Australian vets one FULL year before the paper listed below was published. **[Read here](#)**

- vi. ***'Megaoesophagus in dogs fed Advanced Dermcare food made by Mars in Australia'*** **[Read here](#)**

I have never recommended Advance. All pets' foods are not equal. I do not like the salt level in many of them, including Advance. An extraordinary article was done by a final year vet student for C&T in which the dangerous salt levels of many foods are listed. This has pride of place on my clinic walls. **[Read here](#)**

I also intensely dislike another Mars Product: Greenies. Is this toxic treat on its 4th or 5th reincarnation? I was appalled at the bad science behind versions 2 & 3 so ran the rep from the room when they tried to flog version 4 to me. This product has made many dogs very ill. I will not let it darken my clinic door.

All commercial food is not the same.

Not recommending raw doesn't mean it is open slather for any alternative to be fed. There are commercial diets out there that I abhor at a level that even a RMB person would be taken aback at, so intense is my dislike of particular brands of pet foods.

I just don't understand how the RMB lobby fails to understand the approach taken by me and many other vets: **[I recommend only those foods that pass my scrutiny and are appropriate for the pet, the breed, age and co-morbidities of the pet in front of me.](#)**

'The aim is not to feed a natural diet, as feeding live prey is hardly socially or ethically acceptable. But cats and dogs have evolved to eat prey, bones, and all. Selective breeding for phenotypic traits has not altered their fundamental biology, and the key to this way of feeding is simply to mimic 'wild' as closely as possible.'

So, if humans are even less phenotypically altered than modern pets, why are we not eating the above diet? We don't eat 'wild' because we, like our co-evolving companion pets, have progressed to a more advanced diet.

'And when these slab fractures occur the teeth can usually be saved by root-canal therapy.'

Sadly, slab fractures are rarely detected in time and even then, most Australia clients won't finance root fill for their dog's tooth. The very reason the tooth was left is many of the owners feeding bones think vets are also an unneeded evolutionary aid and don't seek veterinary attention in time.

Periodontal disease: We do hardly any dentals in our practice—mostly because of the good dry food and appropriate home foods we recommend to our patients.

Sewer mouth is rarely seen in dogs on a diet free of tinned food, free of slop rolls, junk pet treats and junk human food. Dogs and cats maintained on top level dry diets and good human food do very well. My own 18-year-old-cats went to their deathbed with pearly whites having only ever had Hill's dry diet and cooked chicken meat.

'More alarming to me was the view of one vet that he did not really care, as he was not going to recommend raw. Is this the attitude of enlightened professionals?'

I too find that upsetting. One should care; one should know 360 about what you recommend or what is recommended. I am surprised that vets didn't know that cooked bones are a whole new level of risk. BUT more distressing is a vet knowing about the APN risk and not caring; When the APN alert was first published the glib dismissive response a RMB vet wrote of their sales, of raw bird meat to their clients' pets; *'All laden with Campylobacter, or so we believe'* is in my opinion a far more alarming comment than your example.

'As we have said, sawing can lead to very unnatural multi-spiked pieces almost designed to get stuck—chop-bones are a perfect example. And on the subject of slab fractures, avoid beef marrow bones—a popular choice perhaps, but the dense cortical bone of the femur is not the most appropriate bone for even the largest dog. Choking on raw is not a common experience.'

Sadly, I have been called out to animals with the bone firmly stuck in the now dead pet.

'I would avoid meaty bones for brachycephalics, but I would personally never have a brachycephalic mutant anyway.'

Plenty of pets have choked on kibble.'

Choked and died or just gagged?

Unlike bone-induced death I have only seen dry food gagging, not **dying**.

'Contrary to Aine's assertion, in the UK most vets who recommend raw are very keen to discuss risk mitigation.'

'Oh, and do not feed necks—too much thyroxine!!—but a risk so easily avoided once you know.'

THANK YOU FOR INCLUDING THIS; SO FEW VETS WHO RECOMMEND RAW NECKS ALERT CLIENTS TO THIS RISK.

My article was written from Australian conditions, experienced by Australian Vets, in an Australian publication. I would never be so presumptuous as to comment on conditions in an unrelated country.

'In the UK, many corporates and vet schools are now treating raw-fed pets as potential bacterial time-bombs, insisting on barrier nursing them when an in-patient.'

You can't pick and choose the 'science of nutrition' and then chose to ignore an inconvenient truth like a zoonotic risk. I have had several elderly clients admitted to hospital on multiple occasions (including one patient's son being investigated as trying to kill his mother given she was food poisoned so often—we solved the 'crime' by identifying the source as the #pet bowl/raw chicken/poor hygiene link).

'And yet we know that a considerable proportion of the cats and dogs we see and hospitalise every day (maybe 10-25%?) are carrying the same bugs that are so feared, and they are totally asymptomatic. The same clients and vets do not worry if their cats catch and eat small prey animals and birds which also contain these bacteria. If carriage of these 'potential-pathogens' is so dangerous, should we not be barrier nursing every in-patient? Indeed, should we have them in our houses sharing our lives?'

I am not sure what the English view now is but here *Songbird fever* is as big an issue as I saw when practising in both Ireland and the UK. Owners were not impressed to have their pets catch and eat prey and birds, as much because of the bacterial risk as anything else.

To those owners that didn't realise the disease risks—once they knew of the risks, they were concerned. Maybe more raw feeders need to know more about this risk?

A survey—in the newspaper the day after the Melbourne Study on *Campylobacter*-raw chicken-APN was released—asked Yes/No if one would keep feeding raw chicken: 75% replied they no longer would. To me, that means the risks of feeding raw was not explained to owners and—once they knew the full story—they made an informed decision, and many stopped feeding it.

'When humans do contract pet-associated bacterial diseases, does anyone ever ask, or report, the hygiene standards of the human patient? In a recent UK case of Shiga-toxin, the owner was sharing his toothbrush with his dog!!??? We accept chicken carcasses in the home knowing that 50% are contaminated with Campylobacter spp, knowing that we can successfully mitigate most of the risks. It is no different with raw feeding. But it needs client education—preferably by someone who has had extensive experience of feeding raw.'

There is also a risk of introducing antimicrobial-resistant bacteria as raw food commonly exceeds hygiene thresholds for *Enterobacteriaceae* bacteria which often encode resistance to critically important antibiotics. Raw-fed pets create an elevated risk of shedding such resistant bacteria.

A 2008 study showed a strong association of raw feeding and the shedding of resistant *E. coli* showing AmpC-type resistance amongst therapy dogs in Canada.

Fire away advising owners of the allegedly 'tiny' risks to pets and owners; but prepare for hell when the client's pet dies or a family member falls ill. I know exactly how this plays out because some new clients have come to me and tried to reflect their grief and anger onto me. Those owners held the belief that as ALL vets supported pets eating raw chicken: we were ALL guilty for the preventable death of their fur-kids, especially from APN. Luckily, the many posters around our clinic (some many years old) demonstrated that our clinic had always warned owners about the dangers of feeding raw chicken to their pets. I could defuse

such angry clients because we were innocent and had the paperwork to prove it. Good luck calming an angry traumatised owner reliving the slow suffocation death of their beloved fur-kid when you were the vet that advised that raw chicken.

'So, Aine, I do not think all our pets will ever be safe. Life can never be so certain.

But if we are to give dietary advice, we should firstly know the real risks—and have an idea of their frequency. Over the last 20 years of raw-feeding I have had one serious raw-related incident—oesophageal obstruction...'

You might find these references of blockages of interest:

- Gianella P, Pfammatter NS, Burgener IA. Dec 2009, *J Small Anim Pract*. Oesophageal and gastric endoscopic foreign body removal: complications and follow-up of 102 dogs, Dec 50(12):649-54
- Nemeth, T., Solymosi, N. & Balka, G. (2008) Long-term results of subtotal colectomy for acquired hypertrophic mega colon in eight dogs. *Journal of Small Animal Practice*, 49, 618-624

'I have seen 4 cases of renal/ureteric stones requiring investigation for subcutaneous ureteral bypass (SUB) surgery in the last 12 months alone and have treated maybe a dozen diabetics over the same timescale. Surely it is not logical to condemn a manner of feeding because of the rare occurrence of adverse incidents.'

Yet that is exactly what is done by the RMB and Biologically Appropriate Raw Food (BARF) societies here, but the reverse is not allowed. That is a double standard.

'If we did, both we and our pets would have starved ourselves to extinction by now. So, let us bring on the science and look at the real evidence. Let us educate ourselves to offer dietary alternatives from a position of genuine knowledge.'

I think when you read my references mentioned in the body of this reply plus the additional 35 reference below, you will realise I only ever comment from a position of deep research and genuine knowledge. I have included some extra references to share with you about the risks of raw diet.

'So, I make my plea—can we please discuss this topic in an objective manner. Raw is not a universal panacea, but neither is kibble a totally safe risk-free way of feeding our pet carnivores.'

Now that is something, I can agree on with you. Nothing is 100% safe.

We should be allowed to customise the care of our patients to the best of our abilities and not be accused, when choosing a different path to another, of being driven by Money-Over-Medicine.

If the RMD lobby really does believe in their product, allow the results to speak for themselves and leave the inference that non-RMB believers are profiteers and/or under-informed vets out of the discussion.

I hope having now read my reply:

- You will appreciate that the vast majority of vets are making ethical well-informed decisions
- That the majority of vets know their science
- That they might not agree with you or me all of the time and that is not a bad thing
- That the debate does not need accusation of Money-Over-Medicine
- That the debate does not need a one-sided belief that their side is the sole keeper of genuine science and informed decision making

I believe you are a good vet. I can see that from your reply.

I can see you do more than any other RMB vet that I am aware I know of (there may be a few colleagues who hide that fact from me) to give advice on what can go wrong with RMB and how to reduce risks. Regardless, I will not be replying any more on this topic.

I have other veterinary directions I want to explore in 2021.

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Pete Coleshaw's Reply

Aine

Firstly, thanks for the time you have taken to elaborate and counter my arguments—I very much appreciate your input and thoughts and will go through the references and comments you provided at leisure and will relish my further education. Let me initially point out that my rebuttal was not an attack on your personal integrity, intelligence or knowledge in any shape or form. I was simply intending to kill the idea that all bones are bad along with the inference that RMB should therefore be discouraged.

My further comments, observations and insinuations were intended for the wider profession. Sadly, I see my beloved vocation becoming an industry in which the largest player is now the Mars Corporation. I have had numerous approaches to buy my practice at what I see as a stratospheric valuation. Big finance is now involved and whilst the personal integrity of

the front-line workers is not in doubt there has to be payback to the money-men higher up the line.

It is very apparent from your reply that you have invested as much (and maybe more) thought and effort as I in the field and even though we disagree on some things I reckon we actually have a lot in common, and would welcome further dialogue with you. However, vets like you are the exception in the UK—the only colleagues I come across who have a keen and critical interest in nutrition tend to be raw feeders.

You seem to have a general disdain of the raw brigade lumping everyone together as cranks. There are zealots and misguided individuals in both camps. I have always contended that the most dangerous of diets is a poorly self-formulated raw mix (but equally that the best is a properly formulated raw!). Sadly the rising popularity of this way of feeding is inevitably attracting those with \$\$\$ in their eyes. The industry badly needs regulating. The RFVS are currently involved in the development of an accreditation scheme for raw producers, which has to be the way forward.

Moreover, the group has always believed that education of both vets and pet owners is key. Pet owners will feed raw with or without professional endorsement, and the current situation where they dare not reveal their way of feeding through fear of chastisement is not healthy. There is no pretence that RMB is free of risk—but I really do not believe that the majority of the 'processed' side of the profession accepts that their way of feeding presents any real issues.

So let's discuss the failings of various dietary routes honestly, evaluating real risk not damning foods based on a paper recording half a dozen cases making out it is an everyday happening. Let's encourage the situation where no dietary recommendations are made without the advisor knowing what the current diet is. Moreover, the advisor should have the knowledge to critically evaluate the benefits of the proposed diet over the existing food, commensurate with the diagnosed pathology, rather than blindly 'prescribing' an alphabet diet with a name corresponding to the organ system involved.

It might be a long haul.

Large

WHAT'S YOUR DIAGNOSIS?

C&T No. 5882

Dinky is a 14-year-old Brumby gelding with an abnormal penis.

What is your differential diagnosis? How would you investigate this case further and what are the treatment options?



PLEASE SEND US YOUR ANSWER

Please email your answer to cve.enquires@sydney.edu.au before 1 May 2021. The best answer will be published in the June issue; the winner will receive a CVE\$100 credit prize.

INCONSISTENCIES IN THE PINKEYE (INFECTIOUS BOVINE KERATO-CONJUNCTIVITIS, IBK) STORY (SO FAR)

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Mac grew up on a sheep and cattle property near Bonshaw, northern NSW, and graduated BVSc (Hons I) University of Queensland in 1985. For 5 years he worked as a mixed animal veterinarian locum and travelled before opening Border Veterinary Surgery in Goondiwindi, Queensland in 1990. He achieved MANZCVS in Veterinary Pharmacology in 2012 and Graduated MVS in Small Animal Practice Murdoch University 2017.

Mac is currently a PhD student at the University of Sydney studying pinkeye in cattle with Meat & Livestock Australia (MLA) support.

He is not your average student; having been a fulltime practicing mixed animal vet for 35 years, he owns and operates his own practice with his wife Cathy (also a vet), in Goondiwindi, QLD. He does not think the pinkeye situation on farms has improved in this time, despite many apparent laboratory successes. He wants to fix pinkeye or at least improve outcomes for cattle with this horrible disease.

Why study pinkeye?

Pinkeye has animal welfare issues as well as economic impact.

But isn't the story well-known?

According to the 2015 Meat & Livestock Australia (MLA) report, *Priority list of endemic disease for the red meat industries*.

- Pinkeye is an infection of the eye caused by the bacterium *Moraxella bovis*
- Risk factors for the disease are well understood, and
- Vaccination is available (Lane, Jubb, Shephard, Webb-Ware, & Fordyce, 2015)

Sounds like game over. Pinkeye under control. The clinical reality is nothing like this. In this report southern Australian cattle producers ranked pinkeye the **second most important disease** after parasites (Lane *et al.*, 2015). There has been no obvious improvement in occurrence rates or outcomes for pinkeye since I was a kid growing up on a cattle and sheep farm. Producers are frustrated and looking for direction about the disease.

Pinkeye (also known as infectious bovine keratoconjunctivitis or IBK) is stark evidence of the speciesism of our veterinary profession. 'Every eye is an eye', except apparently if it is a cow's eye. In the *Color atlas of veterinary ophthalmology* by Gelatt and Plummer (2017) pages dedicated to different species are: dogs 182, cats 47, horses 31, exotic pets 12, food and fibre animals (including cattle, sheep, goats, camelids) 16.

Why has IBK, the most important ocular disease of all animal species, not raised the interest of specialist veterinary ophthalmologists?

The conventional infectious bacterial paradigm of IBK is accepted even though it is contrary to many

ophthalmological principles. A pitfall of veterinary ophthalmology is labelling corneal disease as 'eye infection' when primary bacterial infection of the cornea basically does not exist. The mammalian cornea is so highly resistant to infectious disease that, except for viral corneal inflammation, almost all keratitis is triggered by some form of environmental injury. *'We nonetheless are often guilty of drawing excessively simplistic (and usually erroneous) conclusions about the significance of bacterial or fungal isolates in the pathogenesis of corneal disease'* (Wilcock, 2008). If *M. bovis* is the cause of IBK it may well be the only bacterial primary pathogen of the cornea in any species Maggs (2018).

Is it even infectious?

Over the years, reviews have noted that pinkeye and IBK are imprecise umbrella terms that suffer from a lack of case definition. Wilcox (1968) stated IBK was ill-defined and could refer to conjunctivitis, keratitis preceded by conjunctivitis, conjunctivitis preceded by keratitis and keratitis alone. Baptista (1979) noted *'from time to time the name IBK has been applied to nearly all eye conditions of cattle characterized by lachrymation, conjunctivitis or keratitis'*. In other species, such a constellation of non-specific clinical signs would generate interest in more than one condition.

Researchers and clinicians describe the non-specific, predictable inflammatory response seen in eyes with IBK in fine detail as if it were somehow special or pathognomonic. It is not. The eye has a limited ability to respond to noxious stimuli, what is seen is largely stereotypical. They are describing the normal response of the bovine eye to injury. Little has changed since Penberthy (1897) described outbreaks of *'contagious ophthalmia'*, an *'acute inflammation of the cornea and conjunctiva without appreciable cause'* in cattle in England stating *'the symptoms shown are those usually exhibited in inflammation of these parts.'* This has not deterred some (myself included) from *'reinventing the wheel'* by producing pinkeye scores. Such scores are only describing the extent or timeline of the bovine ocular inflammatory response. There is no need for them. They perpetuate the myth that pinkeye is a unique, incomparable ocular disease when it is much more likely that pinkeye is not even a single disease entity.

Apart from herd features of high morbidity and rapid spread, there is nothing unique about clinical presentation of IBK in an affected individual. We would be better served to use the ophthalmology approach used in all other species than inventing



Figure 1. Corneal changes are visible and indicate disease process, timeline and severity

new pinkeye scores. It is simple enough as characterisation of any animal eye lesion involves only **4 ophthalmic descriptors: conjunctiva, cornea, other ocular involvement and ocular pain**. Any conjunctival disease is described, such as hyperaemia, swelling, oedema, and amount and type of discharge. Any corneal disease classified as acute or chronic, ulcerative (along with size, depth, shape, and fluorescein staining properties of any ulcer and presence of descemetocoele) or non-ulcerative, plus any corneal neovascularisation and colour of cornea is described. Then any other ocular involvement e.g. *'aqueous flare'* indicating anterior uveitis, iridospasm, increased intraocular pressure, buphthalmia, and loss of eye. The fourth ophthalmic descriptor is pain; indicated by blepharospasm, photophobia, lacrimation and changes in behaviour.

Acute pinkeye is characterised by conjunctivitis with serous or mucopurulent ocular discharge and fluorescein-positive circumscribed oval-shaped acute corneal ulceration that tends to be centrally located with blepharospasm, photophobia, and epiphora. The most significant changes in pinkeye involve the clear cornea. Corneal ulcers are visible and made more so by fluorescein stain, UV lights and magnification. Ulcers are characterised as simple or complicated by persistence and depth. Corneal ulcer size is the most important parameter in any pinkeye assessment as simple corneal ulcers heal at constant rate related linearly to ulcer size. Corneal clarity and colour are important indicators of disease process and severity; infiltration by fluid, cells, blood vessels and scar tissue are visible as opacity and colour. The healthy cornea is transparent and avascular. Corneal opacities should be clearly characterised as their correct interpretation is a critical step in diagnosis.

The first questions to ask are:

- **What colour and form is the opacity?**
as these are limited and indicate cause, for example blue (whether focal or stippled like cobblestones) is oedema, greyish-white (sometimes feathery or wispy) opacity is fibrosis, yellow-green or tannish is inflammatory cell infiltration and red is blood (free or in blood vessels).
- **Corneal opacities are further characterised by asking: Where, why and when they occurred.**

The form of corneal blood vessels and their location are also clues to the nature of the disease process. If a corneal lesion is too large or contaminated to be healed by the epithelium directly, neovascularization of the cornea occurs to deliver inflammatory cells to this otherwise avascular organ. Shallow corneal injuries are vascularised from the sclera in the form of tree-like blood vessels that will grow from the sclera that is nearest to the injury. These vessels appear after a lag of 3 or 4 days and then grow into the cornea 1mm per day. Therefore, tree-like blood vessels 5mm long indicate that the injury happened 8 days ago. If these blood vessels are not tree-like but spikey and hedge-like they are coming from the ciliary vessels deeper in the eye and mean the corneal ulcer is not shallow, it is deep. So, tree-like vessels developing hedges on the tips of the branches means the ulcer is becoming deeper. If the ulcer is deep from the onset, hedging is seen as a rim of spikey blood vessels around the entire cornea.

Researchers state a small number of IBK cases may be associated with causes other than *M. bovis* (Levisohn, Garazi, Gerchman, & Brenner, 2004; O'Connor, 2007a). More than a small number of IBK cases in clinical practice are not *Moraxellosis*. There appears to be 'research pinkeye' that may be *Moraxellosis* and 'clinical pinkeye' that could be anything; mycoplasma, herpes, adenovirus, thelazia, trauma from ear tags or a poke in the eye, it could even be *Moraxella*.

In my studies I started by asking: If pinkeye is caused by *M. bovis*, carrier state, close contact, flies, dust, wind, UV, and other damage, **why don't we see it in local feedlots?** Local cattle have pinkeye, yet these same cattle do not have pinkeye in feedlots. January 2014 to December 2016 our clinic sold **3,423 tubes of cloxacillin** eye ointment (Orbenin® or Opticlox®). Only 128 tubes were sold to enterprises with feedlots. We sold 43 bottles of home-mixed subconjunctival pinkeye mix, none to feedlots.

One problem with pinkeye is that it is usually diagnosed in the first instance by producers with little or no veterinary knowledge. **Misdiagnosis is a real possibility** particularly as practitioners are often only consulted at end stage disease (Alexander, 2010).

Clearly not all producer-diagnosed pinkeye is infectious, and not all veterinary-attended IBK is *Moraxellosis*. It suits stakeholders to consider all eye disease of cattle pinkeye—a well-understood, self-limiting, catch all phrase warranting no potentially critical industry evaluation or difficult to implement changes to management. For 130 years most IBK research effort has been directed towards *M. bovis* but without a diagnosis there is no way of knowing if disease is being caused by this bug. Cattle, like other species, are subject to many different eye diseases. This is not what stakeholders want to hear; **that a diagnosis may be required before treatment and prevention is possible.** *M. bovis* may simply be a convenient scapegoat—one thing to focus on, a known enemy to treat with certain antibiotics or prevent with vaccination.

It is possible that not all pinkeye is infectious and not all IBK is caused by *Moraxella bovis*.

Using a clinical problem orientated medical approach to **pinkeye** we should ask:

1. Is the disease contagious?
2. Is it *Moraxellosis*?
3. What other factors could be contributing?

As well as more attention from veterinary ophthalmologists, pinkeye would benefit from interest by epidemiologists. The microbiologists have failed to improve outcomes on farms. If the only tool you have is a hammer... Microbiologists have blamed the inability to translate laboratory advances into clinical success on the bacteria. Experimental challenge studies struggle to cause clinical IBK and vaccines don't work because of the '**variable pathogenicity of *M. bovis***', the bacterium requires virulence factors; pili to attach to the cornea and haemolysin to cause damage, **OR** *Moraxella* is highly pleomorphic and many strains are not pathogenic **OR** *Moraxella* forms biofilms, **OR** *Moraxella* requires other organisms—*Moraxella bovoculi* or *M. ovis* or mycoplasma or herpes **OR** abiotic factors not available in the experimental setting, like wind, dust, pollen, flies, heat, rainfall, UV light, carrier

animal OR...(how about) **it is not a simple infectious bacterial disease.** The *Moraxella bovis* paradigm of IBK is stretched to the limits of plausibility.

As well as the mounting evidence that the epidemiology of pinkeye is not well-known, there is a body of publicly available evidence that **pinkeye vaccines have no protective effect** (Cullen, Engelken, Cooper, & O'Connor, 2017; Funk et al., 2009; O'Connor, Brace, Gould, Dewell, & Engelken, 2011; O'Connor et al., 2019). **You cannot vaccinate against a poke in the eye.** You may be able to control a disease without knowing proximate cause using epidemiology by decreasing risk factors.

M. bovis is the only organism that reliably produces typical IBK in eyes in challenge studies including in gnotobiotic calves (Chandler, Turfrey, Smith, & Gourlay, 1980) (Rogers, 1987), but experimentally produced IBK is often milder than natural IBK, and the relative contribution of *M. bovis* to IBK remains unclear. Apart from features associated with *M. bovis* bacterium itself, there could be various other requirements to produce severe IBK typical of field cases, including ocular injury, host immune response, environment and season, and concurrent pathogens. Disease studies cannot hope to account for all the variables in naturally occurring IBK. Experimental models may not resemble the real-world situation. Experimental reproduction of IBK with *M. bovis* have evolved to commonly include high doses of highly virulent strains with some form of corneal injury

mostly by burning the cornea with heat lamps or scarifying it with wire prior to inoculation.

IBK may not have a simple causal mechanism (O'Connor, 2007b) and the focus on *M. bovis* as a single causative organism in IBK, when most ocular disease in livestock is considered multi-aetiological in nature may explain the lack of progress in control of IBK. The conclusion *M. bovis* is the cause of IBK may only be an example of confirmation bias considering the huge research effort directed towards it. Like their early counterparts, current day IBK researchers are once again questioning if *M. bovis* is a true necessary component of all cases of IBK (O'Connor, 2007b) or a secondary opportunist (Prieto et al., 2013), whether IBK is a multifactorial disease (J N Cullen, A Lithio, A S Seetharam, Y Zheng, G Li, et al., 2017; Schnee, Heller, Schubert, & Sachse, 2015) caused by abiotic and biotic factors alone or in combination (Zheng et al., 2019). Some have even raised the unlikely possibility that although frequently recovered from IBK lesions *M. bovis* may be incidental and not causative (O'Connor et al., 2011). A fresh approach to IBK is warranted.

Molecular based tests, such as PCR, and 16S rRNA microbial sequencing analysis which simultaneously detect multiple microorganisms and even describe the entire ocular microbiota are increasingly available. Even these exquisitely sensitive tests have, thus far, failed to clarify the aetiopathogenesis of IBK (J N Cullen, A Lithio, a S Seetharam, Y Zheng, D Nettledon, et al., 2017). A study on naturally occurring IBK in beef calves utilizing PCR could not show a temporal association between exposure to *M. bovis* and *M. bovoculi* and subsequent IBK (O'Connor, Shen, Wang, & Opriessnig, 2012). A PCR study in IBK-affected cattle in Germany found *M. ovis* to be the most prevalent *Moraxella* species. The prevalence of *Moraxella* species could not be readily correlated with clinical IBK but high prevalence of another bacteria, *Mycoplasma bovoculi* (NOT *Moraxella bovoculi*) in a herd meant they were more prone to acute IBK. The authors concluded that synergistic interaction between *Myc. bovoculi* and *Moraxella* species may predispose to IBK (Schnee et al., 2015).

Some **other gaps and incongruities** in the *M.bovis* pinkeye story include:

The most common treatment for pinkeye in Australia is 'Pinkeye ointments' containing benzathine **cloxacillin**. *M. bovis* is Gram-negative and a penicillin derivative would not be a first-choice antibiotic. Originally marketed for mastitis as an intramammary infusion, cloxacillin appears to have become the pinkeye drug of choice by accident or trial-and-error by farmers who, when desperate, will try anything. Whilst active against some Gram-



Figure 2. IBK is typically characterised as a centrally located melting ulcer, but *M. bovis* cannot itself melt a cornea, as it does not produce coagulase



Figure 3. One reason to not treat IBK is that disease is 'self-limiting', so is a broken leg it just may not heal well

negatives, cloxacillin has a narrower spectrum of activity than natural penicillins and is inactive against *Mycoplasma*, *Rickettsia* and *mycobacteria* (Plumb, 2008). Australian strains of *M. bovis* are thought to be sensitive to cloxacillin (McConnel, Shum, & House, 2007), but American researchers found ***M. bovis* was so resistant to cloxacillin** (Webber, Fales, & Selby, 1982a) that they made a semiselective media for culturing *M. bovis* by adding it (Webber, Fales, & Selby, 1982b). Cloxacillin killed off competing bacteria but not *M. bovis*.

If *M. bovis* can cause direct damage to the cornea it may well be the only bacterial primary pathogen of any mammalian cornea (Maggs, 2008). Clinical IBK can range from mild conjunctivitis to severe progressive corneal ulceration known as a melting ulcer. One frustration of the *M. bovis* IBK paradigm is that the cause of extensive corneal damage observed in clinical IBK remains obscure. ***M. bovis* does not produce any coagulase** (Frank and Gerber, 1981).

Flies, and other insects, have been suspected; of being the direct cause of pinkeye themselves; of being a component cause (or risk factor) for IBK by causing ocular damage that facilitates the entry of *M. bovis*; of mechanically spreading *M. bovis*; or of being true vectors, harbouring the bacteria, for the transmission of *M. bovis* in a herd (Arrends, Wright, Barto, & Lusby, 1984; Berkebile, Hall, & Webber, 1981; Glass & Gerhardt, 1984; Hall, 1984). **While the association of flies and IBK** is commonly presumed, the relationship is not fully understood. Cases of IBK consistently increased with numbers of face flies (Cheng, 1967), but even in the absence of pathogenic organisms feeding activity of female face flies was shown to cause mild bovine keratoconjunctivitis by injury (Shugart *et al.*, 1979) and nearly all studies on pinkeye and flies have been done on the **face**

fly (*Musca autumnalis*) which does not occur in Australia.

Many do not treat pinkeye, because it is 'self-limiting'. So is a broken leg if you wait long enough!

I want to fix pinkeye!

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