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vet 360

Vol 05 | Issue 03 | July 2018

Feline CKD
Therapeutic Goals

ABCs of Veterinary Dentistry
"N" is for No

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Gastrointestinal Therapy
Anti-emetics and Antacids

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Editor's Note

Nothing is constant except change. The CPD article on antimetics and antiulcerogenics highlights some new developments and efficacy data now available for the drugs we commonly use. Things have changed, and we need to make sure we aware of these changes as it does affect how we use these medication in clinical practice. A medication is only effective if used at the correct dose and frequency for the appropriate condition and administered correctly (route and timing with food).

Changing norms of treatment is also highlighted in the article on CKD treatment in cats where emphasis is shifting from poly-pharmacy to only using medications with proven efficacy – especially in this breed which is difficult to dose and may develop nausea and anorexia. The owner pet bond and QOL of the patient is emphasised.

The article on KCS is a gentle reminder that this condition occurs frequently and we as veterinarians should be more pro-active in diagnosing and treating it - especially earlier cases. KCS can also sometimes be a early clinical sign of an underlying metabolic, immune mediated or neurogenic condition - so make sure you evaluate the patient for possible causes before just treating symptomatically.

I hope you enjoy the edition. Please feel free to send requests or comments to me email address.

Liesel

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Madaleen Schultheiss

A Brief Manifesto for Happy Team Relations

By Dr Dave Nicol
BVMS Cert Mgmt MRCVS

I like questions. And good questions deserve answers, plus they often lead to fascinating conversations. So, when a member of a Facebook group I'm active in posted a great question, I was ready to get stuck in.

Here's what she asked

"Question for practice owners, managers, directors: what do you want from vets? What makes you want to keep a vet? I expect high turnover is a big one...? I am curious about how we can be better vets so that we increase our value to the practice?"

And here's what happened next... [Profanity warning].

I want you to give a major F*@K about the following:

- "us" - the team.
- Your clients - if you can do clients then you can do vet med. If you can't then you are in trouble.
- Your patients (obvs).
- You.
- Really, YOU in quite a big way.
- Money, and don't be embarrassed or ashamed about it. It'll never come first, but it is there and we all need to work on this.
- I want you to be willing to learn and grow.
- I want you to help others learn and grow.
- I want you to be willing to be amazing.
- I want you to leave your ego at the door.
- I want to laugh and cry with you at the shit we have to deal with.
- I want to be able to have a beer with you.
- I want you to understand that I care very deeply about helping you get where you want to go and helping you become what you can be.
- I want you to accept that you are not perfect, that none of us is and that's OK. Failure is OK.
- I want you to be happy.

If you can do that, then you'd be a lot better than I was when I started out, and I'd be very, very proud to call you a teammate. (And if you can be that, or be willing to work towards it.... could you also never leave?)

Now let me ask you back.... what do YOU want/need me to be as a leader? Because I think that probably matters more..."

The vet who asked the question then responded with this:

David, for me, off the top of my head it is similar to many of the things you said you want from a vet.

- Plus either be a good role model or be there to support and facilitate us vets to achieve the desired outcome.

- To understand what it is really like to do the job and care about whether or not I have had my lunch today.

- Have good systems in place to make everything run smoothly and efficiently and listen when we give feedback.

- I want you to care that I get away on time and that I have had to deal with a really obnoxious client and notice that I dealt with it well.

- Call me needy, but I do like acknowledgement when I work hard. I want you to empathise when sh*t happens and take time to understand the whole story before you criticise.

- I think it would be great if somehow bosses and/or colleagues could positively feedback regularly

on what we appreciate, value and admire in one another.

- Sometimes I think it's lack of communication, but generally, I want you to show that I am valued every so often.
- I want you to provide an environment and facilities conducive to practising good medicine.
- I also want to be inspired by you and be able to respect you.
- And I want you to totally trust me and have my back if something goes wrong.

Just a few things off the top of my head...
And when we were done, it felt like maybe, unwittingly, we just had a really honest conversation that helped to clarify what we have to do as team members and leaders to improve relations and be happy working together. So, my nameless veterinary colleague (you know who you are), anytime you need a job, look me up. ;-)

Until next time...

THE VETERINARY ACCELERATOR PROGRAM

Dr Dave Nicol

UK Based Graduate Support Community Extends And Opens to Global Membership

Following the successful launch of the VetX Graduate Community pilot scheme in the UK, the VetX Academy are pleased to announce the program has been opened now for global enrollment. Aimed at veterinary surgeons in the first five years of their career, VetX offers monthly veterinary non-clinical skills training, online mentoring, and a peer support network of young vets from around the world. The major benefit is that community members learn the foundation life skills upon which a successful career is built.

VetX Graduate founder and So You're A Vet...Now What? book author, Dr Dave Nicol said, "Since September last year we've welcomed more than 50 vets to the community. Since then we've successfully delivered training and support; improving member outcomes and helping them to engage with their work and practices in a healthier way. Adding, "The feedback from members has been very incredibly positive. Training, mentoring and support is what young vets crave, so it's heartening to see them respond so well."

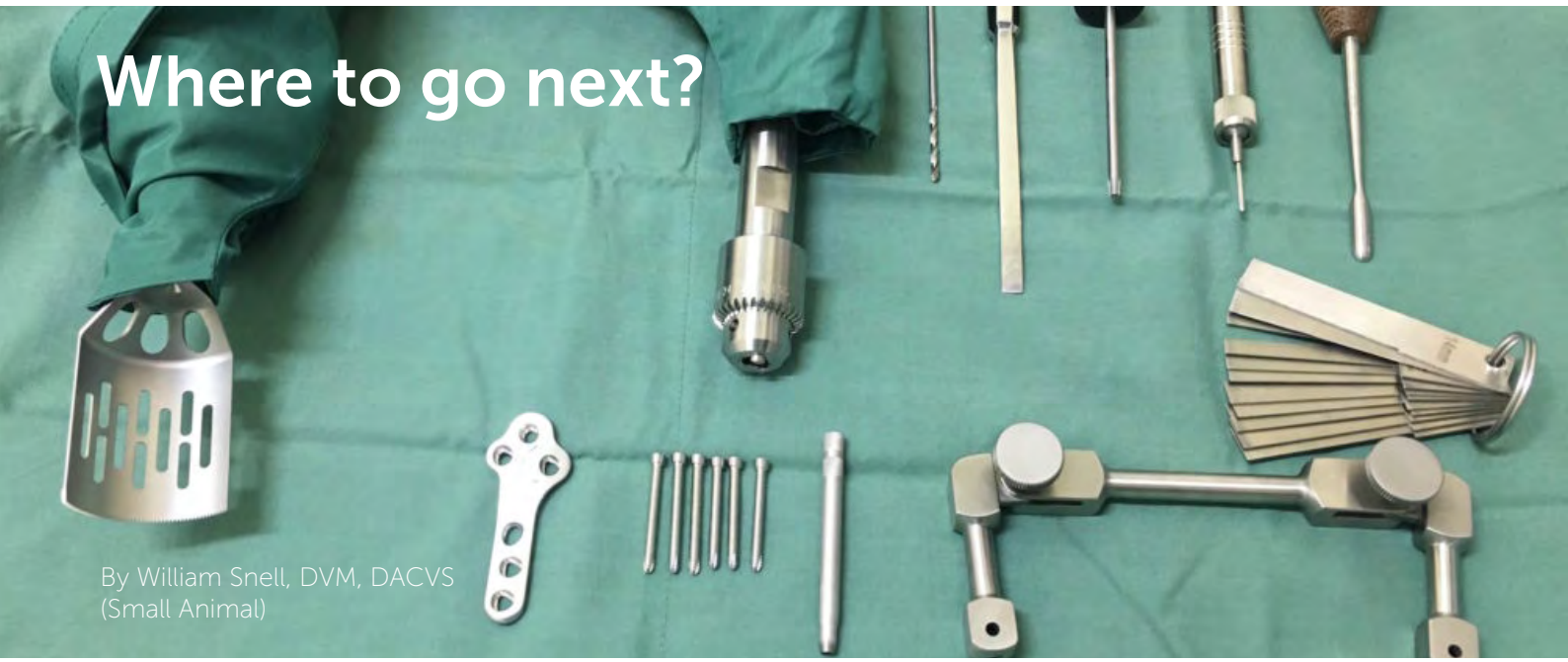
Since launching in September 2017, the VetX team have been working hard to improve both quality and value based on feedback from members and practices. To that end, there are now double the number of training hours available, including a new "clinical rockstars" stream. Dr Nicol has also recruited a community manager and added two new, world-class mentors in the US and Australia to the team, allowing improved support within the community and truly global mentor access at convenient times wherever community members are on the planet.

Even the technology has been overhauled with the successful migration to a superior teaching platform that improves interaction, membership management and tracks progress so clinics and vets know how well they are getting on. The VetX Graduate program is truly unique. Each day I see a passionate community coming together to learn, support each other and grow. It's a special thing and it's getting better each week as new people join and more training becomes available."

VetX Graduate is open to all and is entirely complementary to in-house training or other clinical CPD training. But clearly offers smaller, independent practices the chance to compete in the recruitment market with larger corporate groups who offer in-house mentoring schemes in a very cost-effective way.

A 12-month membership is priced at only £365/year or £35/month paid over 12 months.
Practices and individual vets interested in learning more about the VetX Graduate Community should visit http://www.drdavenicol.com/vetx-grad

Cranial Cruciate Ligament Injuries in Dogs



Where to go next?

By William Snell, DVM, DACVS (Small Animal)

Canine cranial cruciate ligament (CCL) disease is the most common cause of hindlimb lameness seen in our canine patient population.[1] Rupture of the ligament may occur as a traumatic avulsion, an acute tear from excessive strain or more commonly as a progressive degenerative disease resulting in partial or complete ligament rupture. While any dog breed can suffer from the condition, there are certain breeds that appear to be predisposed. Patients may present at any age, and both neutered and female patients may be over-represented.[2, 3] Timing of neuter has not been shown to correlate with this disease, and the disease is thought to be multifactorial. We understand certain genetic, developmental and environmental factors play a role in the degenerative process, including limb anatomic conformation, tibial plateau angle, body condition and activity type, but all factors have not yet been identified. Since the degenerative form of the disease is thought to be progressive in nature, patients may present at varying ages and stages of ligament integrity. It has also been stated that 30% to 40% of patients who tear one cruciate ligament are predisposed to tearing the contralateral limb within one or two years.[4]

The cranial cruciate ligament is important to neutralizing cranial tibial subluxation, internal rotation and hyperextension of the stifle. The ligament itself consists of two bands: craniomedial and caudolateral.

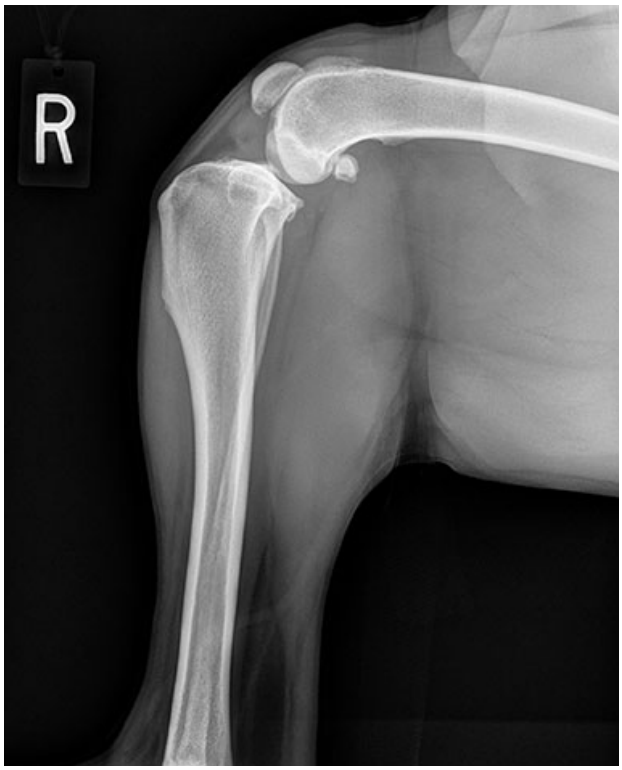


Figure 1: Right Stifle, lateral view. The infrapatellar fat pad is reduced as a result of joint effusion and or capsular thickening. Mild osteophytes are seen on the distal femur, patella and tibial condyles. All images courtesy of Dr. William Snell.

The craniomedial band is taut in both flexion and extension, while the caudolateral band is only taut in extension. For this reason, the craniomedial band is often the first portion of the ligament to fail.[1] These patients are considered to have a partial tear, and medical management of these cases has shown variable success depending on the techniques used.

Diagnosing a CCL tear

High suspicion of partial or complete CCL tear is typically identified on physical examination coupled with two-view stifle radiographs. The most common physical exam findings include stifle joint effusion, medial buttress (with chronicity) along with cranial drawer sign and tibial thrust. Cranial drawer sign may only be present on flexion of the limb in patients with a partial CCL tear. These patients also tend to be painful on hyperextension of the stifle. Radiographs will typically confirm evidence of joint effusion and may show signs of osteoarthritis in the joint. Some patients may also show the tibia cranially displaced (in drawer) when compared with the femur (Figure 1).
Minimally invasive procedures including standard stifle arthroscopy or, more recently, arthroscopy via needle scope may also be used as a diagnostic tool to confirm the diagnosis of a partial or complete CCL tear Magnetic resonance imaging (MRI) is an uncommon diagnostic tool used for identification of CCL tears or meniscal disease in veterinary medicine at this time.

Non-surgical management

Medical management options for partial CCL tears may consist of varying combinations of rest, nonsteroidal anti-inflammatory drugs (NSAIDs), nutraceutical use and physical therapy. But more recently platelet-rich plasma (PRP) and/or canine stem cells derived from bone or fat have been used with varying and, unfortunately, inconsistent success.[5] There's also an argument that early surgical intervention in patients with partial tears may help in preserving the remainder of the intact ligament and that these patients may have a better postoperative success.

Surgical management options

Surgical options for these patients are broken down into two general categories: ligament replacement versus biomechanical techniques. More recent literature has suggested that using a combination of a ligament replacement and biomechanical techniques may result in a more stable stifle joint postoperatively.[6] Ligament replacement techniques can be further subdivided into intra-articular or extra-articular techniques.

Regardless of stabilisation procedure, it's routine to first perform a joint exploration. This may be achieved by an open arthrotomy or arthroscopically. All intra-articular structures should be evaluated, with special attention paid to the articular surfaces of the femur, tibia and patella as well as the long digital extensor tendon, both the cranial and caudal cruciate ligaments and the medial and lateral menisci. Meniscal tears should be debrided if present. There's also continued debate over the procedure known as a "meniscal release." This technique involves transection of the caudal attachments of the medial meniscus with the goal of reducing the risk of subsequent meniscal tearing. However, it has been shown to result an increased contact area of the medial joint compartment, contributing to accelerated development of osteoarthritis in the stifle joint.[7]

Intra-articular stabilisation techniques are the mainstay of human anterior cruciate ligament (ACL) repairs. These techniques employ harvesting a biologic graft from another tendon in the body or from a cadaver and then drilling bone tunnels and replacing the torn ACL. The graft will then go through a period of devitalisation, followed by revascularisation and ligamentisation over an approximately 20-week period. During this time, the graft is at a diminished tensile strength, and overuse may lead to graft failure and returned stifle instability. More recent research has focused on synthetic intra-articular replacement or biologic scaffolds as opposed to the biological replacement techniques that have fallen out of favour in the veterinary surgical field. Today, however, most surgeons are finding themselves electing extra-articular or biomechanical stabilisation techniques.

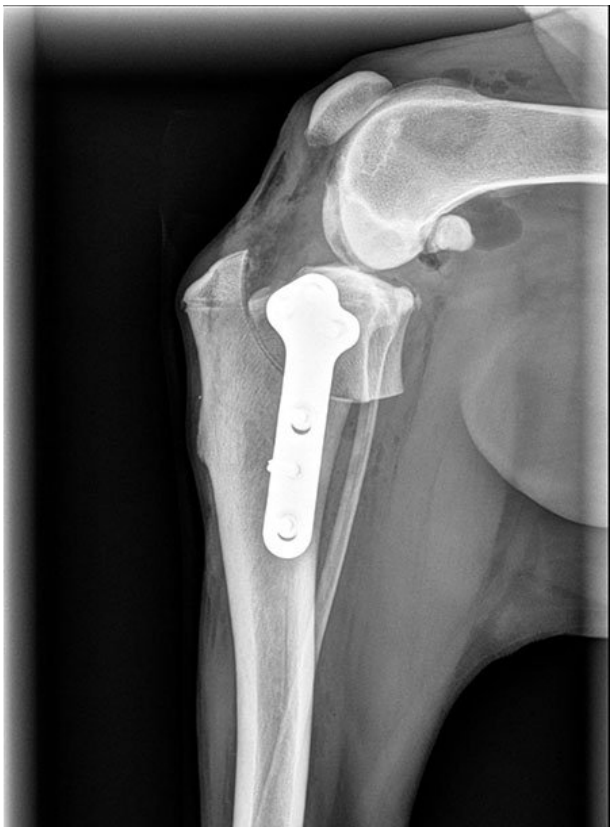


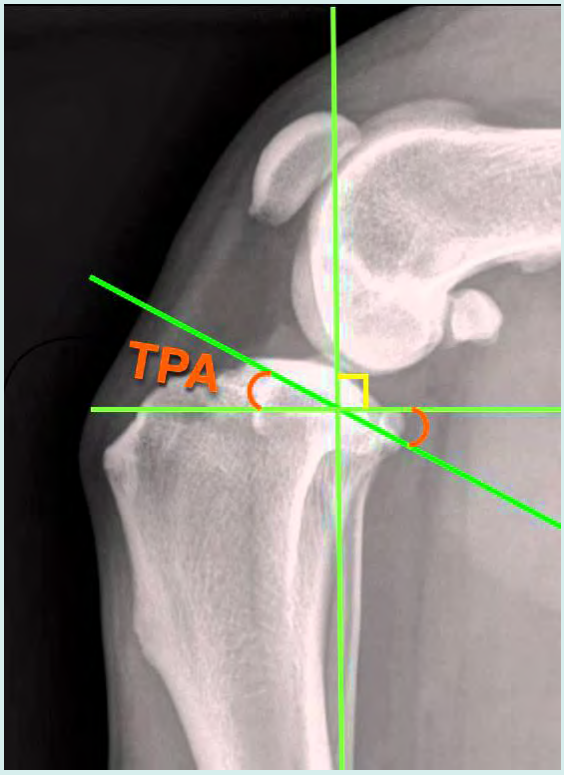
Figure 2: Left Stifle, lateral view. Immediate postoperative TPLO.

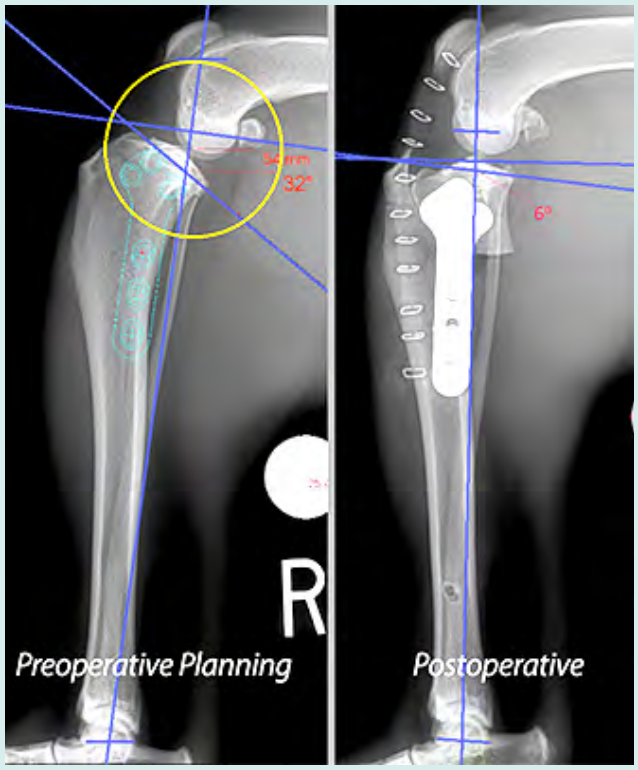
Extra-articular repair techniques utilise synthetic materials to traverse and stabilise the stifle joint. The most commonly performed techniques include the lateral fabellar suture, bone anchor techniques and the Tightrope technique. In all cases, material is anchored at relative isometric points outside the joint on the distal femur and proximal tibia. Given that there are no true isometric points in the stifle due to the cam-shaped femoral condyles, the synthetic material is subjected to cyclic loading that eventually results in implant fatigue and failure. The goal of these repair options is to stabilise the stifle long enough to allow for periarticular fibrotic tissue to develop and mature over a 16-week period. This periarticular fibrosis becomes the long-term stabiliser of the stifle joint. The main complications of these techniques include implant infection along with premature implant failure.[8]

Biomechanical techniques, also known as osteotomy procedures, involve cutting and manipulating the tibia in various ways that result in biomechanical neutralis ation of cranial tibial thrust in the CCL-deficient stifle. The most common procedures used today are the tibial plateau leveling osteotomy (TPLO), the tibial tuberosity advancement (TTA) and, more recently, the center of rotation angulation (CORA)-based leveling osteotomy (CBLO). Of these, the TPLO is probably the most widely used osteotomy technique.

With the TPLO procedure (Figure 2) the 90-degree flexed lateral radiograph of the tibia (including stifle and hock) allows for measurement of the tibial mechanical axis, and a bisecting joint line of the stifle allows for measurement of the needed tibial plateau angle (TPA). Average TPAs in dogs range from 25 to 30 degrees (see text box).[9] Once the patient's TPA is measured, a dome osteotomy of the proximal tibia is performed and the proximal tibial fragment is rotated a predetermined distance based on the patient's specific TPA. This results in a reduction of the tibia slope to approximately five degrees, thus neutralising cranial tibial thrust. A bone plate with screws is then used to bridge and stabilise the fracture on the medial tibia. The patient is then limited in activity over the next six to eight weeks while the osteotomy heals.

The TTA (Figure 3) is a technique that involves a linear cut in the tibial tuberosity and advancement (cranial displacement) of the tibial tuberosity fragment in a cranial direction to achieve and maintain a patellar tendon angle of approximately 90 degrees with respect





Editor: There are specific radiographic positioning techniques to obtain the image and measurements are made from very specific points on the radiographs. In essence the vertical line is drawn connecting the tibial bony attachment of the cruciate down to the middle of the tibio-talar joint. The horizontal line is drawn perpendicular to the vertical line. The tibial plateau angle (TPA) is determined by drawing a line through the intersection of these two lines which runs parallel to the tibial plateau. The angle thus created is the TPA.

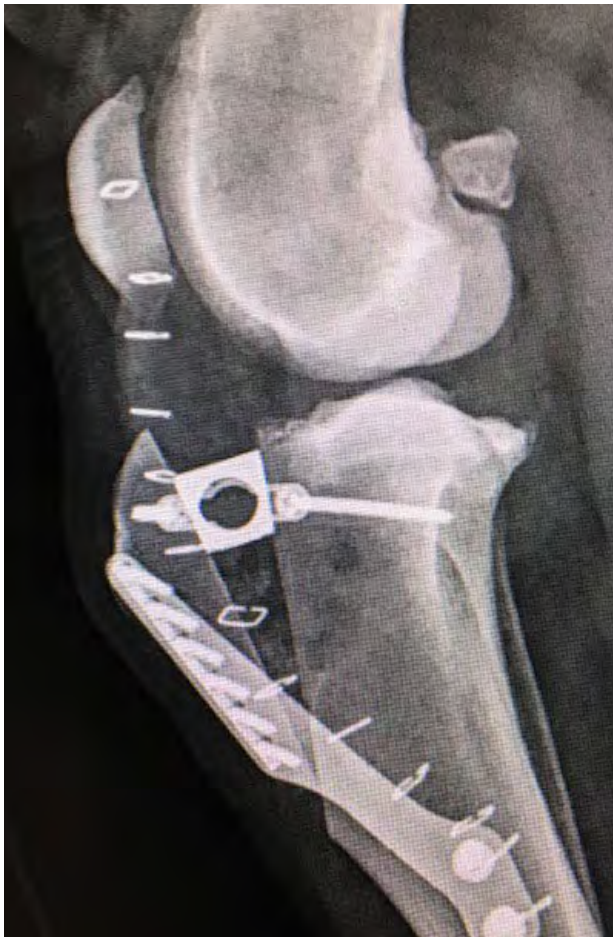


Figure 3: Left Stifle, lateral view. Immediate postoperative TTA.

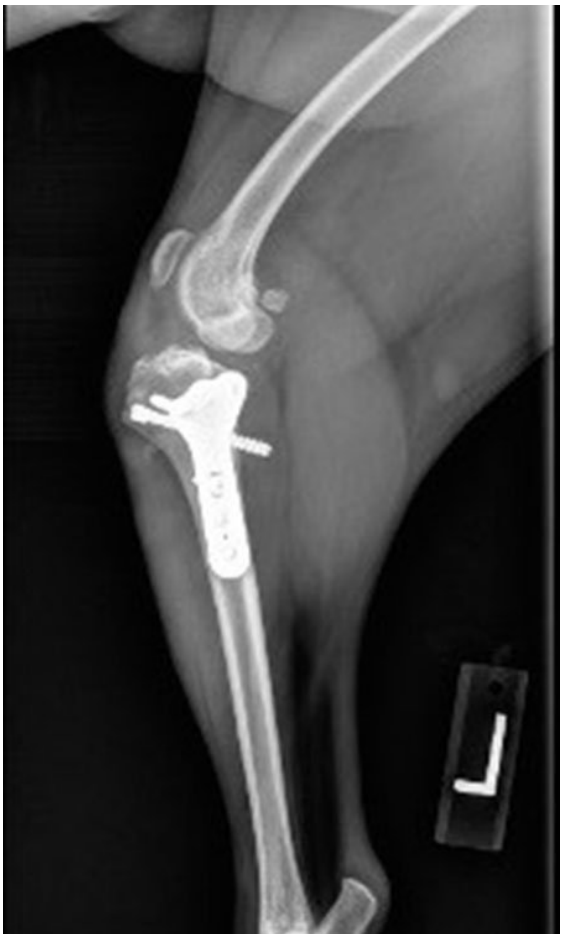


Figure 4: Left Stifle, lateral view. Six weeks post-CBLO.

to the premeasured tibiofemoral contact point when the limb is in near full extension. This will in turn result in neutralisation of the tibiofemoral shear force in a CCL-deficient stifle joint. The implant system consists of a cage and a bone plate that acts as a tension band. The cage is inserted within the proximal portion of the osteotomy and is size-specific to the intended cranial advancement of the tibial tuberosity fragment. The plate is then secured proximally to the medial aspect of the advanced tibial tuberosity fragment and distally to the tibial diaphysis acting as a tension band. The patient is then limited in activity over the next six to eight weeks while the osteotomy gap fills in and heals. Case selection is important with this technique, as specific tibial tuberosity conformations and excessive TPAs (> 30 degrees) may result in less favorable outcomes.

The CBLO (Figure 4) procedure is the most recently described procedure and is similar to the TPLO in that it is a dome osteotomy technique that attempts to level the TPA. The location of the osteotomy is based on the CORA of the tibia and attempts to place the axis of correction (ACA) in line with the CORA to limit tibial translation, thus limiting a caudal shift in joint forces that have been described with the TPLO procedure. An additional advantage over the TPLO includes the fact that it can be performed in juvenile patients, as the osteotomy and plate placement do not affect the proximal tibial physis and tibial tuberosity apophysis. Further advantages include an increase in fracture contact area for more load sharing, increased proximal fragment bone stock for additional implant placement and ease of adding on ancillary stabilising procedures.

While each osteotomy procedure may have its own subset of complications specific to each individual technique, all procedures pose similar risks and complications of implant infection and/or failure. At the end of the day the procedure or procedures that are recommended are often determined based on the age, size, body condition of the patient, concomitant morbidities, the comfort level of the surgeon with the described techniques, the expectation of the owner and the pet owner's ability to financially support the recommended procedure.

References available online

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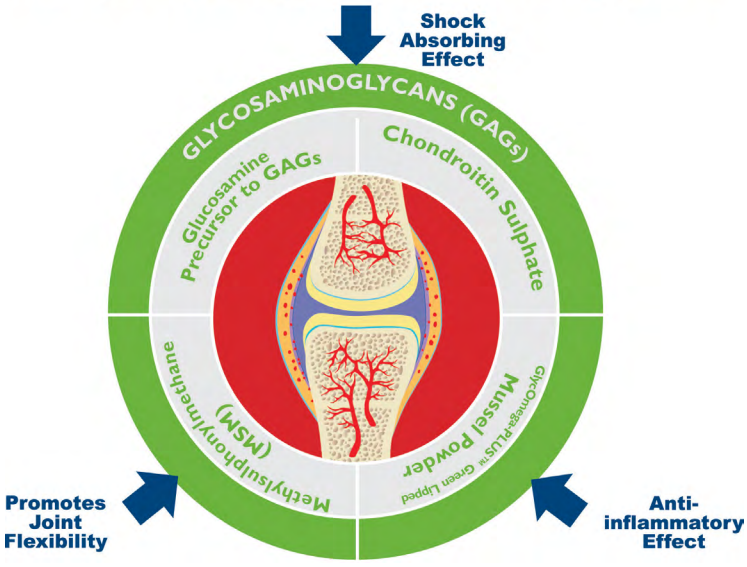

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Just Ask the Expert
Light Anaesthesia Anxiety

By Andrew Claude, DVM, DACVAA

Are your surgical patients all sufficiently anesthetised? There are lessons for all veterinary practices in deciphering the cause of inadequate anesthetic depth in routine procedures. Here is a step-wise guide on what to consider as you perform your root-cause analysis.

Q. Recently, we have noticed that our anesthetized patients have heart rates that don't correspond with their respiration rates in terms of their plane of anaesthesia. Their heart rates seem to reflect stage 3, plane 2 anaesthesia, but their respiration rates reflect stage 3, plane 1 anaesthesia (e.g. a 100-lb dog with a heart rate of 100 to 110 beats/minute but respiration is 60 breaths/minute or more). These patients are easily awakened by stimuli such as turning them over or stimulating a sore tooth when performing a dental cleaning. We find we are constantly having to turn up the isoflurane. We originally thought we had a problem with our medical gas scavenging system. However, we recently performed a test to determine if the scavenging system has the correct amount of suction, and the results were normal. We are back to square one. I would greatly appreciate any thoughts you have on this.

A. This is a very interesting question with two major categories to consider—the circumstances regarding the patient and those of the scavenger system.

Based on the context of the question above it seems you have placed most of the concerns for the patient issues on the scavenging system. Granted, there are key points about the scavenging system to consider, but there are also important details concerning the patient that are of equal importance.

1. What are the typical anaesthetic protocols being used for these patients?

Commonly, premedication protocols use the combination of a tranquilizer and an opioid (neuroleptanalgesia). **Multiple publications state that analgesics used before and during surgery decrease the dose of both the induction drugs and the maintenance inhalants** (mean alveolar concentration, or MAC). Local and regional anesthetic techniques are also effective in decreasing the MAC of inhalant anesthetics. Without preoperative and intraoperative analgesia, anesthetised patients will be subject to sudden light planes of anaesthesia necessitating multiple top-up administration of induction drugs and increased doses of inhalant anaesthesia.

In my opinion, in dogs and cats, if you are routinely running your isoflurane vaporizer greater than 1.75% (sevoflurane > 2.5%) during surgical procedures (e.g. ovariohysterectomy, orthopaedics), your analgesia protocols should be reconsidered. Contrary to

popular belief, butorphanol is NOT an effective primary analgesic (especially in dogs) and does not last very long. Morphine, hydromorphone, methadone, fentanyl, oxymorphone and other full mu agonist opioids are much more effective analgesics.

2. What is the time-frame between premedication, induction and maintenance anaesthesia for surgery?

Is there enough time allotted for the premedications to work before induction and surgery? For example, buprenorphine, although an effective partial mu agonist, takes 20 to 30 minutes for full analgesic effects. Starting surgery before analgesic onset will result in elevated levels of nociception, contributing to patient arousal.

3. How are patients being monitored?

In response to an increase in sympathetic tone, heart rate will increase during light periods of anaesthesia; however, judging anaesthetic depth solely based on heart rate is inaccurate. Because heart rate can be affected by so many other variables, it is a poor indication of anaesthetic depth. For example, a patient entering a deeper plane of anaesthesia (level 3 to 4) will commonly become tachycardic because of increased sympathetic tone from decreased ventilation and elevated partial pressure of carbon dioxide (pCO2). The person monitoring the patient notices the tachycardia and mistakenly turns up the inhalant anesthetic, thinking the patient is arousing. Gradually the patient will reach deeper levels of anaesthesia, which can become serious. Besides heart rate, other monitoring parameters include arterial blood pressures, end-tidal pCO2, jaw tone, eye position, response to a toe pinch, respiratory rate and character, mucous membrane color and capillary refill time.

Other than tachypnea, no other parameters included in the question help judge the patient's level of anaesthesia. However, a 100-lb dog with a heart of 100 to 110 beats/minute is inconsistent with a surgical plane of anaesthesia (keeping in mind heart rate is a poor indication of anaesthetic depth). Any external stimuli—nociception, position changes—will cause the patient to quickly arouse. It is important to realise that anaesthesia is not the same as analgesia. Many anaesthetic drugs (e.g. isoflurane, sevoflurane, propofol) have little to no analgesic properties. In addition to monitoring a patient's cardiovascular status, respiratory status and depth of anaesthesia, it is imperative to monitor the level of the patient's nociception. Movement during anaesthesia could be an indication of inadequate anaesthesia (light plane) or reflexes (nociception). When used alone, a high dose of inhalant anesthetic drug is required in order to eliminate all muscle movement reflexes due to surgical stimuli during general anaesthesia (this is called "MAC no movement"). Administering analgesic drugs before and during surgery (intermittent or continuous rate infusions) decreases the necessity for high vaporizer outputs.

4. What breathing systems are being used? Are the breathing systems being checked for leaks? Are the patients intubated?

There are two major types of breathing systems used on conventional anaesthetic machines for small animal patients: rebreathing and non-rebreathing. The rebreathing systems can be further divided into adult and pediatric circle systems. The choice of breathing system is based on patient size: patients weighing < 5 kg require a non-rebreathing system, those between 5 and 10 kg need a pediatric rebreathing system, and those > 10 kg require an adult rebreathing system.

Because oxygen flow rates and carbon dioxide removal differ between non-rebreathing and rebreathing systems, an important question needs to be clarified. Has the author noticed the difference only in large dogs (adult rebreathing) or small patients (non-rebreathing), or both? For example, a patient on a non-rebreathing system with an inadequate flow rate will rebreathe carbon dioxide (due to dead space ventilation within the expiratory tube), which, in turn, will cause the patient to initially become tachypnoeic and tachycardic.

Are the patient breathing systems (both rebreathing and non-rebreathing) checked for leaks? Leaks within the breathing system can cause the anaesthetic gases (oxygen, carbon dioxide and anaesthetic vapor) to leak out, resulting in an inadequate supply to the patient, or room air to enter in, resulting in dilution.

Are the patients intubated or are face masks used? Because face masks frequently do not provide a tight seal against the patient's snout, anaesthetic gases can leak out and room air can leak into the breathing system. Intubating the patient allows for controlled ventilation, which is helpful in controlling the depth of anaesthesia. Combining the use of a mask for inhalant anaesthesia and an active scavenger system would almost confirm air being pulled into the breathing system through the mask. This would result in a significant amount of anaesthetic gas loss and/or dilution.

5. What kind of ventilation is being used?

Patients allowed to spontaneously ventilate will normally have cyclical anaesthetic depth levels. A lightly anaesthetised patient will have a higher minute ventilation, thus breathing more anaesthetic gases into the lungs. Gradually the patient will breathe themselves into a deeper plane of anaesthesia, resulting in a decreased minute ventilation and decreased uptake of anaesthetic vapor. Eventually the patient's anaesthetic

depth will begin to lighten and minute ventilation will increase again, completing the cycle.

It's recommended to use the rebreathing bag to periodically apply a positive pressure breath (intermittent sighing) to the patient at least once every five minutes. This will help decrease positional pulmonary atelectasis and improve the cyclical levels of anaesthesia in spontaneously ventilating patients. Controlled ventilation, employing a mechanical ventilator with intermittent positive pressure ventilation (controlled IPPV), provides the most consistent control level of anaesthesia because the patient is not relying on its own minute ventilation to determine anesthetic vapor uptake.

Tachypnoeic anesthetised patients can develop ventilatory dead space, resulting in little to no anaesthetic gas exchange in the alveoli. Positive pressure ventilation (intermittent or controlled) will help mitigate tachypnoea and dead space ventilation. Some people use the oxygen flush valve to provide positive pressure ventilation to their anaesthetised patients. Because the oxygen flush valve bypasses the flow meter and vaporizer, when pressed, 50 to 80 psi will be delivered to the patient's alveoli, causing barotrauma. In addition, using the oxygen flush valve as a mode of ventilation will cause the patient to gradually become light due to anaesthetic vapor dilution.

6. Can you describe your active scavenging unit?

Is the active scavenging system made by a credible manufacturer? Active scavenging systems are manufactured to provide the correct draw against the patient breathing system and should include ample safety mechanisms. The interface between the scavenging system and breathing system contains its own reservoir bag, a pressure control needle valve, and a positive and negative pressure safety valve. If the negative pressure safety valve malfunctions, there will be an excessive draw against the patient breathing system, resulting in anesthetic gases being pulled away from the patient. This problem is typically manifested by a continually deflated breathing system reservoir bag and variable levels of patient ana esthetic depths. Ways to mitigate excess scavenging system draw against the patient breathing system are to turn up the flow rate to match the draw, adjust (close) the interface needle valve, or partially close the adjustable pressure limiting device (pop-off valve).

Scavenging systems problems are rare, but they can occur. I would recommend you continue to use the passive system until the problem is solved.

Onset and Peak Clinical Effects of Premedication Agents

Drug	Onset of action	Peak effect	Comments
Acetylpromazine	15 minutes	45 minutes	Tranquilisation, not sedation – wait until peak effect before induction. No analgesic properties.
Morphine	10 minutes	45 minutes	Clinical effects relevant within 10 minutes.
Butorphanol	15 minutes	45 minutes	Provides good sedation although minimal analgesia.
Buprenorphine	20 minutes	45 minutes	Analgesia to the same level as NSAID's although bind receptors with higher affinity than morphine.
Diazepam	30 seconds	3 minutes	Functions better as a co-induction agent, although not consistently induction agent sparing. No analgesic properties. Strictly IV – propylene glycol formulation poorly absorbed via other routes.
Medetomidine	5 minutes	20 minutes	Excellent sedative and analgesic, although effects last approximately an hour.

These parameters can be applied to intramuscular administration in healthy, adult animals. Effects may be prolonged in cardiovascularly unstable animals or those with hepatic or renal dysfunction.

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Keratoconjunctivitis Sicca (KCS): Common and Underdiagnosed

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Tears are needed to lubricate the cornea, and remove debris and pathogens which may cause harm. Additionally tears function to nourish with oxygen, glucose, electrolytes, cleanse, lubricate, and maintain corneal clarity and protect with immunoglobulins, enzymes, anti-bacterial, growth factors and fibronectin. Changes in any of the above may lead to discomfort, infection, poor wound healing, visual abnormalities, and adjacent tissue dysfunction of the conjunctiva and eyelids.

Normal tear film distribution relies on normal anatomy, normal tear film quality, and normal tear film quantity. The tear film is composed of lipid, mucin and water. A deficiency in the aqueous layer is referred to as quantitative KCS, whereas a lipid or mucin deficiency is termed qualitative KCS. Any condition which limits the production of any of these components has the ability to result in "dry eye".

Traditionally, dry eye was always considered a deficiency of the aqueous layer of the tear film, however more recently the definition of dry eye disease has been updated by the Dry Eye Workshop II (2017) and is described as,

" ... a multifactorial disease of the ocular surface characterised by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyper-osmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles."

Sounds complicated, right? Well it is!

The most common clinical signs seen with KCS include: Mucoid discharge, hyperaemia, neovascularisation, blepharospasm, epiphora, and a dull lacklustre cornea

Eyes affected with KCS are generally red and painful, and the condition can be uni- or bilateral. Concurrent corneal ulcers and conjunctivitis may be present. Chronic, untreated KCS may can result in scarring, neovascularisation and pigmentation and, if severe, decreased vision.

Causes of KCS are numerous but can be classified aetiologically:

- Congenital
- Senile atrophy
- Immune-mediated
  - Local
  - Systemic e.g. Sjögren's syndrome, systemic lupus erythematosus, pemphigus foliaceus, rheumatoid arthritis, polymyositis and polyarthritis, atopy, glomerulonephritis, and ulcerative colitis
- Infectious
  - Canine Distemper Virus
  - Viral or bacterial conjunctivitis
  - Feline Herpes Virus
- Endocrine Disease
  - Diabetes Mellitus
  - Cushings Disease
  - Hypothyroidism
- Irradiation
- Neurogenic
- Iatrogenic
  - removal of the gland of the third eyelid
  - uncorrected prolapsed gland of the third eyelid ("cherry eye")
  - Facial nerve damage e.g. after total ear canal ablation surgery
- Breed-related
  - American cocker spaniel
  - Bloodhound
  - Boston terrier

- Cavalier King Charles Spaniel
- Chihuahua
- English bulldog
- English Cocker Spaniel
- English springer spaniel
- Lhasa apso
- Miniature schnauzer
- Pekingese
- Pug
- Samoyed
- Shih Tzu
- West highland white terrier
- Yorkshire terrier

- Idiopathic
- Debilitated and dehydrated animals
- Trauma
- Drug-induced – this is important to takeinto account when testing STT
  - Sulphonamides
  - Atropine
  - General anaesthesia
  - Topical anaesthesia
  - Etodolac

QUANTITATIVE DISORDERS

Aqueous Deficiency

Most cases will fit into this category. Deficiency of the aqueous portion of the tear film is more commonly seen in dogs than cats or horses, and leads to the typical KCS we see.

The aqueous portion of the tear film is produced by the lacrimal gland and the gland of the third eyelid. Here, true quantitive KCS needs to be differentiated from animals in which corneal drying is due to increased tear film evaporation. Increased evaporation occurs when the ability to blink is decreased due to facial nerve disease, lagopthalmos (incomplete closure of eyelids over the globe), exophthalmos and macroblepharon (abnormally large eyelid opening) which is exceptionally common in the Pug and Pekingese breed. Many of these dogs warrant attention to preventing central dryness.

Decreased corneal sensitivity to discomfort (trigeminal nerve disease) also plays a role in this category and can negatively affect the blink reflex. True quantitive KCS will have a reduced Schirmer Tear Test (STT) value, whereas the other conditions mentioned will be normal.

QUALITATIVE DISORDERS

Mucin deficiency

The mucin component of the tear film is produced mainly by the conjunctival goblet cells and is the innermost layer. Decreased numbers of conjunctival goblet cells or changes to the corneal and conjunctival epithelial cell glycocalyx can a cause mucin deficiency. Clinical signs may be mild, such as conjunctival hyperaemia, with normal to increased STT values. The latter may occur due to increased aqueous tear production in an attempt to compensate for a loss of mucin. Mucins are required to hydrate, lubricate and protect the ocular surface. Additionally they provide an "anchor" for the aqueous portion of the tear film to the hydrophobic ocular epithelium.

A reduced Tear Film Break Up Time (TBUT) test is used to assess for a qualitative defect in tear production.

- Apply 1 drop of fluorescein stain to the eye, holding the eyelids open.
- Under cobalt-blue illumination, examine the cornea. Note how many seconds it takes for dark spots to appear as the tear film "breaks up" the fluorescein layer.
- A normal TBUT is approximately 20 seconds in dogs and 17 seconds in cats. Animals with quantitative deficiencies often have a TBUT of < 5 seconds, which indicates an unstable tear film.

Alternatively, a conjunctival biopsy and histopathology can be done to quantify the goblet cell density.

Lipid deficiency

The lipid portion of the tear film is the outermost layer and hence its loss causes rapid evaporation. Lipid deficiency is probably one of the most under diagnosed conditions. Lipids are produced by the meibomian glands on the eyelid margin. Blepharitis (e.g. caused by Staphylococcus sp. or Malassezia), seborrhoea, atopy, meibomitis and even demodicosis can all affect lipid production. Clinical signs include swollen, hyperaemic eyelid margins, "bulging" of the gland opening, chalazia, exudative crust formation of the eyelid margins and a whitish-yellow appearance of the glands when viewed from the conjunctival surface. Gland expression yields a thickened, caseous-type substance. Mild keratitis may be present with conjunctival hyperaemia and mucoid discharge.

As we broaden our view on the varying types of KCS, we aim to have a better understanding of the underlying pathophysiology in order to provide a holistic and individual approach to the management to each patient.

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Feline CKD Therapeutic Goals

Quality of life and maintaining the human-animal bond are the primary focus

When it comes to therapeutic goals in manaing chronic kidney disease in cats, the target has moved. Yes, you still monitor and manage azotaemia, blood pressure and urine protein loss, but there's a whole new realm of client-centered care that dictates how you set your goals.

In real life, this looks like:

- Ensuring normal interactions with the family
- Ensuring normal day-to-day behavioural patterns
- Providing pain management
- Avoiding polypharmacy (see "The fallacy of polypharmacy in CKD")

The fallacy of polypharmacy in CKD

Polypharmacy signifies using many drugs at the same time to treat one condition. Just because you can prescribe more medication to manage CKD doesn't mean you should, says Dr. St. Denis says. This is because prescribing multiple medications may interfere with your primary therapeutic goals of maintaining the client-cat bond and quality of life.

Pick treatments based on evidence-based medicine and known outcomes and those the client is willing to give. Avoid empirical treatments. Before adding a medication into a CKD cat's daily regimen, ask the client, "How do you feel about this? How are you doing with your medicating?" When you're working with a client to design a treatment plan for their CKD kitty, talk less and listen more. Advice from Fetch dvm360 speaker Kelly St. Denis, DVM, DABVP (feline), owner of Charing Cross Cat Clinic in Brantford, Ontario,will help you achieve these goals in your practice.

Early Intervention

Early intervention is key, and starting conversations

about therapy when cats are in the early stages of CKD (International Renal Interest Society [IRIS] stages 1 and 2) will help pave the way to successful management. These early stages are a good time to talk about nutritional changes. For example, if the client is feeding Fancy Feast, which is high in phosphorus, then talk about switching to a high-quality senior diet.

This is also a good time to plant the seeds about regular follow-up and explain that it's important to track changes over time. These simple conversations will help guide your client and avoid overwhelming them, which erodes the human-animal bond. Early CKD is also a good time to talk about doing some additional diagnostics to determine if there's identifiable disease that can be treated, such as imaging to look for kidney stones or urine cultures to look for chronic infection, says Dr. St. Denis. If there are money constraints, then you can plan to test over time to help soften the financial impact. Utilize the phrase "If not today, how about next month?" to direct your client's actions.

Therapeutic kidney diets—they get the job done

The most important thing to ponder about nutrition in feline CKD is whether the cat is eating enough, Dr. St. Denis says. This is more important than what the cat is eating. Maintaining adequate caloric intake and muscle mass is critical to avoid protein malnutrition. But, as you already know, ensuring adequate intake in a cat can be very challenging. Working with the client and providing education in this area is an important therapeutic strategy. If the cat isn't eating enough or is underweight, then Dr. St. Denis recommends feeding 1.2 to 1.4 times the resting energy requirement (RER). Most geriatric cats need at least 1.1 times RER.

We all know that switching diets on a CKD cat that's experiencing decreased appetite can be challenging (understatement of the century). Dr. St. Denis recommends changing the diet earlier rather than later. Cats with CKD should be eating a diet that contains restricted phosphorus and highly digestible protein that meets or exceeds minimum standards. A therapeutic kidney diet that meets protein needs and contains complete amino acid profiles, plus omega-3 fatty acids, which have renal protective properties, will help ensure that your clients' cats will get everything they need.

Old, scrappy and not hungry

If your CKD patient isn't eating, determine if the inappetence or nausea is due to a contributing disease, dehydration or azotaemia and treat that first, before starting appetite stimulants. Maropitant is safe to use in cats with CKD, and it's labeled for oral, subcutaneous and intravenous administration. Ondansetron is also effective for nausea, either orally or intravenously. Mirtazapine also works well for nausea and inappetence. If the cat won't eat or is severely malnourished, don't hesitate to place a feeding tube to ensure caloric intake and medication administration.

What about uraemic gastritis and ulcers? Historically, we prescribed antacids in CKD cats to prevent gastritis, but a new study has found that cats with CKD may not have elevated stomach acidity as compared with healthy cats.[1] Dr. St. Denis hypothesizes that the inappetence associated with uraemia is more likely due to chemoreceptors in the brain and less likely to be associated with elevated gastric acidity, making antacid treatment in cats with CKD less likely to help with nausea and inappetence. Avoid polypharmacy! Don't reach for the antacid unless you are seeing melena or bloody vomitus.

If nausea is under control, then appetite stimulants are helpful. In fact, the FDA recently approved a transdermal formulation of mirtazapine for cats. Mirtazapine can be used daily, but if you're noticing serotonin syndrome with administration, ("hyperactive meowing," as Dr. St. Denis calls it), then dial down the dose and use cyproheptadine as an antidote.

Pain management

When I graduated from vet school, we didn't automatically associate pain with feline CKD, but now we know that these cats are hiding pain, due not only to CKD but also to occult degenerative joint disease (DJD), which is prevalent and underreported in senior cats and a serious obstacle to a good quality-of-life goal. Dr. St. Denis recommends talking to your clients about subtle signs of stress and pain from DJD and getting these cats on pain management sooner rather than later.

Here are Dr. St. Denis' favorite pain medications for cats:

- Buprenorphine, as a sustained release formulation or given intravenously for hospitalised patients
- Gabapentin (Dr. St. Denis recommends starting with evening administration because of initial ataxia and sedation and says to be sure to advise clients of initial side effects)
- Nonsteroidal anti-inflammatory drugs (NSAIDs): robenacoxib (Onsior—Elanco) and meloxicam have good studies that show safe long-term administration, provided that the cat is hydrated and properly monitored and that the client understands the risks and benefits.
- Ketamine intravenous constant-rate infusion for hospitalised patients
- Polysulfated glycosaminoglycan (Adequan—Luitpold Animal Health)
- Duralactin (PRN Pharmacal)
- Prescription Diet k/d + Mobility (Hill's).

High blood pressure

Amlodipine is the most effective drug for high blood pressure in cats. But don't use it unless there's direct evidence of target organ damage, says Dr. St. Denis. Clinicians should also be certain the cat is hypertensive, following recommendations from the International Society of Feline Medicine (ISFM) guidelines for managing hypertension. She starts with 0.625 mg every 24 hours and reassesses in three to five days, and if blood pressure is higher than 160 mm Hg, she increases the dosage by 0.5 mg/kg/day. She maxes out amlodipine dosages at 2.5 mg/cat. Monitor blood pressure every three to four months.

If amlodipine is not controlling blood pressure, first check adherence with the client. If everything checks out and you've maxed out your amlodipine dosage, Dr. St. Denis recommends adding in telmisartan (Semintra), a new angiotensin receptor blocker from Boehringer Ingelheim. Anecdotally, Dr. St. Denis finds it is a nice adjunct to amlodipine administration in uncontrolled hypertensive cases.

What to do about phosphorus and calcium concentrations

Therapeutic kidney diets are the first line of defense against abnormal calcium and phosphorus concentrations. If you find that phosphorus is still increasing or unacceptably high even if a cat is on a therapeutic diet, then a phosphate binder, such as aluminum hydroxide, is in order, Dr. St. Denis says.

She advises avoiding any aluminum hydroxide products that contain calcium, splitting the daily dose, and giving with food because phosphate binders bind to phosphate in food.

Renal secondary hyperparathyroidism is due to deficiency of calcitriol, which causes calcium dysregulation and increased ionized blood calcium in the bloodstream. If you notice hypercalcemia, it's important to measure parathyroid hormone (PTH) and ionized calcium concentrations. If the PTH is normal or low, then hypercalcaemia is not due to renal secondary hyperparathyroidism.

If you elect to prescribe calcitriol to manage calcium concentrations, Dr. St. Denis says to give twice weekly, ensure that phosphorus concentrations are less than 6 mg/dl in the blood, and monitor regularly. Check ionized calcium and PTH concentrations every four to six weeks until they normalize, then check them every three to six months, depending on what your client is willing to do.

Reference available online

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Techniques for Better Radiographs

By Michelle Fabiani, DVM, DACVR
VETTED

A radiograph can quickly become an expensive and a dangerous waste of time (think of that X-ray exposure!) if it's not showing what is needed. Here are some tips to make you a pro.

The lights in my office are off—the room dark the way I like it. The barks, meows and other various noises of our busy practice are hushed outside my closed door, which prevents interruption of my structured evaluation of each film. I search every pixel of my two black-and-white, high-definition medical-grade monitors. I'm intent on finding the cause of this patient's breathing difficulties.

Even though I will review radiographs on 25,000 patients this year, this one is exceptionally challenging. There's an entire portion of the lung field that's all black. This area lacks information, as it is burned through. I need to see what's not possible to visualize. I don't have the needed information to obtain a diagnosis for this sick patient. I sigh. I cannot help this one this time.

With the benefit of high-quality images, a teleradiologist can help obtain an accurate diagnosis and improve the well-being of patients all over the globe, including those at your practice. I've worked in both general and emergency practices. I understand you're busy! Almost every veterinarian and veterinary staff member I've ever met is empathetic and caring and wants to do what is best for the pet.

Most days I know it feels like you are running back and forth all day without a single second to do even one more thing. Your technician takes the radiograph of Fluffy you ask for. You look at the films and decide you want a consult. A history is necessary. The technician sees that Fluffy came in for coughing. The tech knows you're busy and doesn't want to bother you with one more question, so the tech puts "coughing" down for history and off the consult goes. You feel a little victory and think, Great, now that the radiographs are submitted, I'll know what is going on by the end of the day! But sometimes teleradiologistis can't help with limited history such as "coughing."

I like to say that general practitioners know information that is a mile wide—spanning several animal species, breeds and diseases. Conversely, I know information specific to radiology that is only an inch wide—but it's a mile deep. As a radiologist, all I do all day, every day is imaging. All of my journal reading and continuing education meetings involve what is new in imaging. What's the best modality to diagnose a disease? How sensitive and specific is that diagnosis? How can

diagnostic imaging techniques help our patients? As a result, radiologists' understanding of what diseases look like is vast and helpful, but only if we're provided high-quality images and high-quality supporting information.

That's why taking time to support better understanding of effective radiology practices is imperative, and improves the diagnostic quality of every imaging study performed in the future. In doing so, we ensure greater accuracy in diagnoses and further support the well-being of our patients. Below are seven techniques veterinarians and technicians can use to take better images today.

1. No mystery in history

One challenge of working as a teleradiologist is that I never see my patients in person. Without physically examining them, being familiar with their history and talking to the owner, quite a bit of background can be lost. It's critical for whomever is taking and sending the radiographs, whether veterinarian or technician, to provide context that can help my assessment.

One practice we've implemented in our hospital is for the veterinarian to fill out the physical exam and history sections, thus providing more detail and nuance that can be quickly relayed to a technician or read from the presenting complaint in the chart. Our doctors also copy and paste pertinent parts of the electronic medical record into the radiology consult, relaying additional information to the radiologist.

Succinct but thorough information, coupled with our understanding of breed- and-age-related diseases, can help tailor a thought process in order to arrive at a more accurate diagnosis. Otherwise, I won't know the "coughing," 20-lb, mixed breed is actually an obese, 12-year-old Yorkie that lives in a house with a chain smoker, reeks of smoke and never goes outside. That kind of information changes how helpful a teleradiologist is to you.

2. Collimate and compare

It seems obvious, but before taking a radiograph it's important to know what you're looking at and where you're looking. For instance, when evaluating a patient with lameness, an examination should further direct you to a localized area like the hip, stifle joint or foot. Radiographs should then be focused only on this area, with the remaining areas collimated out of the primary radiographic beam.

The benefit to collimating—focusing in on a single region of the patient's body is to improve the image quality of that region (Figures 1 and 2). There are only so many pixels on a digital radiograph plate. If the majority of the radiograph isn't really part of the area that you're interested in (for instance, taking a whole-body radiograph for a stifle lameness), then as a result, the majority of the pixels are useless. The actual area

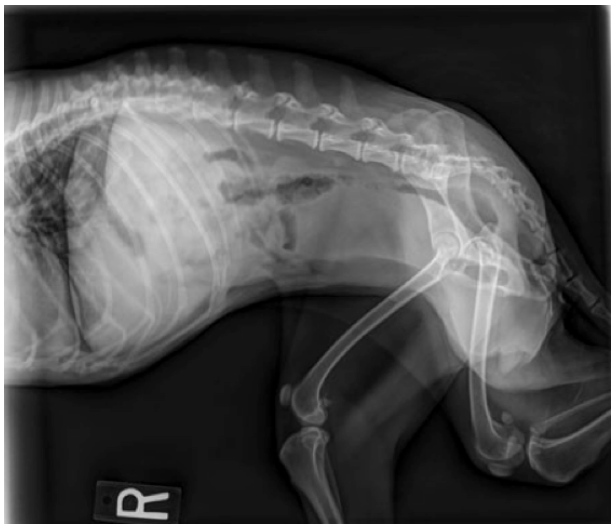

Figure 1. This lateral radiograph was taken without sedation and without collimation, significantly limiting interpretation of the spine. (Photos courtesy of Dr. Michelle Fabiani)

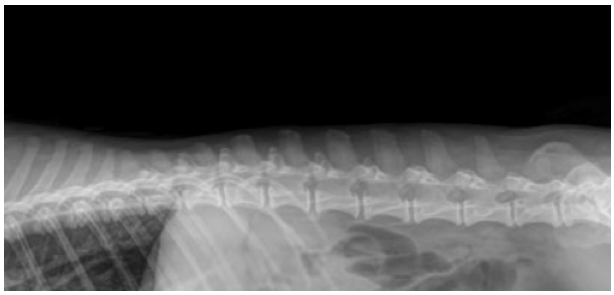

Figure 2. This radiograph was obtained after sedation. Adequate centering of the radiograph beam and collimation were utilized allowing for excellent interpretation of the lumbar spine.

of interest doesn't have enough resolution to evaluate adequately.

How can you tell if this is happening on your images? Magnify to the region of interest and see if you notice a pixelated appearance. Collimating provides better image resolution and greater ability to accurately diagnose, resulting in better patient outcomes.

Additionally, most of our patients have a conveniently accessible normal leg that we can use for comparison. If the left elbow is where the pain is localised, you should also take an image of the right elbow so we have an idea of what this patient's normal looks like. And remember you should always take images in orthogonal pairs—both a lateral and a cranial to caudal—of the affected and the normal leg. For most diagnostic images, you should be taking separate radiographs of each leg—that is, don't take both elbows in one lateral view nor both elbows in one cranial to caudal view.

3. Wanna be sedated?

At some point, we've all dealt with an angry cat whose claws create havoc in the clinic. Or a dog whose long limbs flailed on an imaging table. Their fear-based response to an exam or procedure can harm

themselves and staff alike. In this excited state, the chances of taking an image that is useful in making a diagnosis are low, and you've charged the client for an unnecessary procedure as well as irradiated the staff unnecessarily.

As part of our initiative to provide better patient care at our specialty clinic, we're becoming a Fear Free hospital. We take patient stress, pain and fear seriously. Sedation is standard for all patient imaging in Fear Free hospitals—not only radiographs, but all other imaging as well. Our goal is to make the entire patient visit (including imaging) a positive, calm, stress-free event, which ultimately makes the process safer for our staff and also increases the quality of images obtained.

Through sedation, we both assure patient safety and our ability to take the diagnostic images necessary to support treatment of the patient. Sedation also decreases radiation exposure to our staff because those images we do take are of the necessary quality and don't need to be repeated multiple times. For us, sedation is better for the patient and the staff—and it's better medicine.

4. Don't take it easy

Many images I see are taken from what I'd call the "easy" perspective, with the patient laying on its side—that is, a lateral view. Taking a single view from that position is one of the biggest ways to miss pathology. Taking two images (one with the patient on their side and the other with the patient on their back) should be standard for every patient and is always worth the effort for the significant amount of diagnostic return on investment. Because most of our patients are skinny, ventrodorsal images can be taken more easily with the help of a soft, padded, V-shaped trough. These are inexpensive, easy to clean positioning devices that help the patient lie both comfortably and still on their back for the duration of the imaging study.

5. A snapshot versus a photograph

I work closely with both general practices as well as my radiology technicians, educating them about how to obtain a high-quality radiograph. For those individuals who haven't received appropriate training, they may assume that if the patient's in the radiograph, it's also of adequate diagnostic quality. This is not their fault—it's simply lack of training! In order to support understanding of a quality radiograph, I teach technicians and veterinarians objective criteria that can be easily utilised while obtaining the images.

For thoracic radiographs, the position of the retrosternal lucency and the lumbodiaphragmatic recess during inspiration can be counted and compared to rib position. It's easy to count the vertebrae compared to the position of the diaphragmatic cupola and the costodiaphragmatic recess as well. How do I know if the ventrodorsal radiograph is straight? Look to see if the sternum is on top of the vertebrae. Providing specific, objective criteria will help everyone assess the quality of their own images while the patient's still on the imaging table and helps determine if the radiograph needs to be taken again.

Anyone can take a snapshot on their phone. It takes a lot of training, practice and understanding to actually be a photographer. This is the difference between someone who can take a radiograph by pushing the expose button and someone who can take a good quality diagnostic radiograph. Training results in expertise, which in the end is an exceptional value for the patient and practice.

6. It's all in the timing

Haven't we always heard, "it's all about timing"? That adage is definitely true in radiology. An image of the thorax should be taken during inspiration, when the lungs are completely inflated. Conversely, the ideal time to take an abdominal radiograph is at complete expiration, when the lungs are the smallest. When the lungs are halfway between inflation and expiration, you're taking a poor image of both areas. When an x-ray machine salesman says that a single whole-body radiograph can be taken versus taking a collimated radiograph of either the thorax or abdomen, he's not telling you what is best practice or most diagnostic. He hasn't gone to veterinary school, nor is he a radiologist. Remember, just because you can do something doesn't mean you should. When you collimate, focusing on a specific region of the body with the appropriate image timing, you do the best for your patients and your clients.

7. Marker my words (and images)

Many practices have grown accustomed to using digitised right or left markers, added to the image after it is taken. Unfortunately, sometimes those digital markers don't transfer when images are sent to the radiologist. The result? Time-consuming phone calls to discuss what images were obtained. This can ultimately end in the teleradiologist not knowing left from right, thus limiting the ability to obtain a useful diagnosis.

We encourage the use of an actual physical lead marker placed in the primary radiographic beam on every image. Markers are a quick and inexpensive solution that supports the accurate interpretation of images by a teleradiologist.

Now it's time to put these tips to work!
Using the above helpful techniques will help us radiologists help you. This will ultimately help all your patients. After all, isn't that why we all got into the veterinary field? Happy imaging!

The ABCs of Veterinary Dentistry

'N' is for No

By nature of the alphabet, we must get through all of the noes in veterinary dentistry before we can reach the yeses—but that doesn't mean you won't feel positively inspired to better your dental practices after reading.

By Jan Bellows, DVM, DAVDC, DABVP, FAVD
DVM360 MAGAZINE

As veterinarians, we respond to clinical signs in our patients and do something about them. But knowing what not to do is just as important. Here are 14 things to say no to in veterinary dentistry:

1. Say no to treatment estimates related to oral malodour (halitosis) before you've examined the entire mouth—including every tooth

Quoting a fee (or even a fee range) for "bad breath" before you know the cause may lead to a disgruntled client and an untreated patient once you discover that a dozen teeth suffer from advanced periodontal disease and need to be extracted. Instead, let your client know you'll call while the pet is anesthetised to discuss what care the pet needs after dental scaling, probing and full-mouth intraoral radiographs.

2. Say no to dental procedures without general anaesthesia

Anaesthesia allows the practitioner and assistants to carry out dental procedures safely and effectively, minimizing the risk of injury to the team, equipment and patient. The American Veterinary Dental College (AVDC) launched a website to deter pet owners and veterinarians from considering anaesthesia-free dental cleanings in any context. It advises pet owners that "Anaesthesia-free dental cleanings provide no benefit to your pet and do not prevent periodontal disease at any level. In fact, it gives you a false sense of security as a pet owner that because the teeth look whiter that they are healthier." A similar position statement was ratified by the American Veterinary Medical Association: "When procedures such as periodontal probing, intraoral radiography, dental scaling, and dental extraction are justified by the oral examination, they should be performed under anaesthesia."

3. Say no to dental procedures without an examination

In some veterinary clinics, the pet owner calls the office to arrange a drop-off for a teeth cleaning because the pet has oral malodour. But if your dental assistant only removes the pet's plaque and tartar from the crowns without a tooth-by-tooth examination, you've accomplished little besides the cosmetic removal of crown debris. Oral malodour

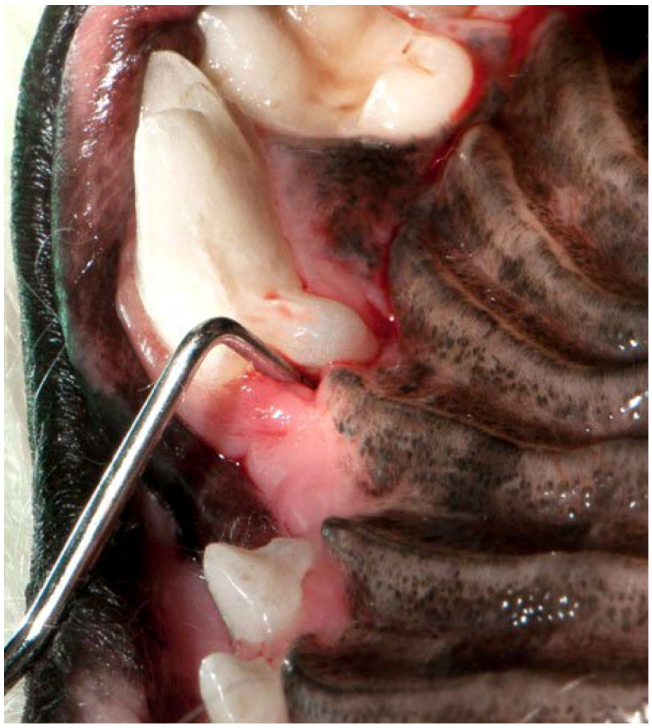

Figure 1A. 12-mm probing depth along the mesial aspects of the left maxillary fourth premolar; extraction indicated. (All images courtesy of Dr. Jan Bellows.)

occurs secondary to food putrefying in periodontal pockets. Unless you treat the pockets (through deep scaling and root planing, gingivectomy or placement of local antimicrobials into cleaned pockets) and institute home care, malodour will soon return and periodontal disease will progress.

A healthier way to approach dentistry with long-term positive results is to examine the conscious patient first (including the oral cavity), followed by a tooth-by-tooth examination under general anaesthesia (with probing and intraoral radiology). If the tooth and support structures are in good shape, move on to the next tooth. If not, diagnose the pathology and formulate a treatment and prevention plan (Figures 1A-1D).

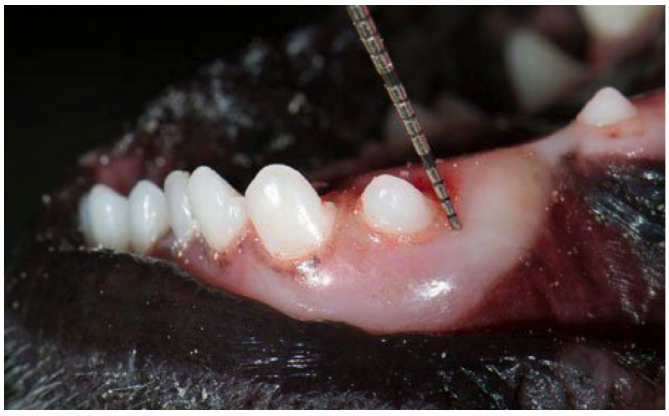

Figure 1B. Periodontal probe before insertion into a dog's partially erupted left mandibular canine

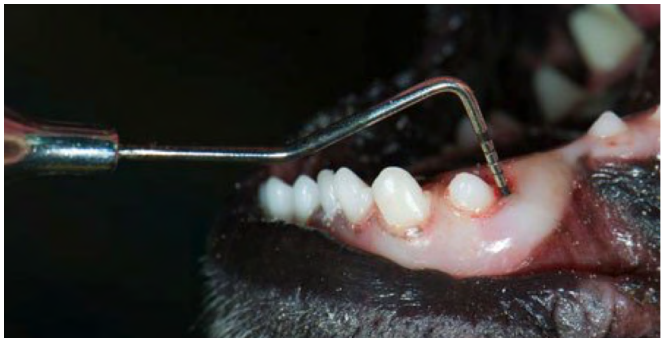

Figure 1C. 10-mm periodontal pocket; gingivectomy, mucogingival surgery or extraction indicated.

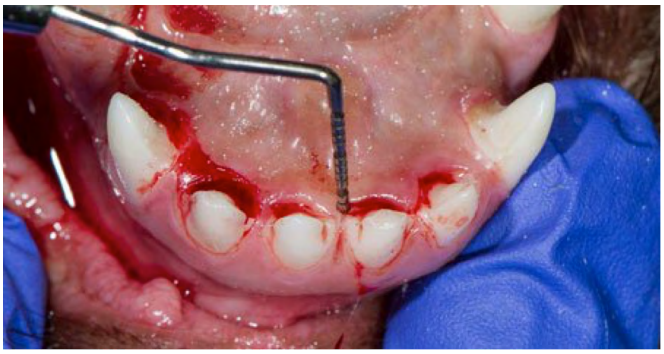

Figure 1D. Bleeding on probing with 3-mm periodontal pockets; root planing and instillation of local antibiotics indicated.

4. Say no to the following phrase: "The patient is here for a dental today ..."

When properly performed, what we do is a comprehensive oral prevention, assessment and treatment visit. If you regularly vocalize all of these terms, the client develops a better understanding of and appreciation for what's involved. Note that prevention is listed first. Stressing prevention first will hopefully result in less future discomfort and extractions.

5. Say no to too many dental cases per veterinarian per day

Once the entire team embraces the comprehensive oral prevention, assessment and treatment concept, everyone wins—especially the patient. And with 42 "patients" in every normal dog's mouth and 30 in every normal cat's, you'll need to give yourself a lot of time to properly treat the cause of oral malodour.

6. Say no to dentistry without patient warming systems

Small animal dental procedures are commonly conducted in an air-conditioned environment, which decreases the patient's core body temperature over time. Dental diagnostic and treatment procedures can be lengthy, and managing the patient's core body temperature is recognized as one of the best ways to minimize the risk of an anesthetic complication. Careful monitoring and treatment of falling body temperature can help you avoid significant physiological and surgical complications as well. The patient's temperature is monitored through esophageal or rectal probes, with the former being a more accurate representation of core body temperature. Provide a safe method of thermal support such as forced air and radiant heating systems (Figure 2). You must take care to avoid thermal injury to the skin with other types of heating devices

7. Say no to dental diagnostics and extractions without full-mouth radiographs

Scaling plaque and calculus from crowns and probing pockets only goes so far. At least 60% of the patient's teeth

Figure 2. Radiant energy warming device (Hot Dog Patient Warming System).

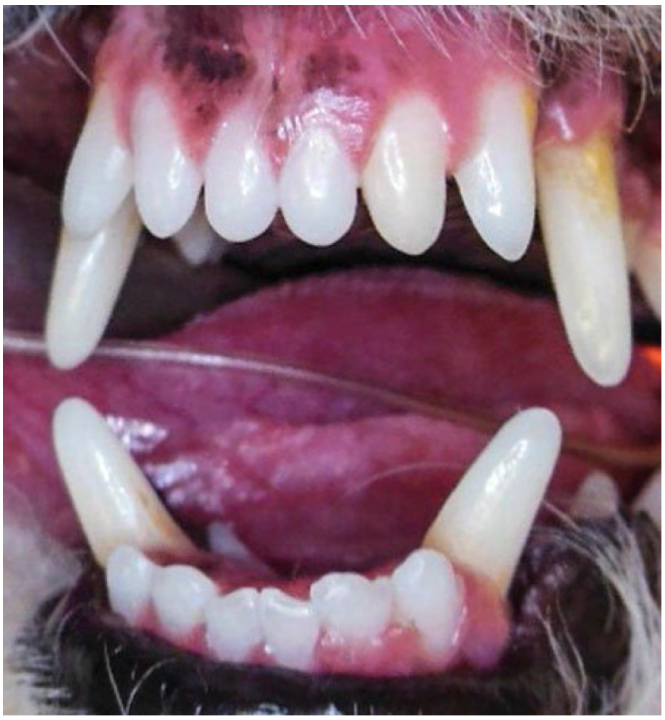

Figure 3A. Seemingly normal incisors in a canine patient.

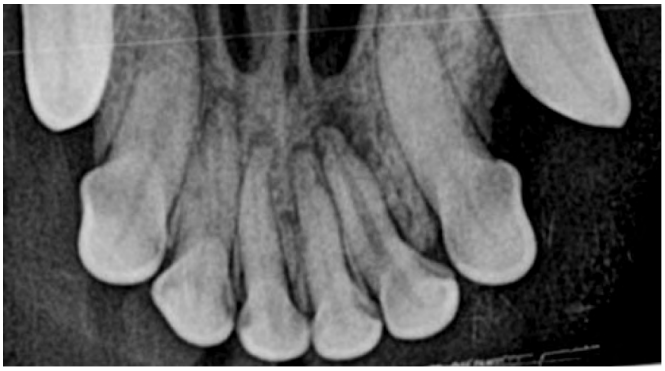

Figure 3B. Enlarged root canal and periapical lucency consistent with a nonvital tooth in the canine patient from Figure 3A; root canal therapy or extraction is indicated.

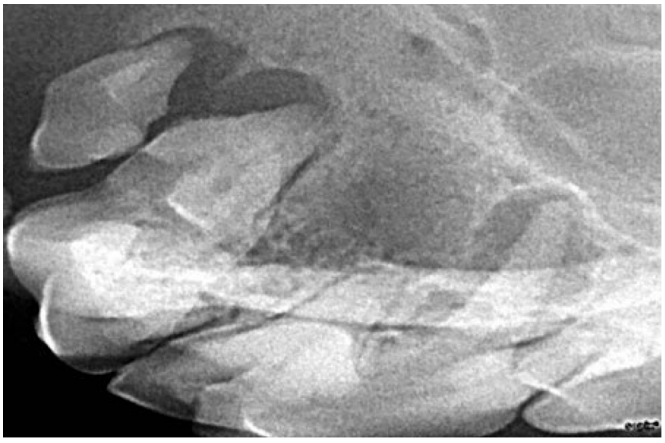

Figure 3C. Advanced periodontal disease in the canine patient from Figure 3A affecting the apices of the right maxillary fourth premolar and the first and second molars; extractions indicated.

lie below the gum line. Intraoral radiographs are essential to help you evaluate these areas (Figures 3A-3D).

8. Say no to technician extractions

Some state practice acts allow technicians to extract teeth; however, veterinarians are the only professionals allowed to perform animal surgery. Surgery is defined as opening a body part to treat disease using instruments. Operative dentistry is surgery. Our veterinary degrees specify veterinary medicine, surgery and dentistry, and we have the most knowledge and experience regarding our patients' anatomy and physiology and how their tissues react to surgery. None of us would allow a human dental assistant or hygienist to extract our teeth. Why should it be different for veterinary patients?

9. Say no to unsterilized and dull instruments

Can you imagine your dentist opening up a drawer and rummaging through the instruments inside before dipping them in cold sterile solution to extract your tooth? Sterilized packs for diagnostics (periodontal probe, mirror and curette), extractions (separate packs for feline and small, medium and large dogs) and oral surgery make great sense. To increase efficiency, keep all of the instruments you need for a specific procedure together in one sterile pouch or cassette. Charging a "sterile surgical pack fee" easily covers the expense of additional instruments and sterilization. Your instruments need to be sharp, too. Before sterilization, sharpen your curettes and your wing-tipped and periosteal elevators with an oiled sharpening stone (Figures 4A-4D).

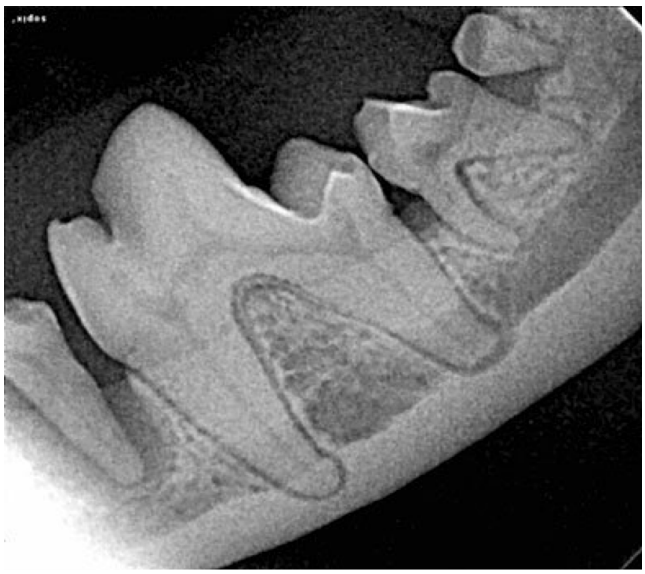

Figure 3D. Advanced periodontal disease in the canine patient from 3A affecting the left mandibular fourth premolar and second and third molars; extractions indicated. Stage 3 periodontal disease affecting the left mandibular first molar; root planing coupled with home care or extraction indicated.

Figure 4A. Unsterile surgical instruments; note the dog hairs.

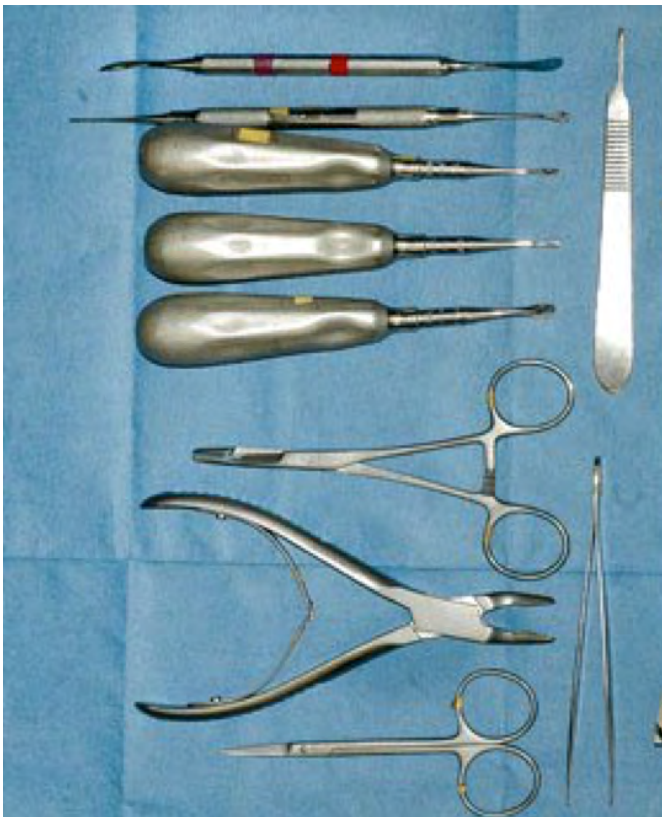

Figure 4B. Instruments in the author's extraction pack.

Figure 4C. Sharpening stone and oil.

Figure 4D

10 Say no to extractions without regional anaesthesia and postoperative pain medication

No animals under our care should experience pain when it can be prevented. The benefits of regional anaesthesia include decreased pain during and after surgical procedures, decreased risk of vagally mediated reflex bradycardia, lower inhalant requirements, and a level plane of general anaesthesia reducing the variation of anesthetic depth when painful stimulation occurs.

The three most common regional blocks in veterinary dentistry are the caudal maxillary, infraorbital and caudal mandibular blocks. Frequently administered single-agent local anaesthetics include lidocaine and bupivacaine. Many practices use a combination of 0.5% bupivacaine hydrochloride with epinephrine (Marcaine) (1 mg/kg) and lidocaine 2% (1 mg/kg) in a 4:1 ratio. Mixing 0.8 ml of bupivacaine with 0.2 ml of lidocaine in the same tuberculin syringe accomplishes the 4:1 ratio.

The recommended volume for regional anaesthesia is 0.1-0.3 ml per injection site. Maximum patient dosage of this mixture is 0.2 ml/kg bupivacaine, or approximately 0.25 ml per jaw quadrant (in case all quadrants need anaesthesia for a 5-kg cat or dog).

Another option is to mix small volumes of an opioid with a local anaesthetic. Buprenorphine has been shown to extend anaesthetic duration up to threefold compared with bupivacaine alone. Small volumes of buprenorphine 0.003 mg/kg can be mixed with bupivacaine hydrochloride in the patient's regional block volume.All dogs and cats should receive postoperative pain relief medication after extractions for at least three days.

11. Say no to spring-loaded mouth gags

You can insert a mouth prop or lap sponge between the maxillary and mandibular canines or between the cheek teeth to keep the mouth open during dental procedures. Placing spring-loaded gags between canines is not recommended because of potential iatrogenic damage to the teeth, temporomandibular articulation and decreased maxillary blood flow to the brain. In cats, this decreased cerebral blood flow may result in neurological impairment, including blindness. Alternatively, cut endotracheal tubes or syringes will prop the mouth open while allowing for flexibility (Figure 5).

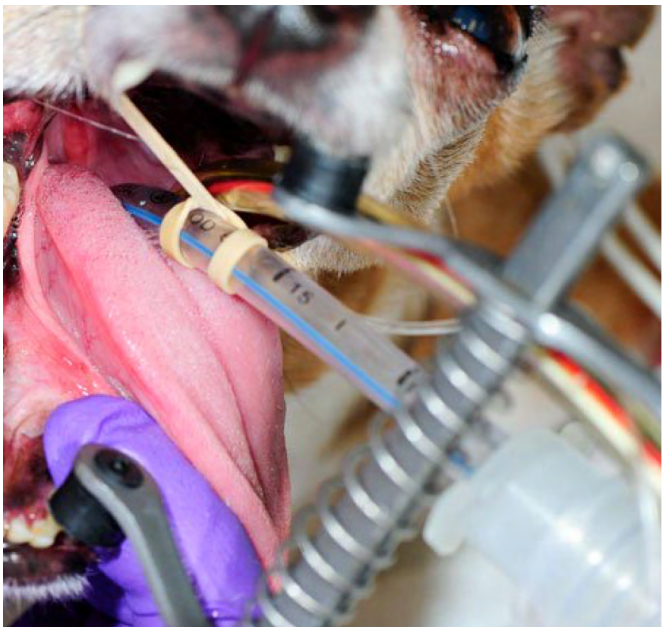

Figure 5A. Don't use spring-loaded mouth gags like this one. They can cause overextension of the temporomandibular joint.

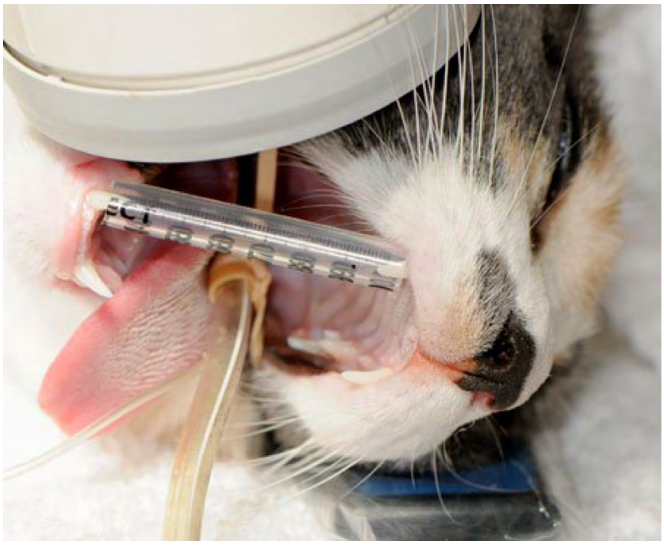

Figure 5B. Don't overextend the temporomandibular joint as in this image with a cut syringe.

12. Say no to systemic antibiotics—except in cases of advanced periodontal disease or in compromised patients

Forty years ago, the standard of care included giving a penicillin-streptomycin injection after every ovariohysterectomy. Once the science proved this wasn't necessary, it stopped. Similarly, systemic antibiotics are not indicated before, during or after most dental procedures, other than cases of multiple extractions for advanced periodontal disease where we are removing the cause of the problem (e.g. plaque, tartar and periodontally affected teeth). There appears to be a place for local administration of antimicrobials, however, especially in cleaned periodontal pockets less than 5 mm (Figure 6).

Figure 6. Injection of local antibiotic into a cleaned stage 2 periodontal pocket.

13. Say no to recommending tooth brushing

Wait. Did you read that right? No to the gold standard of tooth brushing? What's wrong with tooth brushing? Simple: Virtually no one does it twice a day, or once a day, or even every other day, and anything less is worthless. So instead of continuing to push brushing, recommend twice-daily wipes, cotton-tipped applicators (in cats) rubbed along the gingival margin, and accepted Veterinary Oral Health Council products to help decrease the formation of plaque and tartar.

14. Say no to forgetting to schedule follow-up dental examinations

Removing the plaque and tartar from crown and root surfaces and extracting teeth affected by advanced periodontal disease without periodic home care monitoring makes little sense. Plaque and tartar will soon return and inflame the gingiva. After the comprehensive oral prevention, assessment and treatment visit, schedule a follow-up appointment to discuss a tailored plaque control program, including monthly to quarterly rechecks, to monitor compliance and efficacy.

Now that the negativity of saying no is out of the way, I look forward to getting to "Y" ("Say yes to … ") in a future piece.

Gastrointestinal Therapy
Anti-emetics and Antacids

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The drug arsenal available to the veterinarian to treat gastrointestinal related diseases has seen some advances in recent years.

The release of maropitant on the South African market has replaced some of the older drugs as the empirical choice to manage nausea and vomiting. Various recent studies in the pharmacokinetics of antacids in dogs and cats have suggested that proton pump inhibitors may very well be the only truly clinically useful drugs to prevent and treat conditions manifesting in intestinal ulceration in these species.

ANTIEMETICS

Vomiting is a clinical sign for which owners commonly seek veterinary attention for. Anti-emetics are commonly used to address this complaint and to avoid possible consequences such as dehydration and electrolyte abnormalities due to protracted vomiting. One should however be cautious of using anti-emetics in suspected self-limiting cases as they may mask disease progression.

Maropitant

Maropitant is one of the most effective anti-emetics. It acts by inhibiting the binding of substance P to neurokinin-1 (NK-1) receptors as an antagonist. These NK-1 receptors are found within the vomiting centre, chemoreceptor trigger zone and in vagal afferent nerves in the gastrointestinal tract[1] (Figure 1). This inhibition gives a relatively broad spectrum of coverage against emetogens, compared to, for example, metoclopramide and chlorpromazine, which are

primarily effective against central stimulation.

Maropitant has demonstrated efficacy in dietary indiscretion, pancreatitis, canine parvoviral enteritis and gastritis of unknown aetiology[2]. The lack of prokinetic properties with maropitant allows for safer use in patients with suspected gastrointestinal obstruction relative to for example metoclopramide. A single daily dose of maropitant was also shown to be more effective at managing vomiting due to miscellaneous causes compared to metoclopramide administered two to three times daily[3]. Maropitant has become the standard of care in chemotherapy patients due to its efficacy in preventing vomiting following cisplatin and doxorubicin administration.

Maropitant has a 24-hour duration of effect in dogs, but is cleared more slowly in cats with a half-life of 13-17 hours compared to only 4-hours in dogs[4]. The dosages vary depending on the formulation and required effect.

- The injectable dose of 1mg/kg, q24h, sub-cutaneously should be administered at least 1 hour prior to anaesthesia or chemotherapy for optimal effect.
- The oral dose is higher (2mg/kg, q24h) due to decreased oral bioavailability but food does not affect oral drug absorption.

The intravenous use of maropitant is reportedly well tolerated off-label. A 2-day rest period is recommended following the label dosing maximum of 5 consecutive days of administration. This is due to saturation of the hepatic cytochrome p-450 enzymes responsible for its biotransformation. However, a dose of 2 mg/kg, orally, once daily in healthy beagles was safely administered to healthy beagles for 14 days[5]. Maropitant undergoes primarily hepatic clearance and little renal clearance, which suggests that doses are unlikely to need reduction in renal dysfunction be the drug must be avoided in cases with hepatic dysfunction. In cats, maropitant is approved for injectable use but oral tablets are also commonly prescribed off-label. The drug is highly protein bound which may cause interactions with other highly protein bound drugs such as some non-steroidal anti-inflammatories and benzodiazepines. Adverse effects at label doses are uncommon. Pain on injection can be avoided by refrigerating the vial[6].

Maropitant is not approved for use in puppies younger than 8 weeks of age and kittens under 16 weeks of age. The higher dose for use against motion sickness in puppies is also only approved for puppies 16 weeks or older, this is due to bone marrow hypoplasia being reported in 8-week-old puppies given 6-10mg/kg once daily.

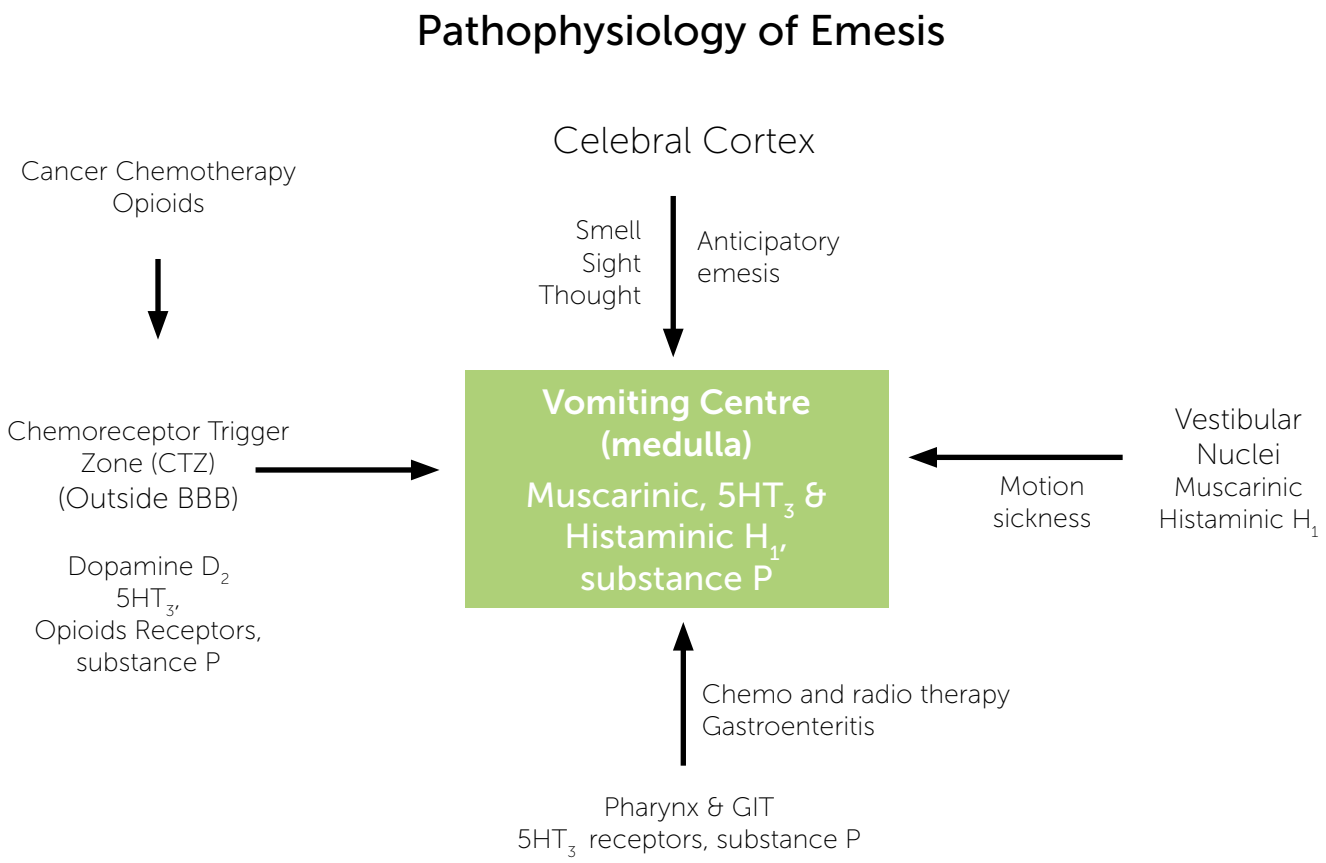

Figure 1: Graphical representation of Pathophysiology of Emesis

Metoclopramide

Metoclopramide is often used as an alternative to maropitant due to its additional weak prokinetic effects on the proximal gastrointestinal tract. This dopamine antagonist is a very effective anti-emetic which acts both centrally and peripherally. By increasing the tone and amplitude of gastric contractions and increasing pressure within the gastroesophageal sphincter, metoclopramide counteracts vomiting.

Metoclopramide is very useful for the treatment of conditions such as oesophagitis, gastric motility disorders but is contraindicated in patients with suspected gastrointestinal obstructions. For this reason, maropitant may be a safer empirical antiemetic to use in suspected self-limiting cases where an intestinal foreign body can't be excluded as a possible cause.

The antiemetic effect of metoclopramide appears to be more pronounced in dogs than cats. This is probably because there are only a few dopamine receptors present in the central nervous system of cats. Metoclopramide is thus a poor antiemetic choice (and apomorphine a poor emetic choice) in this species as both require dopamine receptors.

Metoclopramide is contraindicated in patients with gastrointestinal haemorrhage, obstruction, or perforation and must be used with caution in patients with cranial trauma and phaeochromocytoma. Central nervous system signs are the most common side effect seen with metoclopramide use which may manifest subtly as mild sedation or behaviour change or overt extrapyramidal signs.

Metoclopramide is used in dogs as an intermittent bolus at 0.2 – 0.5 mg/kg, PO, SC or IM, q6-8h or as a constant rate infusion at 0.01 – 0.09 mg/kg/hour. Most package inserts claim that metoclopramide is light sensitive but a study looking at the admixture of metoclopramide with different solutions, including 0.9% NaCl, showed that the drug remains physically and chemically stable for at least 48 hours with a negligible effect of lighting on drug stability[7].

Ondansetron

Ondansetron is the third, commonly used antiemetic at the practitioner's disposal. This serotonergic antagonist has historically been used in cases receiving chemotherapy due to its potent effect of blocking neuronal transmission in the chemoreceptor trigger zone and vagal afferent nerves. As a premedication for chemotherapy, ondansetron is given at 0.5 - 1.0 mg/kg, PO, 30 minutes prior to administration of the chemotherapy.

Its use, at lower doses, has also been described in cases with gastrointestinal causes of vomiting In dogs with canine parvoviral enteritis doses of 0.1 – 0.15mg/kg, slowly IV, q12h have been recommended.

Unfortunately, here is relatively little information available regarding the pharmacokinetics of ondansetron in dogs and cats to define clear clinical applications and indications for the use of this drug, especially when considering that it is expensive. However, ondansetron appears to be well-tolerated in dogs and cats.

Due to the efficacy and relative safety of the aforementioned drugs, the use of phenothiazine derivatives such as chlorpromazine and prochlorperazine have fallen out of favour. Phenothiazine derivatives causes hypotension, which may be dangerous in cases suffering from haemodynamic instability or dehydration, and they also cause tranquilisation even at low doses, which interferes with the assessment of a patient.

ANTACIDS

The integrity of the gastric mucousa is dependent on a delicate balance between ulcerogenic factors and the counteracting defence mechanisms of the gastrointestinal tract. Ulceration of the gastric mucousa may be associated with nonsteroidal anti-inflammatory drug use, excessive gastrin release, excessive histamine release associated with mast cell tumours, hepatic dysfunction, chronic kidney disease, pancreatitis and many more. The primary endogenous ulcerogenic factors are hydrochloric acid and pepsin.

Gastric mucousal defence mechanisms compose of a pre-epithelial mucous-bicarbonate-phospholipid barrier, the epithelial barrier and continuous cell renewal of epithelial cells, continuous mucosal blood flow through gastric microvasculature, an endothelial barrier, sensory innervation and the generation of vasodilatory prostaglandins and vasodilatory nitric oxide. Both the preventative and repair mechanisms are dependent on mucousal prostaglandins. Ulceration therefore occurs in the presence of excessive ulcerogenic factors or impaired defence mechanisms.

Antacids act to antagonise acid production, promote healing or potentiate the natural protective mechanisms of the gastric mucousa. Acid production is stimulated via neuronal and hormonal pathways mediated via the binding of gastrin, acetylcholine, histamine, and prostaglandins to specific receptors on acid-producing parietal cells. Hydrochloric acid is eventually produced from hydrogen molecules secreted by proton pumps on the luminal surface of the parietal cells. The mediators of this physiological process are targeted for reducing acid production as demonstrated in Figure 2.

The invention of catheterless radiotelemetric pH monitoring devices have facilitated the accurate and noninvasive measurement intragastric pH in dogs and cats since 2002. This has resulted in great advances

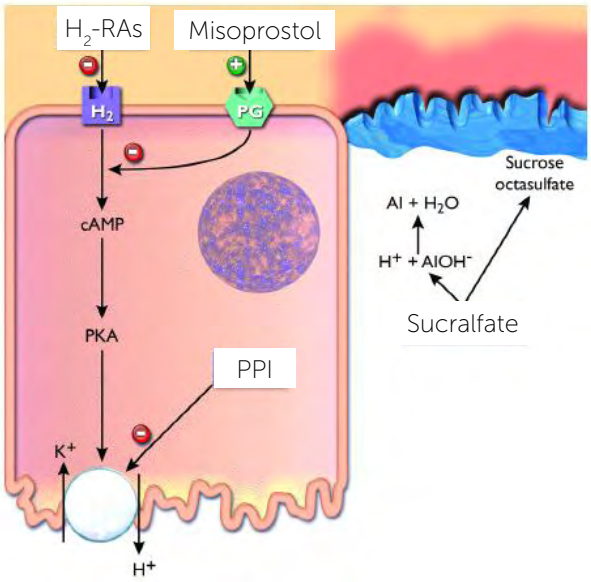

Figure 2: A shematic representation of the gastric mucousal parietal cell. The proton pump is on the luminal cell surface. the receptors for acetylcholine, prostagalndin, gastrin and histamine are on the inner cell membrane. Proton pump inhibitors work on the luminal surface and thus will block the effects of all receptors on the inner cell surface. Sucralfate attached to damaged and exposed gastric mucousa

in understanding of the effects of various antacids on intragastric pH in animals.

Omeprazole

Omeprazole is a potent proton pump inhibitor commonly utilised in the management of gastrointestinal ulceration. Proton pump inhibitors block the final step of acid production on the luminal surface of the parietal cells, thus also blocking the effect of other acid stimulatory factors such as histamine, gastrin and acetylcholine.

Omeprazole acts by irreversibly binding to the proton pump. This initially causes transient activation of additional proton pumps in an attempt to bypass the blockade. Omeprazole thus takes approximately 2-4 days for optimal acid suppressive effects with only a 33% reduction occurring during the first day.

Parietal cells re-establish acid production only by producing new proton pumps lending the drug some lasting effect after discontinuation of a few days. Considering the efficacy of omeprazoles' blockade of the acid production pathway it is not surprising that several recent studies have shown that it is superior to H2 blockers at inhibiting acid secretion. Moreover, additional studies have shown that the twice daily administration of omeprazole further improves acid suppression relative to the previously used once-daily practice in dogs and cats.

Because the amount of proton pump enzymes present in the parietal cell is maximal after a prolonged fast it is advised that proton pump inhibitors be administered 30–60 minutes before feeding for optimal effect. The current recommended dose is 1 mg/kg, q12h, PO. Omeprazole is sensitive to degradation by gastric acid and the enteric-coated granules packaged in gelatin capsules needs to be repackage in gelatin capsules if a smaller dose is needed.

A recent study in cats comparing fractionated omeprazole, enteric-coated omeprazole and famotidine to a placebo indicated that omeprazole is a superior antacid in this species as well and that disruption of the enteric coating did not negate the acid suppressing effect of omeprazole[8].

H2 blockers

Drugs such as ranitidine, cimetidine and famotidine antagonise the H2 receptor on the gastric side of the parietal cell and decrease acid production. However recent studies have raised concerns regarding their lack of efficacy in managing gastric ulceration. Bersenas *et al.* determined the degree of gastric acid suppression associated with the administration of ranitidine, famotidine, pantoprazole and omeprazole compared to saline solution in healthy dogs[9]. Famotidine, pantoprazole, and omeprazole significantly suppressed gastric acid secretion compared to saline and ranitidine failed to do so. In addition, administration of famotidine three times daily instead of twice daily did not affect gastric pH significantly, suggesting that increased frequency of administration of this drug is of no benefit. There also appears to be a diminishing effect over time when this drug is used for a prolonged period[10].

Some clinicians use the histamine antagonists together with a proton pump inhibitor in the initial phase of treatment due to the slight delay in maximal acid suppression seen with proton pump inhibitors compared to the relative quick onset of action with H2 blockers. However, there is concern that this practice may interfere with proton pump inhibitor activation, decreasing the efficacy of this drug. In addition, the approach of using famotidine and a proton pump inhibitor simultaneously was shown not to be superior to using a proton pump inhibitor alone[11].

Ranitidine is approximately 10 times more potent than cimetidine at receptor blockade. With recent evidence suggesting that ranitidine is not superior to saline at causing acid suppression it can be argues that cimetidine would not be affective either. In conclusion, famotidine appears to be the only promising H2 antagonist with some effect on gastric acid suppression and this drug is not readily available in South Africa.

Sucralfate

Sucralfate is commonly used in the treatment of oesophagitis and gastric ulceration. This sulphated disaccharide forms an adherent gel that binds to the ulcerated surface, protecting it from further degradation by hydrochloric acid and pepsin.

Sucralfate also stimulates the synthesis of capillary blood flow-promoting prostaglandins potentiating the natural defence mechanisms of the gastrointestinal tract. Because sucralfate can also bind other oral drugs, medications should be given 1–2 hours prior to sucralfate administration, as absorption is minimal,. Toxicity is uncommon. Long-term use may lead to constipation because of its aluminium content.

There is no evidence to support that simultaneous therapy with other antacids provides added benefit compared to therapy with either sucralfate or an antacid alone. However, conceptually the two differences mechanism of action would suggest that this practice may be beneficial. In cases suffering from oesophagitis it is important to note that only liquid solution is considered to be effective.

Misoprostol

Misoprostol is valuable in managing cases suffering from nonsteroidal anti-inflammatory induced gastric ulceration. This drug is a synthetic prostaglandin E-analogue that can be used prophylactically or as treatment in these cases. It has been shown to significantly reduce aspirin-induced gastrointestinal ulceration compared to a placebo[12]. This study led to the suggested dose of 3 µg/kg, q8h, PO. Where misoprostol was used to prevent ulceration in dogs with intervertebral disc disease which were given a single dose of dexamethasone at 2mg/kg followed by a tapering course of prednisolone (2 mg/kg to 0.5 mg/kg over the course of 5–6 days), no effect was seen at a dose of 2 µg/kg, q8h PO [13]. The most common side effect is diarrhoea and the drug may also cause abortion.

Clinical Application of Antacids

Despite the recent advances in our understanding of antacids in dogs and cats there is very little information on the effect of these drugs in animals with spontaneous disease. Most of the studies to date have been performed in healthy animals utilising criteria put forward in human medicine for the level and duration of acid suppression considered adequate to promote ulcer healing.There is even less information on the possible side effect of long-term use of particularly proton pump inhibitors and H2 antagonists. The lack of information makes it difficult to provide clear clinical guidelines and indications for the use of antacids.

A human a study revealing a highly significant correlation between acid suppression and ulcer healing has solidified the practice of using antacids in gastrointestinal ulceration and has been extrapolated to veterinary medicine [13]. Nowadays proton-pump inhibitors are commonly used in humans to treat all acid-related disorders such as peptic ulcers, Helicobacter-associated atrophic gastritis, mast cell neoplasia associated hyperhistaminaemia, gastroesophageal reflux disease and nonsteroidal anti-inflammatory drugs-associated gastroduodenal ulcers. In addition, antacids are also commonly used in conditions such as gastritis, pancreatitis, hepatic disease and renal disease.

Proton pump inhibitors are commonly employed in the treatment Helicobacter-associated atrophic gastritis despite little evidence to support this practice in dogs. The pathogenesis of gastrointestinal ulceration in hepatic disease is poorly understood and not well described in literature. Further studies are necessary to determine the efficacy of antacids for this application.

Gastrointestinal ulceration associated with hypovolaemia, ischaemia and focal peritonitis as seen in acute pancreatitis cases has led to the prophylactic use of antacids in these patients. However, no benefit was reported with the administration of proton pump inhibitors in a placebo-controlled study in humans with acute pancreatitis[14] and there have been no studies performaed in veterinary medicine to support this practice. Patients suffering from oesophagitis may also benefit from antacid therapy.

Chronic kidney disease is another common condition where antacids may be considered. End-stage renal disease in humans has been associated with hypergastrinaemia, gastritis, gastrointestinal bleeding and gastrointestinal ulceration but there are no reports evaluating the gastric acid secretion profile in dogs or cats with renal disease. Recent evidence suggests that gastric ulceration is not a feature of chronic kidney disease in cats as is seen in dogs and humans with uraemic gastritis[15]. This finding suggests that there is little evidence to justify the use of antacids prophylactically in cats with uraemia. A recent study evaluating the use of sucralfate as a phosphate binder in cats with chronic kidney disease was unable to confirm any benefit for its use in cats and the study had to be discontinued prematurely due to excessive vomiting, anorexia and increases in azotaemia in the group with chronic kidney disease[16].
Generally, there are less studies critically evaluating the efficacy of antacids in cats compared to dogs and most dosages used in cats have been extrapolated form those used in dogs.

Adverse Effects of Antacids

The expected adverse effects of chronic acid suppressant administration in dogs and cats have largely been extrapolated from that described in humans. Cobalamin deficiency, diarrhoea, *Clostridium difficile* infection, increased risk of hospital-acquired pneumonia and increased risk of fractures in geriatric people are the most commonly described side effects[17].

Diarrhoea is the most common adverse effect reported in association with proton pump inhibitor administration in dogs but does not appear to be common in cats[18]. The possible effects of proton

pump inhibitors on calcium metabolism and bone remodelling in dogs and cats suffering from chronic kidney disease may further increase the risk of osteoporosis and pathologic bone fractures in this population.

Early studies in cats did not show overt changes in serum cobalamin levels but did raise concerns for changes in bone mineral density. For these reasons antacids should be used judiciously in dogs and cats, especially when suffering from renal disease, and its use should be limited to patients with clear risk factors or overt evidence of gastrointestinal ulceration or gastrointestinal haemorrhage.

SUMMARY

To summarise, our understanding of antiemetics and antacids have improved significantly over the last few years and is likely to continue to change and improve in the near future with new research. Maropitant has largely replaced many of the older antiemetics as an empirical choice and can often be used in combination with older antiemetics in refractory cases with even greater success.

Proton pump inhibitors such as omeprazole and sucralfate form the cornerstones for the management of gastrointestinal ulcerations and there is little evidence for the use of older alternatives such as histamine receptor blockers. Omeprazole is the most readily available proton pump inhibitor to practice and has been shown to have a superior effect on gastric acid suppression when used twice daily instead of once daily as previously advised. Finally, these drugs are not without complications and side effects and should be used prudently.

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CPD Questions AC/1935/18

Article - Gastrointestinal Therapy, Anti-emetics and Antacids

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Question 1: Which one of the phrases below regarding the vomiting mechanism is INCORRECT?
a. The CTZ relies on dopamine receptors.
b. Vestibular nausea relies on histamine receptors.
c. The VC is in the medulla within the blood brain barrier.
d. GIT stimulation of vomiting is seratonergic
e. The CTZ has a inhibitory effect on the VC

Question 2: Which one of statements regarding omeprazole is CORRECT?
a. Omeprazole is active on the gastric side of the parietal cell.
b. Omeprazole is proton pump activator.
c. Omeprazole is not effective in cats.
d. Omeprazole is most effective administered BID.
e. Omeprazole has no evidence of efficacy in dogs.

Question 3: Which one of the statements regarding anti-emetics listed below is INCORRECT?
a. Metoclopramide has mild prokinetic activity.
b. Maropitant has demonstrated efficacy in cats.
c. Maropitant relies on inhibition of substance P binding to NK receptors .
d. Maropitant has mild prokinetic activity.
e. Ondansetron blocks seratonergic transmission in the chemoreceptor trigger zone and vagal afferent nerves.

Question 4: Which one of the components of the gastric mucousal defence systems listed below is INCORRECT?
a. pre-epithelial mucous-bicarbonate barrier
b. the epithelial barrier and continuous cell renewal of epithelial cells
c.continuous mucosal blood flow
d. normal gastric through-flow and emptying
e. generation of vasodilatory prostaglandins and nitric oxide

Question 5: Which one of the statements listed below regarding gastric acid production is CORRECT?
a. Acid production is stimulated by the binding of gastrin to the proton-pump mechanism.
b. Hydrogen molecules are secreted by proton pumps on the luminal surface of the parietal cells.
c. Acetylcholine and histamine receptors are present on the luminal surface of the parietal cell.
d. H2 blockers will also block other stimulatory factors for acid production.
e. Decreased blood flow will decrease acid production.

Question 6: Which one of the statements below regarding omperazole is CORRECT?
a. Omeprazole acts by reversibly binding to the proton pump.
b. Proton pump inhibitors should be administered 30–60 minutes before feeding for optimal effect.
c. Omeprazole takes approximately 7 days for optimal suppression of acid production.
d. The efficacy of omeprazole decreases over time.
e. Once daily administration of omeprazole is sufficient for clinical benefit.

Question 7: Which one of the statements regarding chronic kidney disease (CKD) in cats is INCORRECT?
a. CKD is a common condition where antacids must be considered.
b. Recent evidence suggests that gastric ulceration is not a feature of CKD in cats.
c There is little evidence to justify the use of antacids prophylactically in cats with uraemia.
d The use of sucralfate in cats with CKD had to be discontinued prematurely due to excessive vomiting and anorexia.
e. There are less studies critically evaluating the efficacy of antacids in cats compared to dogs.

Question 8: Which of the followings statements regarding medications affecting gastric mucousal blood supply is CORRECT?
a. Misoprostil is a natural PGE (prostaglandin E)
b. PGE is vasodilatory and effective in the gastric mucousa.
c. Sucralfate blocks the synthesis of vasodilatory prostaglandins.
d. Misoprostol is valuable only in the treatment of NSAID induced ulceration.
e. Hypovolaemia and shock have no effect on gastric blood flow.

Question 9: Which one of the statements below regarding the administration of maropitant is CORRECT?
a. A single daily dose of maropitant was more effective compared to a single dose of metoclopramide.
b. Maropitant should be used with caution in patients with suspected GI obstruction.
c. Maropitant has a 24-hour duration of effect in dogs and cats.
d. The injectable dose of maropitant should be administered at least 1 hour prior to chemotherapy for optimal effect.
e. The oral dose is higher due to decreased oral bioavailability and is absprbition is decreased with the presence of food.

Question 10: Which one of the statements below regarding metoclopramide is most CORRECT?
a. Severe CNS signs are the most common side effect of metoclopramide.
b. Metoclopramide mixed with 0.9% saline is not light sensitive and remains stable for 48 hrs.
c. Metoclopramide is a serotonin antagonist.
d. Metoclopramide acts only centrally.
e. Metoclopramide is useful for the treatment of oesophagitis as it is a strong prokinetic.

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