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Vol 05 | Issue 05 | November 2018

Aspiration Pneumonia

The ABCs of Veterinary Dentistry
Periodontal Pockets

CPD Accredited Article
Feline CKD and Proteinuria

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Editor's Note

Well here is the last issue for 2018.

The article on aspiration pneumonia by Dr Vanessa McClure provides some insights into the diagnosis and management of this condition. This brought to my mind the high risk of aspiration in patients with acquired neuromuscular disease. It also opened up a small can of worms regarding what people think is the ideal management course to reduce the risk of gastro-oesophageal reflux and resultant aspiration or oesophagitis. I've summarised findings from publications since 2005 which have dealt with the subject.

Also included is a really nice short practical piece on local anaesthesia in surgery and a more contentious journal scan regarding the efficacy of Tramadol in canine patients.

I must at this time thank Madaleen and Hein, at the Vetlink offices, who always work full out at the last minute to get the magazine to print on time. I am the worst "last minute. com" person - so their nerves are usually shot!

I wish you all a safe and happy Christmas / Holiday Season

Regards

Liesel

Index:

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SAVA South African Veterinary Association Suid-Afrikaanse Veterinêre Vereniging

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AFAST^3 for You and Me
ABCs of Veterinary Dentistry: "O" is Oral Masses
Histopathology in Oncology Patients

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Madaleen Schultheiss

The Finer Points: Injecting Common Sense into your Regional Anaesthesia Strategy

By Hilal Dogan, BVSc, CCTP

I've always been afraid of doing local blocks for some reason—probably because I just never really got comfortable with them. So they usually aren't top of my mind in practice until somebody says, "We can do a local block." And I think to myself, "Well, duh! Why didn't I think of that?"

Testicular blocks

Testicular blocks will make your neuters go more smoothly and can help reduce the use of systemic drugs.

I tried this once and had so much bleeding I vowed to never do it again because I was convinced the lidocaine was making this dog bleed like a hemophiliac on speed. I ended up sending him to the emergency room for "observation." Yeah, so, I never used a testicular block again.

Then I realized I'd done it wrong. My mistake? I'd dripped the lidocaine into the incision site postneuter, happily thinking I was doing this dog a favor. It's much more effective to use the lidocaine preneuter, injecting it directly into the testicle, and not to use it after you've already taken the testicles out!

Lidocaine does cause vasodilation and can increase bleeding; however, used appropriately, it will make your neuter go much more smoothly and cause your patient to feel less to no pain. You don't have to use a long-acting agent here, such as bupivacaine—most of your local anesthetic will end up in the trash when the testicles are discarded.

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How local blocks work

Simply put, a local nerve block involves delivering a local anesthetic close to a key nerve via injection, says Dr. Mike Barletta, who discussed the pharmacology of local anesthetic drugs during his part of the Fetch dvm360 session.

To put it more precisely, local anesthetics close the sodium channel in the nerve, effectively blocking different types of nerve fibers, such as A-delta and C fibers. A-delta fibers are responsible for processing and transmitting sharp, acute, well-localized pain when activated, while C fibers are involved in slow-onset, burning, non-well-localized pain.

Local anesthetics fall into two classes—aminoamides and aminoesters. Here are some of the most commonly used:

Aminoesters	Aminoamides
Procaine	Lidocaine
Benozocaine	Etidocaine
Chlorprocaine	Prilocaine
Tetracaine	Mepivacaine
Cocaine *(yes, you read that right—Dr. Barletta says that in some countries cocaine is used for its vasoconstrictive action).*	Bupivacaine
	Levobupivacaine
	Ropivacaine

A simple way to remember the difference? If a drug name contains two i's it's most likely an aminoamide (which also contains two i's); if it has just one i it's an aminoester (only one i)—but this doesn't work in every case, so use the rule with caution.

Sacrococcygeal blocks

Sacrococcygeal blocks will make your life much easier when it comes to blocked toms, tail amputations, and basically any surgery you're doing in the urogenital and perineal regions, says the Fetch dvm360 team. According to Dr. Barletta, the beauty of this block compared with an epidural is that your patient will retain its ability to walk immediately after the procedure.

McNerney singlehandedly convinced me to try this the next time I encounter a blocked tom that proves difficult to catheterize. "Instead of wrestling with the penis until it becomes a swollen, bloody mess, just sacrococcygeal-block that baby," she says. "The penis pops right out."

She also says to Instagram-story her when that happens and she'll do a happy dance for you. It's a win-win-win situation. You win, the cat wins and McNerney wins.

What not to do

As I realized when I used lidocaine after a neuter, there are definite no-nos when it comes to local nerve blocks. Here are a couple:

1. Thou shalt not mix two anesthetic agents.
For example, don't mix bupivacaine and lidocaine in hopes of achieving a middle ground in terms of speed of onset and duration of effect. They don't work that way, Dr. Barletta says—you'll end up with an in-between onset and duration, deriving the benefits of neither. It's better to use anesthetics sequentially when necessary: first a fast-acting agent, followed by a longer-lasting one—for example, at the end of the procedure for more postoperative analgesia.

2. Thou shalt not use epinephrine mixed with a local anesthetic on terminal blood supply areas.
For example, don't use epinephrine on the tail area or ears because it causes vasoconstriction, and you may cause ischemic necrosis of the area because you cut off the blood supply for too long.

An eternal conundrum

Last but not least, I leave you with the eternal sodium bicarbonate conundrum: to mix or not to mix? The Fetch dvm360 experts say yes, it's OK to mix sodium bicarb with your anesthetic to decrease the sting of a local block. However—and this is a big however—some local anesthetics precipitate out when mixed with sodium bicarb, so it's important to know which ones! Commit them to memory—they are bupivacaine and etidocaine

Tips from the tech

In McNerney's experience, local nerve blocks are most commonly used in dentistry—specifically the caudal mandibular block and the maxillary block. Her favorite anesthetic for these blocks is bupivacaine.

Other common applications include:

- Retrobulbar blocks for enucleation
- Testicular blocks for neuters, which can cut down on the need for inhalant anesthetic (McNerney usually uses lidocaine)
- Incisional blocks—inject a local anesthetic along the incision site

Sacrococcygeal blocks for urethral obstruction in cats—using a 5/8-in needle, inject bupivacaine in the space where the tail goes up and down and wait 60 to 90 seconds.

The anesthetic will affect the tail, anus, penis and perineum. And no, pets do not poop all over afterward.

Building better business

Nerve blocks make a lot of sense for the business and management side of veterinary practice, says manager Ori Scislowicz. Here are the reasons:

- Nerve blocks require a minimal equipment investment. For most blocks, all you need are a syringe and needle, although Dr. Mike Barletta does say you can use a nerve locator and ultrasound machine to help with the process.
- Nerve blocks result in a smoother recovery for the patient and help pets maintain greater comfort throughout a procedure and the recovery period. This can set your practice apart if you educate clients about the details and even market these services more broadly.
- If you empower techs to learn how to do nerve blocks, you build positive morale on your team and allow technicians to build their patient advocacy toolbox.

All in all, Scislowicz says, you'll have happier employees who are more skilled, and you'll have happier patients that are more comfortable.

CPD ACCREDITED ARTICLE

Proteinuria

and the Progression of Chronic Kidney Disease in Cats

Liesel L van der Merwe, BVSc MMed Vet (Med) Small Animals
Senior lecturer: Outpatients

Chronic kidney disease is common in cats, with an increased prevalence in older animals, affecting 30-40 % of those over 10 years of age. The average life expectancy is 1-3 years once clinical signs become apparent. There are some congenital and some disease related causes of renal impairment in cats, but the majority appears to be a gradual age related deterioration in function.

Ureteroliths and nephrolithiasis are increasing in incidence and 98% are calcium oxalate. These typically occur in younger cats, so not part of the typical CKD, and have no association with the progression of CKD. Renal lymphoma, infections, nephrotoxic agents and systemic hypertension are all associated with decreased renal function.

Risk factors such as a general anaesthesia or documented dehydration in the previous 12 months or exposure to nephrotoxic drugs may cause undetected acute kidney injury (AKI) which may initiate or stimulate inflammation and fibrosis.

CKD is a silent disease until quite advanced. Health checks in older cats are advised for early detection. The AAHA advise bi-annual checks in cats >7yrs. History and clinical signs suggestive of clinical CKD are weight loss, dehydration, decreased kidney size, PU/PD, systemic hypertension and low USG (1.035-1.040). Clinical signs mean uraemia. Early stage 2-3 renal disease often clinically normal

In cats progression of renal disease does not seem to be characterised by a slow increase in creatinine over time but rather by abrupt increases in creatinine (uraemic episodes) after relatively long periods of stability (3 – 21 months). The median survival time after diagnosis shows considerable individual variation but is as follows: IRIS 2 - 1151 days, IRIS 3 - 778 days, IRIS 4 - 103 days.

Table 1 General Clinical Exam

BCS/ Lean body mass	Severe weight loss
Appetite	Decreased appetite
Oral examination	Ulcers, dental disease, halitosis pale mucous membranes
Renal palpation	Small / large kidneys , Contour chages, painful ?
Ocular examination	Rentinal haemorrhage, detetchment, hypertension
Auscultation	Cardiac disease , thyroid disease , HCM

Further diagnostic tests should include a urinalysis, serum biochemistry, haematology, systolic blood pressure, and ultrasonography.

The aim of the further testing for a CKD is to stage the disease according to the IRIS scoring system and to evaluate the patient for any possible causes of the decreased renal function (lymphoma, hypercalcaemia, ureteroliths etc) as well as to detect any concurrent progressors or complication of the renal disease such as hypertension , proteinuria and hyperphosphataemia.

Urinalysis

Dipstick: to exclude diabetes mellitus as a cause for the clinical signs.

SG: There is a loss of concentrating ability in CKD caused by solute diuresis from tubular overload, loss of the hypertonic medulla and impaired response to ADH. However urine concentrating ability does not correlate with GFR in cats and they can maintain SG even if mildly azotaemic.

Sediment: due to the lower SG false negative results for bacterial UTI can occur. Send for culture if you suspect this complication

UPC: UPC correlates well with 24-hour urine protein loss. Check sediment first to exclude lower urinary tract inflammation as a source of protein. In a healthy cat UPC is generally very low (UPC <0.1). In health, filtered proteins reabsorbed in the proximal convoluted tubule by endocytosis so proteinuria is generally low. The UPC must be a repeatable finding.

A UPC > 0.4 = proteinuric, UPC 0.2 – 0.4 = borderline proteinuric and a UPC <0.2 = non-proteinuric. Proteinuria occurs due to hyperfiltration, glomerular hypertension and altered glomerular permselectivity.

Endogenous markers for GFR

Creatinine

Creatinine is specific marker for the kidney. It is affected by the muscle mass of the patient and is positively correlated to increased muscle proportion body mass and negatively correlated with age *(very young and old)*. The sensitivity of creatinine is low and it will only pick up renal disease when GFR is <75% of normal.

With creatinine interpretation patient normals are much more reliable than population based ranges. Creatinine shows very little individual variation in healthy adult animals and any trends on patient creatinine are very sensitive. Changes within the normal ranges may already be an indicator of a decrease in GFR.

Urea

Changes in urea are not very specific for renal disease as GIT haemorrhage and haemolysis can cause elevation. It is not very sensitive either as it parallels creatinine with increases only at about 75% renal damage.

Symmetric methylarginine (SDMA)

SDMA is an endogenous protein, formed by the methylation of arginine, which is released into the blood and of which ≥ 90% is excreted by the kidneys and doesn't undergo any biotransformation. It is an accurate and precise assay for dogs and cats and on average increases with only 40% loss in renal tissue.

MAKING A DIAGNOSIS OF CKD IN CATS:

A diagnosis of CKD is made on an increase in serum creatinine >140umol/L together with an inappropriately low SG (<1.035) and evidence of sustained changes for at least 3months.

However:

- some healthy cats on wet food may have a low SG
- some early CKD cats can still concentrate to SG 1.035
- a 15% increase on **any** previous creatinine is significant
- CKD with renal origin proteinuria and no azotaemia in the early stages does occur.

Systemic Hypertension

Systemic hypertension occurs in 20% of cats diagnosed with CKD and about 60% of cats with hypertension have azotaemic CKD. There is a positive association between age and increasing blood pressure in cats and there is also an increased risk of hypertension in cats with CKD compared to healthy cats. Blood pressure measurement should be part of the health check in all older cats. Systemic hypertension may exhibit ocular, cardiac renal and central nervous system effects.

Table 2 Normal IRIS stages of SBP in cats

Normal	SBP < 150 mmHg
Stage 1	SBP 150 – 160 mmHg
Stage 2	SBP 160 – 179 mmHg
Stage 3	SBP ≥ 180 mmHg

The maladaptive renal response to injury is the cause of progression of CKD: haemodynamic adaptations, activation of RAAS, systemic hypertension, proteinuria, hypoxia and secondary hyperparathyroidism

The kidney tries to maintain GFR by increasing SNGFR (single nephron GFR) but this causes glomerular hypertrophy, hypertension and hyperfiltration, and is ultimately detrimental as it causes self-perpetuating nephron loss.

RAAS activation

Systemic and tissue specific RAAS exist. The kidney has all components for RAAS. Renal Angiotensin II concentrations are higher than plasma concentrations, thus what happens in the kidney is not represented in the plasma.

Angiotension II

- is a potent vasoconstrictor of the EFFERENT arteriole - thus causing glomerular hypertension and hyperfiltration and damage.
- Reduces renal oxygenation due to vasconcostriction of the efferent arteriole, which still needs to perfuse the remainder of the nephron.
- Modulates permeability of glomerulus (podocytes) promoting proteinuria
- Has a direct fibroproliferative and inflammatory effect causing glomerulosclerosis
- Stimulates aldosterone production by adrenals and aldosterone is pro-fibrotic

Chronic RAAS-activation has several harmful effects in the kidneys such as glomerular hypertension, fibrosis and cell infiltration, higher glomerular permeability and oxidative stress, all of which cause progression of CKD.

Tubulointerstitial infiltration of lymphocytes and macrophages is an early feature of CKD and may be induced by proteinuria, reactive oxygen species generation and up regulation of angiotensin II.

Regardless of the initial cause, chronic renal inflammation is believed to play a critical role in the pathophysiology of CKD and the perpetuation of renal fibrosis.

Renal hypoxia

Blood flow follows the afferent arteriole, into the glomerular capillaries and then into the efferent arteriole and on to the peritubular capillary complex for tubular cells. Glomerulosclerosis may decrease down–the-line blood supply and cause tubular hypoxia. RAAS activated vasoconstriction of efferent arteriole will also cause tubular hypoxia. Oxygen delivery by diffusion will also be interrupted by fibrosis and inflammation.

Proteinuria

Marked proteinuria is uncommon in the cat with CKD. Almost all CKD cats (90%) have a UPC of <1, half have a UPC of <0.2 and only 20% are overtly proteinuric (UPC >0.4). Proteinuria is a negative prognostic marker and increases the relative risk for death: three-fold increase with UPC 0.2-0.4, four-fold increase with UPC > 0.4. Another way of looking at it is to correlate the median UPC with the IRIS staging of the CKD; IRIS stage 2 has a median UPC of 0,15; IRIS Stage 3 of 0,22 and IRIS stage 4 of 0,65.

A paper by Syme *et al* (JVIM 2006) showed that hypertension at initial presentation was not associated with the development of azotaemia or survival in cats with CKD. All the cats in the trial were on treatment with amlodipine , which is very effective at controlling hypertension and follow up SBP measurements were in the acceptable range. The take home message should be that cats with CKD and controlled hypertension do not have poorer survival than those without hypertension. This study also showed that increased urinary protein excretion is a very good predictor of reduced survival in cats with CKD.

Proteinuria is a significant predictor of future azotaemia and is also significantly associated with the severity of inflammation, fibrosis and tubular damage on renal histopathology.

Hyperphosphataemia

Hyperphosphataemia is present in 60% of cats with CKD and is a trigger for secondary renal hyperparathyroidism and nephrocalcinosis. Hyperphosphataemia contributes to clinical signs of disease, progression of disease and restriction is recommended in ALL IRIS stages. Phosphate restriction is a key reason why renal diets are effective.

Factors found to be associated with decreased survival time in Feline CKD patients:

- High plasma creatinine
- High plasma phosphate
- High plasma urea
- High blood leukocyte counts
- Low blood haemoglobin and haematocrit
- Uraemic patients - IRIS stage 4
- High UPC and BP at any IRIS stage

TREATMENT

Tailor treatment to the IRIS stage of disease:

- IRIS stage 1 – identify underlying disease process and apply steps to eliminate this disease
- IRIS stage 2-3 – Address factors leading to progression of CKD – appropriate nutrition, control proteinuria, hypertension and hyperparathyroidism.
- IRIS stage 3-4 – also give supportive therapy, dehydration , acidosis, anaemia, hypokalaemia

Renal diet

Feeding a renal diet provides the most positive long term effect on outcome of CKD. The diets are protein restricted, phosphate restricted, sodium restricted and supplemented with Omega 3 fatty acids, potassium and vitamin B and containing blood alkalinising agents. Two separate studies showed survival times of 20.8 months vs 8.7 and 16 months vs 7 months in cats on renal diet and on normal maintenance diets. Uraemic episodes were significantly minimised.

The studies also showed a reduced morbidity and mortality rate in the renal diet group over the test period with 0% uraemic episodes vs 26% in the maintenance diet group and 0% mortality vs 22% in the maintenance diet group. Increased survival is presumed to be due to the attenuation of the severity of the secondary renal hyperparathyroidism as well as lessening the severity of uraemia.

Early intervention is recommended when creatinine is >150umol/L. Acceptance of the diet increases prior to the development of clinical signs as there is less nausea.

Blood pressure

Start treatment when SBP is greater than 160 – 170mmHG or if evidence of hypertensive retinopathy (haemorrhage/detachment) is present. Only start treatment when the CKD is stable. Amlodipine at 0.25-0.5mg/kg oid is the recommended treatment. ACE inhibitors are ineffective for systemic hypertension as they drop SBP by only 10 – 15 mmHg. Telmisartan at 1-3mg/kg oid or divided bid significantly decreased SBP in normal cats (130 to 105/90 mmHg) within the second week of treatment.

Proteinuria

The major detrimental renal effects of angiotensin II are mediated by AT1 receptors: vasoconstriction, increased systolic blood pressure, cell proliferation and fibrosis and proteinuria. AT2 receptors modulate renoprotective actions of angiotensin II such as vasodilation, natriuresis, inhibition of renin secretion and anti-inflammatory, anti-ischaemic and anti-fibrotic effects.

ACE inhibitors: ACE inhibitors prevent the conversion of angiotensin I to angiotensin II. Thus they will decrease GFR and increase RBF. In mammals however, alternative pathways (ACE ESCAPE) exist for angiotensin II generation. In a trial using benazepril at 0.5mg/kg -1mg/kg per day on cats with naturally occurring CKD compared to a placebo in 61 cats over 180 days benazepril significantly reduced proteinuria. There was also a non-significant trend for a lower creatinine at the endpoint in the treatment group as well as a higher quality of life score. Non- progression of the CKD IRIS stage excluding stage 4 patients, was 93% in the benazepril group and 73% in the placebo group (p =.196). The study was underpowered , too few animals and treated for too short a period and differences in survival time which were no significant in the current study may have gained significance

ARBs: Semintra is licensed for reduction of proteinuria associated with CKD. It selectively blocks the Angiotensin II 1 receptor and spares the beneficial Angiotensin II 2 receptor. There is also no ACE escape. A study comparing 240 client owned cats treated with either angiotensin converting enzyme inhibitor (ACEi) benazepril or angiotensin receptor blocker (ARB) telmisartan (Semintra®) was significantly superior to ACEi in decreasing proteinuria.

With any drug affecting the RAAS system a 5-7 d post treatment check is essential to evaluate for the presence of azotaemia and serum potassium which may increase due to a decreased GFR.

Supportive Therapy IRIS Stage 3-4

In advanced CKD treatment need to be aimed at managing hydration, weight loss/nausea, hypokalaemia, any urinary tract infections, and anaemia.

Dehydration progresses CKD. Cats in uraemic episodes need IV fluid administration: aim for euvolaemia and resolution of clinical signs and NOT for decreasing creatinine. Encourage water intake using water fountains and using a broth (water from poached fish/chicken). If this doesn't work the subcutaneous fluids is a possible next step: 50 – 100ml Ringers lactate SQ oid.

Appetite

Manage nausea, which will increase food and water intake. Maropitant – 0.5mg/kg oid, Ondansetron, Mirtazepine (0.5mg/kg q 48 to ¼ tab q 72). Uraemic gastropathy and acidity is not a feature of CKD in cats so antacids and ulsanic are not indicated. Constipation is a common problem in cats with CKD and can also add to the nausea and inappetance. Fluid balance (supplementation) and hypokalaemia need to be addressed in these patients. Check for periodontal and dental disease. Rather risk a general anaesthetic to fix the mouth, this will then result in improved appetite as there is less oral pain.

Appetite stimulants are also indicated in these patients as all possible is being done to manage the underlying cause. Mirtazepine can be used at 3.75mg (1/4 of a 15mg tab) every 3rd day or Cyproheptadine 2-4 mg oid.

Another option is the placement of an oesophagostomy tube. This can be left in long term and both nutritional and fluid requirements can be met using this route. The cat and the owner learn to tolerate and manage this process without much difficulty.

Hypokalaemia

Hypokalaemia is seen in about 25% of cats with CKD. A severe decrease causes generalised muscle weakness and ventro-flexion of neck whereas milder decreases cause lethargy, weakness and anorexia from ileus. Hypokalaemia can also adversely affect renal function. Renal diets are supplemented. Additional supplementation can be given as required using Plenish K, Slow K or Kaligel

Anaemia

Chronic anaemia is inevitable with the progression of CKD. In the short term minimise blood sample collection volumes and consider blood transfusions, in the long term treatment with EPO can be considered.

Urinary Tract infections

Although UTIs are uncommon in cats in general, there is an increased incidence in cats with CKD due to decreased defence mechanisms (lower SG). Culture is needed to identify infection in many cases as the sediment may not be overtly inflammatory.

MONITORING

Monitoring is essential as you need to pick up new problems and intervene early. Maintain communications with owner, they often need to just bounce details on the why and how of their daily regimen. Monthly evaluation for a clinical examination, weight check and evaluation for dehydration or constipation is important. Once the blood pressure is stable it only needs to be checked every 3 months. If the cat is stable it will only need blood chemistry and a full urinalysis every 6 months.

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CPD Questions

Proteinuria and the progression of chronic kidney disease in cats AC/2024/18

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Question 1
Which of the following statements is correct?
a. Chronic kidney disease is uncommon in cats.
b. CKD has an increased prevalence in middle aged animals
c. CKD affects affecting 30-40 % of cats over 10 years of age.
d. The average life expectancy is 6 months once clinical signs become apparent
e. CKD in cats is a rapid age related deterioration in function.

Question 2
Which one of the factors mentioned below is NOT suggestive of clinical CKD?
a. Weight loss
b. Dehydration
c. Decreased kidney size, PU/PD,
d. Systemic hypertension and
e. Increased SG (SG .1.040)

Question 3
Which phrase regarding progession of renal disease in cats is INCORRECT ?
a. Abrupt increases in creatinine
b. Uraemic episodes
c. Relatively long periods of stability (3 – 21 months)
d. Considerable individual variation
e. Gradual decline in GFR

Question 4
Which statement regarding urinalysis is correct?
a. Urine concentrating ability does not correlate with GFR in cats
b. Cats cannot maintain SG even if mildly azotaemic.
c. UPC doesn't correlate well with 24-hour urine protein loss
d. An active urine sediment will not affect UPC
e. The UPC is generally >0.4 in healthy cats

Question 5
Which of one of the following statements regarding creatinine is INCORRECT?
a. Creatinine is specific for kidney function.
b. Creatinine is a measure of GFR
c. Interpretation of patient normals are much more reliable than population based "normal" ranges.
d. Creatinine shows very little individual variation in healthy adult animals
e. Trends on patient creatinine are not very sensitive

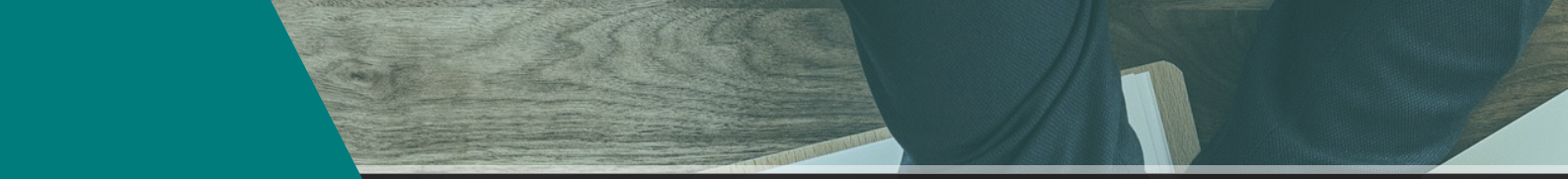
Question 6
Which one of the following findings is NOT a factor in making a diagnosis of CKD in cats?
a. A diagnosis of CKD is made on an increase in serum creatinine >140umol/L
b. A SG of >1.035 precludes a diagnosis of CKD
c. Some healthy cats on wet food may have a low SG
d. Some early CKD cats can still concentrate to 1.035
e. CKD can present with renal origin proteinuria and no azotaemia

Question 7
Which of the following statements regarding angiotensin II is INCORRECT?
a. Concentrations of angiotensin II are higher in the kidney than systemic circulation
b. Systemic and tissue specific RAAS exist.
c. Angiotension II is a potent vasoconstrictor of the EFFERENT arteriole
d. Vasoconstriction of the afferent arteriole causes glomerular hyperfiltration
e. Vasoconstriction of the efferent arteriole causes glomerular hypertension

Question 8
Which of the following statements regarding UPC is CORRECT?
a. Marked proteinuria is uncommon in the cat with CKD.
b. Almost all CKD cats (90%) have a UPC of <1,
c. Half have a UPC of <0.2 and
d. 50% of cats are overtly proteinuric (UPC >0.4).
e. Proteinuria is a negative prognostic marker

Question 9
Which one of the statements listed below is INCORRECT?
a. Cats with hypertension at diagnosis of CKD have a decreased survival rate
b. Amlodipine is an effective treatment for hypertension in cats
c. 60% of cats with hypertension have azotaemic CKD
d. Hypertension is common in cats with CKD
e. Systemic hypertension occurs in 20% of cats diagnosed with CKD

Question 10
Which one of the statements listed regarding supportive therapy for CKD is INCORRECT ?
a. Dehydration progresses CKD.
b. Cats in uraemic episodes need IV fluid administration and rapid volume resuscitation
c. Administer SQ fluids at 50 – 100ml oid
d. Uraemic gastropathy and acidity is not a feature of CKD in cat
e. Manage nausea, which will increase food and water intake.



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The Truth About Antihistamine use in the Allergic Dog

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Zoetis Petcare, Zoetis Inc

Antihistamine therapy is recommended and prescribed frequently for the control of pruritus. While the use of antihistamines may be well-intentioned, not only is the recommendation misleading (because they are largely ineffective), but it also has a potentially significant negative impact on the skin disease, the care of the patient, the owner's perception of the care their pet is receiving and the veterinarian-client-patient bond. To illustrate the impact of chronic allergies on the human-animal bond, one study reported 80 percent of owners felt "sad" about their dog's skin disease.[1]

Do antihistamines effectively control allergic itch?

NO. The recently published guidelines[2] for treatment of canine atopic dermatitis from the International Committee on Allergic Diseases of Animals (ICADA) list antihistamines under "Interventions likely to be of little or no benefit to treat acute flares of canine atopic dermatitis (AD)." Four double-blinded placebo controlled studies conclude a limited or lack of efficacy.[3,4,5,6] Additionally, a clinical pharmacology study,[7] a retrospective study,[8] a prospective clinical study,[9] a single-blinded placebo controlled study[10] and review papers[11,12] conclude a similar poor response to antihistamines. And yet, antihistamines account for almost 10 percent of all treatments dispensed for pruritus by veterinarians;[13] plus, this does not take into account treatment initiated by owners with over the counter (OTC) antihistamines.

Do they treat a major inflammatory mediator in atopic dermatitis?

NO. Previously, we thought atopic dermatitis was a type-1 hypersensitivity response in which high levels of IgE, mast cell degranulation and histamine release were central to the disease process. We now know this disease is much more complex, involving many other cells in the immune system that release proinflammatory and pruritogenic cytokines, such as cytokines IL-2, -4, -6, -13 and -31.[14] None of these cytokines are regulated by antihistamines.

Antihistamines work well in humans to manage upper respiratory allergies. This is not, however, what we are treating in dogs. Atopic dermatitis is more like human atopic eczema, which is not treated with antihistamines.

Will antihistamines work in at least some patients?

POSSIBLY. Antihistamines may provide a small and limited benefit in some dogs.[2] It is not possible to know if this minority of patients is responding to antihistamines or whether this represents a placebo effect. Regardless, response to antihistamines is the exception rather than the rule.

What does this mean?

Antihistamines will provide no benefit for the vast

majority of allergic dogs. Few, if any, owners would find that acceptable for their itchy dog, especially as other safe and efficacious treatments are available.

Are antihistamines a good first choice for dogs that have early, mild disease?

NO. The ability of the veterinarian to manage itch quickly and effectively the first time a dog is presented with allergies has important consequences for follow-up thereafter. If early interventions fail, disease continues, increasing the likelihood for infections to take hold that will require antimicrobial therapy systemically and topically.

Why put patients at risk for the additional burden of this common complication? Many allergic dogs have progressive, life-long disease — 7 out of 10 dogs with acute pruritus eventually require long-term seasonal/chronic treatment.[15] Failing to provide effective relief the first time likely causes mistrust and lack of confidence, which decreases the likelihood for acceptance of later recommendations. An op-ed article by Marty Becker, DVM, in Veterinary Practice News addresses the repercussions associated with the failure of antihistamines to relieve pruritus and includes:[16]

- Cheapening the perception of veterinary medicine
- Damage to the pet, pet owner, practitioner, practice and profession by not recommending the best FDA-approved veterinary pharmaceutical
- Pet suffers unnecessarily
- Pet owner is saddened by their dog's continued misery
- Loss of faith in the practice as a problem solver
- Practice loses present and future income
- The profession is tarnished

Are antihistamines inexpensive?

NO. While the cost of the actual tablets or capsules may be low, the cost of failure (e.g., secondary infections, loss of confidence in the veterinarian, seeking a second opinion, loss of future business and revenue) far exceeds the cost of effective therapies.

Is it true that antihistamines do no harm?

NO. In a comprehensive review,[12] adverse effects of antihistamines in dogs were summarized as the following: "Possible adverse effects listed in textbooks and non-refereed meeting notes include sedation, anticholinergic effects, trembling, ataxia, hyperesthesia, hypersalivation, increased pruritus, panting, and excitation. In addition, cardiac effects of concern in humans (such as prolonged QT interval) with non-sedating antihistamines also occur in dogs; in fact, the dog has been used as an animal model to study such effects." This would not qualify antihistamines as harmless.

Aren't antihistamines more convenient than other options because they are readily available OTC?

NO. When dogs have pruritus, they should always be evaluated by the veterinarian to determine the underlying cause, such as allergies, infections or parasites. The danger of OTC therapies — wrong formula (contains pseudoephedrine), wrong size, wrong dose, wrong dosing interval — can lead to failure or worse, including adverse reactions. Placing treatment decisions for allergic dogs in the hands of owners without veterinary care is very unlikely to result in satisfactory outcomes. By suggesting OTC medications, veterinarians set owners up for failure and disappointment.

Is there any place for antihistamines in atopic dermatitis?

POSSIBLY — IN COMBINATION WITH STEROIDS. The ICADA guidelines[2] state: "Interventions are likely to be of little or no benefit to treat chronic canine AD: Type 1 antihistamines have modest efficacy against pruritus, either alone or in combination with each other, but their effect appears to be variable between individuals. For optimal efficacy, this class of drugs is best used as preventatives before a flare occurs — not during or after it — and they should preferably be given on a continuous daily basis."

Thus, at best, antihistamines may be helpful when used chronically long-term and continuously to prevent possible disease flares. This has not been studied, and it would seem extremely unlikely that owners would continue oral therapy multiple times a day continuously. One study found that the dose of steroids used to control pruritus could be reduced by 30 percent in 75 percent of dogs when a combination of trimeprazine and prednisolone was administered compared to steroids alone.[17]

What is the cost of treatment failure?

ENORMOUS. One study showed the factors having the most impact on pet owners of dogs with skin disease were emotional distress, physical exhaustion, expenditure, and time loss.[18] Client comments express the damage that chronic allergies have on the human-animal bond: "Hopeless," "Frustrating," "I feel like a bad mom," "Stinky," "Sleepless," "Expensive," "He hates the e-collar" and "It's sad that my 4-month-old daughter sleeps longer through the night than my dog." Why prescribe antihistamines that will contribute to these feelings? Dr. Becker states, "Human doctors prescribe the best pharmaceuticals and health care products available regardless of price. Not only does this typically offer the best chance of an effective treatment or cure, it keeps the human patient looking to the family doctor for health care solutions. The patient skips Dr. Google and aisle upon aisle or page

upon page of OTC products (or worse, unproven holistic products and snake oil)." [16]

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Aspiration Pneumonia

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Aspiration pneumonia (AP) is a common diagnosis in canine patients but rarely seen in feline patients. It occurs when a patient inhales oral and/or stomach fluid into the respiratory tract. This process can vary from a mild inflammatory response to a more severe disease, depending on the composition and volume of the aspirate. Low pH and high volume aspirates, as well as the presence of food particles in the aspirates, intensifies the severity of the inflammatory response.

Aspiration first results in a sterile pneumonitis, where the damage to the airways and pulmonary parenchyma is a direct result of the aspirated fluid. This tissue damage triggers an inflammatory response, resulting in necrosis of type I alveolar cells, bronchiolar constriction, pulmonary haemorrhage, increased mucus production, alterations in mucociliary action and increased vascular permeability, causing oedema, and finally alveolar collapse and atelectasis.

The injury to the epithelium in turn, enhances bacterial adhesion, and the decrease in the mucociliary activity, along with the airway collapse causes entrapment of bacteria and subsequently infection, resulting in aspiration pneumonia. The inflammatory response in the affected lung lobe(s) can continue to progress and cause inflammation and oedema in other lung lobes which were not initially affected, further compromising lung function. Most affected patients do recover with supportive medical management, but the mortality rates can be up to 25% in severe cases.

Diagnosis

It is difficult to make a definitive diagnosis of aspiration pneumonia due to a lack of specific underlying features and the fact that the aspiration events are seldom witnessed (<1% of the time). A presumptive diagnosis is usually based on a combination of history (which may include a predisposing condition/event), physical examination findings, clinical signs, and radiographic findings.

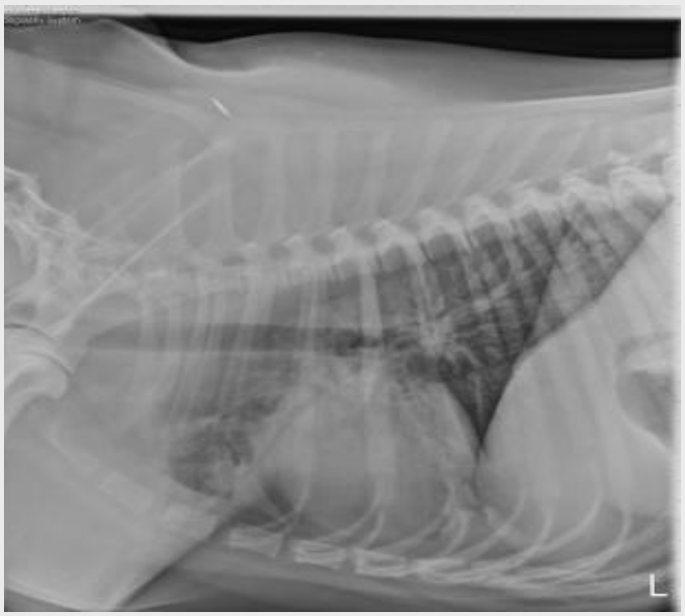

Fig 1 a: Lateral radiograph showing typical presentation of aspiration pneumonia.

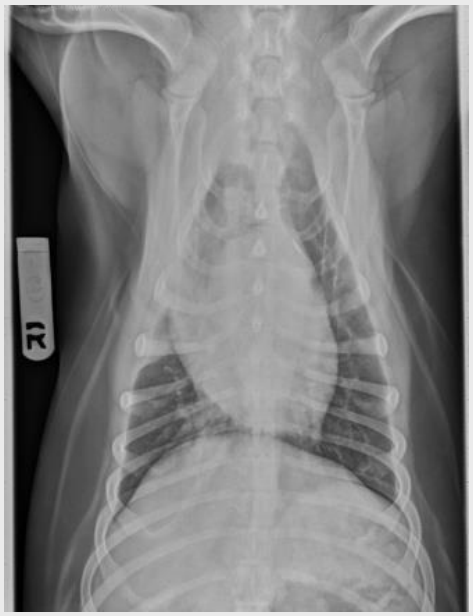

Fig 1 b: DV Thoracic view showing opacification of the right middle lung lobe.

Table 1: Conditions predisposing to aspiration of gastric content

Large volumes of intragastric content	Oesophageal disorders	Impairment of protective airway reflexes	Impaired consciousness	Others
Delayed gastric emptying	Oesophageal obstruction	Airway trauma	Sedation/ general anaesthesia	Nasogastric/ gastric intubation
Pyloric outflow obstruction / Bowel obstruction	Oesophageal dysmotility	Laryngeal or pharyngeal dysfunction	Head trauma	Foreign body
Gastrointestinal motility disorders	Megaoesophagus (various causes)		Seizures	Tracheostomy
Ileus	Reflux oesophagitis		Encephalopathy	Cleft palate
Pain	Achalasia		Coma	Weakness, paresis, paralysis*
Opioid administration	Gastroesophageal sphincter incompetence			Metabolic derangements
Recent consumption of a meal				
Anxiety/ pregnancy/obesity				

Physical examination findings that may be found in patients with AP are lethargy, tachypnoea, dyspnoea, fever, coughing and abnormal lung sounds on thoracic auscultation. However, in a study looking at 125 dogs with aspiration pneumonia, only half of the dogs presented with tachypnoea and fever and less than half of the dogs had a cough and /or abnormal lung sounds.

Further diagnostic tests such as haematology may be helpful as it is an infectious disease so the white blood cell count is expected to be increased with a possible left shift. But not all patients with AP will have these changes as the patient's immune response, the phase of infection and the severity of the infection will alter the white blood cell concentration. No significant findings are usually noted on regular biochemistry.

C-reactive protein (CRP) can be used as a biomarker. A study found that dogs with bacterial pneumonia had significantly higher CRP concentrations compared to dogs with other respiratory diseases. In this study they found that they could rule out bacterial pneumonia in dogs with clinical signs lasting > 24 hours with a CRP of <20mg/L. If the CRP was found to be > 100mg/L with clinical signs that had lasted for >24 hours, a diagnosis of bacterial pneumonia was very likely. Thoracic radiographs should be performed in all cases suspected of having AP. Ideally three views should be taken. In early cases, changes may be interstitial in nature, but the most common findings will be alveolar infiltrate, in the right middle lung lobe. The next most commonly affected lung lobes are the right or left cranial lung lobes, while in more severe cases all lung lobes can be affected. Bronchoalveolar lavage (BAL) or transtracheal wash (TTW) findings may also aid in diagnosis and enable a sample to be obtained for culture and sensitivity, but in severely compromised patients it is not always possible to perform either of these procedures safely.

Several risk factors for aspiration pneumonia have been identified (Table1). These risk factors vary between species, but general anaesthesia, vomiting disorders and megaoesophagus are the most common causes in dogs. In a study looking at the risk factor in dogs, it was shown that if a patient had 2 or more risk factors, they were at a significantly higher risk of developing aspiration pneumonia. By recognising these risk factors, and making an effort to manage or minimise them, the incidence of aspiration pneumonia can be reduced.

Patients with gastrointestinal disease should be treated with prokinetics and antiemetics to help reduce the risk of aspiration of gastric content. Puppies with parvo virus tend to be at a greater risk of AP due to the severe ileus the disease causes. These patients need

to be fed and often have naso-oesophageal tubes placed, but due to the ileus and gastric stasis, the food accumulates in the stomach and increases their risk of vomiting and aspirating.

This risk can be reduced by doing intermittent gastric suction of the stomach content, by either passing a stomach tube intermittently or by placing a nasogastric feeding tube and suctioning out the residual gastric content before feeding. The nasogastric tube can also then be used for enteral feeding. Placing nasogastric tubes as apposed to naso-oesophageal tubes was thought to increase the risk of regurgitation, but a study published in the journal of Veterinary Emergency and Critical Care found that there was no significant differences in adverse occurrences between the groups with naso-oesophageal tubes and those with nasogastric tubes. In people, reinfusing some of the aspirated gastric fluid has been recommended to avoid the development of hyperchloremic metabolic acidosis, however In a small population of dogs and cats that underwent intermittent suction over a 36 hour period none developed hyperchloremic metabolic acidosis.

AP is a well known sequel to general anaesthesia (GA) in dogs. The longer the time under GA, and the more times a patients position is changed are some of the factors that lead to an increase risk for that patient aspirating. Trying to reduce the time under GA along with good presurgical planning could help with these factors.

Prolonged food restriction before GA has also been thought to aid in reducing the risk of AP but this has been brought into question by four different studies in dogs where the impact of food withholding on gastroesophageal reflux was reported. The conclusion was that a short (2 to 4 hour) period of food withholding as well as feeding a smaller portion of food with high moisture content (canned dog food) appeared to reduce the risk of gastroesophageal reflux. The optimal duration of withholding water was also evaluated, and determined to be four hours.

Treatment

Treatment for aspiration pneumonia revolves around treating the underlying cause if possible, administration of intravenous fluids, antibiotics and oxygen therapy along with other supportive care.

Antimicrobial Treatment

Ideally, antimicrobial selection should be based on the results of the culture and sensitivity, but it is not always possible to get samples due to financial limitations or patient instability. In these cases broad-spectrum empirical antibiotics should be chosen using the recommendations made by the International Society for Companion Animal Infectious Disease Working Group on respiratory tract diseases. The group recommends the use of ampicillin or a first generation cephalosporin in patients with aspiration pneumonia and no evidence of sepsis. They also suggest clindamycin could be used for gram-positive and anaerobic coverage, as its lipophilic nature may afford better tissue penetration. When there is evidence of a more life-threatening infection then the recommendations are to add a fluroquinolone and if the animal has a history of being from a shelter or boarding facility and presents with a very mild pneumonia with no systemic signs, mycoplasma could be the underlying infectious agent and doxycycline may be used.

When the following factors are present, then obtaining a sample for culture and sensitivity is strongly advised as they are likely to be associated with resistant bacteria:

1. The patient has been on antibiotics within the last four months
2. The aspiration pneumonia is a hospital acquired infection
3. The patient has recurrent bouts of AP

The duration of treatment of dogs with AP has also been questioned as the traditional recommendations have been to continue administration of antimicrobials for at least 3-4 weeks, or 1-2 weeks after radiographic resolution. There appears to be no evidence however to support this duration of treatment in either cats or dogs. In the human literature the recommendations are to use antimicrobial therapy for 7 to 10 days and only extend the therapy for 14 days or longer for patients who are immunocompromised or those with unusual infections They also do not advise using the appearance of thoracic radiographs to guide the duration of antibiotic therapy.

Specifically, the guidelines recommend "treatment until 72 hours after the patient becomes afebrile and until clinically stable. With this in mind the consensus opinion of the Working Group is that shorter courses of appropriate treatment, might be effective in some veterinary situations as well. They recommend that patients be re-evaluated no later than 10–14 days after starting treatment, and then the decisions to extend treatment should be made based on clinical, haematological, and radiographic findings. The use of CRP may be helpful in decision making at this stage as well.

Fluid Therapy

Fluids are important to replace fluid losses and maintain hydration. Dehydration causes the respiratory secretions to become more "sticky" due to loss of fluids from the aqueous layer of these secretions. This in turn impairs mucociliary clearance. On the other hand, over hydration can be detrimental to the patient, as it will increase pulmonary interstitial and alveolar fluid accumulation and thereby exacerbate hypoxemia. There are no guidelines as to the use of

Table 2: Monitoring for pulmonary oedema whilst on fluid therapy

Exercise intolerance Tachypnoea Referred breath sounds	Inspiration is active, expiration is passive based on the elasticity of pulmonary tissue. Thus increased effort of expiration is an early sign of interstitial fluid accumulation. Referred breath sounds occur when the sound of normal air turbulence in the large bronchi is carried to the lung periphery due to interstitial fluid accumulation (sound travels better through a fluid than air).
Respiratory distress Crackles on auscultation Moist soft cough Cyanosis	Fluid is now also building up in the alveoli - causing crackles.

IV fluids in AP patients. Each patient will be different. Providing balanced fluid support is important, with each patient being monitored closely and the fluid rates adapted appropriately. (Table 2)

Oxygen Therapy

Oxygen therapy is indicated if the patient is hypoxaemic or severely dyspnoeic. The assessment of a patients oxygenation can be done using arterial blood gas or pulse oximetry, with a PaO2 < 70mm Hg or an SPO2 <93% indicating hypoxaemia and the need for oxygen supplementation. If you do not have access to blood gas or pulse oximetry, use the clinical signs to guide you. In my opinion, if you are not sure if the patient needs oxygen, just supplement it, the only contraindication for giving oxygen is if the patient "is on fire"!!

Nebulization

Nebulization with 0.9% saline and coupage is used by most veterinarians with the thought that the this will allow the delivery of small drops of water into the respiratory tract to increase the hydration of the aqueous layer of the respiratory secretions, which will enhance the movement of theses secretions by the mucociliary apparatus. Despite the common use of nebulization and coupage, there is no evidence in veterinary medicine to support its use, in fact one veterinary study found no statistically significant links between nebulization treatments and survival to discharge and coupage has been associated with pulmonary atelectasis and gastroesophageal reflux in dogs. In human studies there is some evidence that suggests nebulizing with 3% hypertonic saline was more effective than normal saline. But even in the human studies the results are conflicting with many studies showing no clear benefit for the use of nebulization. With this in mind, and given the time and effort that goes into performing nebulization and coupage, the question is should we still be doing it? This should be left up to the clinician, to decide, but If nebulization is going to be used rather use hypertonic saline.

Conclusion

Aspiration pneumonia is common in canine veterinary patients and can have high morbidity and mortality rates if not treated. Apiration pneumonia can be prevented by identifying high risk patients and procedures and reducing risk factors where possible. High risk patients should be monitored closely to allow for early diagnosis and treatment when aspiration does occur. Survival rates of 77 – 82% have been reported in patients treated for AP so the prognosis is fair to good for patients treated correctly.

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Peripheral Neuropathy and Aspiration Pneumonia

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A predisposition to aspiration only occurs in patients with any neuromuscular disease which impairs unconscious protection of the airways such as laryngeal paralysis, myasthenia gravis, polyradiculoneuritis, organophosphate poisoning and neurotoxic snake bites .

The neuromuscular junction is a synapse connecting peripheral motor nerves to skeletal/striated muscle fibres. The neurotransmitter is acetylcholine (ACh) and the ACh receptors on the muscle membrane are nicotinic. Calcium is required for effective neurotransmission. ACh esterase is also required to remove and recycle the ACh from the receptor to prevent overstimulation.

- Botulsim is a disruption with the release of ACh vesicles, myaesthenia gravis or MG-like syndrome is from antigens to the post- synaptic receptors which blocks them, and weakness and paralysis in organophosphate poisoning occurs due to overstimulation and down-regulation of nicotinic ACh receptors due to toxin binding to cholinesterase.

Interference with any aspect of impulse transmission across this synapse leads to weakness with several possible clinical manifestations:

Acute flaccid tetraparesis: botulism, neurotoxic snake envenomation, fulminant myaesthenia gravis, and tick paralysis. These cases present with weakness and hypotonia, reduced or absent segmental spinal reflexes and muscle atrophy. Cranial nerve deficits may also present as dysphonia, dysphagia inspiratory stridor, megaoesophagus and reduced or absent gag and palpebral reflexes. Acute polyradiculoneuritis is a major differentia diagnosis. Voluntary tail movement and anal tone are often preserved despite the absence of voluntary movement anywhere else in the body.

Episodic weakness exacerbated by activity: Myaesthenia gravis. Also consider metabolic disease (hypothyroidism, addisons disease), cardiac disease, polyneuropathies and myopathies.

Megaoesophagus

With idiopathic/congenital megaoesophagus a defect in the afferent neural pathway responsive to oesophageal distension is suspected as the underlying cause. Acquired secondary megaoesophagus may result from many diseases affecting the neuromuscular function.

In the dog the upper oesophageal sphincter (cricopharyngeus muscles) and the entire body of the oesophagus is made up of striated muscle and the lower oesophageal sphincter is smooth muscle. In the cat the last third of the oesophagus is smooth muscle.

Idiopathic megaoesophagus is easily visualised radiographically as the oesophageal dilation is severe and obvious. With acquired oesophageal dysfunction there is often very little, to no oesophageal distension visible radiographically. However these are the patients that are MOST prone to aspiration pneumonia. **The reason for this is that all the small muscles controlling laryngeal and pharyngeal function are striated, thus they will be affected by anything affecting the neuromuscular junction.**

Often not obviously dysphagic, these dogs can aspirate if force fed or allowed to eat *ad lib* as the muscles tire easily and their function can become choppy and asynchronous.

I advise withholding food until you are sure the patient can eat/swallow properly using water as a test medium. In cases with neurotoxic snakebite paralysis I withhold food for the day or two required for recovery. In cases poisoned with organophosphates be very sure that there is no problem with swallowing before treating with activated charcoal. If there is any doubt – place a naso-oesophageal tube. Additionally, don't use large volumes of fluid/food laced with activated charcoal as the upper oesophageal sphincter function is compromised and reflux may occur.

Source: Textbook of Veterinary Internal Medicine, 7th ed. Stephen J. Ettinger, Edward C. Feldman

Gastro-eosophageal Reflux and Regurgitation in Anaesthetised Dogs

Liesel van der Merwe BVSc MMedVet(Med) Small Animals

Oesophagitis oesophageal strictures and aspiration pneumonia are potential consequences of peri-anaesthetic vomiting, regurgitation and gastro-oesophageal reflux (GER). A lot has been published recently on what could be risk factors for GER. Various studies have reported rates of regurgitation in anaesthetised dogs from 0.42 – 5.5%. Regurgitation is defined as the passive discharge of gastric or oesophageal fluid from the mouth or nose. GER in anaesthetised dogs is a more common but less visible complication than regurgitation and is detected in 16 – 55% of anaesthetised dogs.

GER occurs when the lower oesophageal sphincter (LES) tone is decreased due to the administration of anaesthetic agents, gastric acidity and withholding food prior to anaesthesia. Gastric contents will enter the oesophagus but not reach the pharynx. Monitoring of lower oesophageal pH has a high degree of sensitivity for detecting GER and is considered the gold standard. Reflux of gastric contents into the oesophagus is recorded when oesophageal pH decreases to <4 (acidic reflux) or increases above 7.5 (biliary reflux). Bile acids and gastric acids can work synergistically to cause greater inflammatory injury to the mucousa than either alone.

GER can be clinically silent and is only clinically detected as fluid reaches the oronasal cavity. Once oesophageal contents reach the pharynx and mouth it is termed regurgitation. Aspiration of the gastric material into the lungs may cause pneumonitis or or it may cause local oesophageal irritation and oesophagitis.

GER during anaesthesia is associated with 46 – 65% of cases of benign oesophageal stricture secondary to oesophagitis in dogs. Approximately 25% of dogs which have GER under anaesthesia have evidence of refluxed gastric content in the pharynx, increasing the risk of aspiration pneumonia. Prolonged exposure (>1-2 hours) of the oesophageal mucousa to acid (pH <4) is an important cause of oesophagitis. Oesophageal acid clearance does not occur under anaesthesia or during sleep.

Practical risk management - endotracheal entubation with inflated cuff until the animal regains full consciousness and laryngeal function. *References available online www.vet360.vetlink.co.za*

Table 1 – Medications and factors affecting GER

INCREASED REFLUX (GER)	DECREASED REFLUX (GER)
Morphine, ACP, Atropine , Xylazine,	IM meperidine (dogs, not cats)
Isoflourane (>> than halothane)	IV Maropitant 45 – 60 min prior (no vomiting with premed but still GER in 30% of dogs compared to 46% of control dogs)
50% of dogs receiving propofol had GER	Feed a small meal 3- hours prior to GA (reduced fasting time) (Savvas *et al* JAAHA, 2016)
Brachycephalic breed disposition due to increased negative intra-thoracic pressures (shown in some reports and not in others)	Maropitant did not prevent the occurrence of gastro-oesophgageal reflux (GER) although fewer dogs in the treated group did show GER.
Prolonged food withholding (18 hrs)	17,4% of dogs receving thiopentone had GER.
Metoclopramide (0.4mg/kg bolus with 0.3mg/kg CRI failed to reduce GER)	Metoclopramide 1mg/kg bolus IV and 1mg/kg CRI resulted in a 54% reduction in the risk of GER
Increased duration of surgery	Diazepam premed
Changes in body position during anaesthesia. GER is also more likely in dogs in sternal recumbency	Cisapride significantly decreased the frequency of GER (11% vs 36% in omeprazole and placebo group)
Intra-abdominal surgery.	
Orthopaedic surgery - often involves pre and post- operative imaging under the same anaesthesia and changes in the dogs position and transfer between areas may result in changes in abdominal and gastric pressures.	
Light meal 3 hour pre-GA (Viskjer *et al* AJVR)	
Patients > 40kg (larger breed size more than obesity)	
Omeprazole/esomeprazole – no effect on number of reflux events. But percentage time pH < 4 was significantly decreased	

**Some drugs and factors are clearly implicated whereas other factors are multivariate – example NSAIDs are implicated - but these are given in dogs for orthopaedic surgery which also generally receive morphine - so there is no direct cause and effect.*

Capital Expenditure in a Vet Practice

Andrew Christie
BComm (Business Management)

What is Capex?

Capital Expenditure (Capex) differs from Operating Expenditure (Opex) in that it refers to an outlay of funds that will produce benefits for a period longer than a year. Opex refers to an outlay of funds that will produce benefits for less than a year. Generally, Opex refers to expenses such as salaries and stock while Capex refers to money spent on vehicles, property, equipment etc.

1. Introduction

One of the weaknesses in many vet practices is the evaluation of the purchase of equipment and other large-scale purchases when little return can be expected. Vets rationalise purchases of newer, better equipment by claiming that they must remain competitive in their market and that better equipment leads to better animal care.

However, the reality is that a practice's clients know very little about equipment used and, in the current tough economic climate, the client is likely to choose a lower consult fee regardless of the equipment used.

Nevertheless, capital purchases remain a necessary component of practice's operations and it is critical for practice owners to begin evaluating their options from a profitability perspective as opposed to the more traditional 'need-to-be-up-to-date' approach.

2. What information do we want from the Capital Expenditure Evaluation (CEE)?

Three questioins need to be considered with CEE in a veterinary practice

a. **Will the item(s) make money?** Simply put, if Machine A is purchased, will it make or lose the practice money. This is particularly important when purchasing something prescribed by Council or something crucial to the day-to-day running of the practice.

b. **Should I choose between 2 or 3 or more alternatives?** If Machine A is selected, are there alternatives and which will make more money for the practice.

c. **Should I choose between several types of projects?** Machine A could be purchased, but how does it weigh up against Client Management System B or Strategic Marketing Plan C?

3. Net Cash Flows

The starting point for CEE is to look at the cash flows that will be generated by a Capex item. Normally this would be an increase in sales but could also be a reduction in costs – for example, a new practice management system reducing shrinkage (theft).

So far so good.

But very often the additional expenses incurred are overlooked. Purchasing a new bakkie may save fuel costs, but the insurance and services will be more expensive.

More subtle expenses must also be factored in – a new x-ray machine may produce wonderful images, but it may take longer to operate – certainly in the first month or two, anyway.

	Project A (R)	Project B (R)	Project C (R)
Initial Investment	**-700 000**	**-100 000**	**-80 000**
Increase in sales	**350 000**	**350 000**	**180 000**
Decrease in costs	**600 000**	**0**	**150 000**
	250 000	**250 000**	**250 000**
Increase in expenses	**-354 000**	**-390 000**	**-190 000**
Insurance	*-120 000*	*-120 000*	*-25 000*
Maintenance	*-234 000*	*-70 000*	*-90 000*
Salaries	*0*	*-200 000*	*-75 000*
Scrap value	**200 000**	**150 000**	**25 000**
Tax	**-105 000**	**-105 000**	**-54 000**
Cash Inflows / Outflows	**-9 000**	**-95 000**	**31 000**

Consider the table:

Any of the three questions that could be asked by a Vet can now be answered:

a. Will the item(s) make money? Project C will result in a net cash inflow.
b. Should I choose between 2 or 3 or more alternatives?
Project C is the only project to show a net cash inflow. (Bear in mind that while Projects A and B both generate negative cash flows, Project B is ranked below Project A).
c. Should I choose between several types of projects? The table does not indicate what the different projects are, but it is clear that Project C remains the most viable regardless.

The table shows the Net Cash Flows for each project – however, it is also vital to know when the cash flows occur.

4. Timing of the Cash Flows

However, there is a very important component missing – while the table shows the net cash flows, it is vital to know when the cash flows occur across the life of the Capex item. The table below shows the annual net cash flows for Projects A, B and C:

	Project A (R)	Project B (R)	Project C (R)
Initial Investment	**-700 000**	**-100 000**	**-80 000**
Net Cash Flow for ***Year 1***	246 000	-65 000	21 000
Net Cash Flow for ***Year 2***	155000	-50 000	19 000
Net Cash Flow for ***Year 3***	50 000	-30 000	17 000
Net Cash Flow for ***Year 4***	40 000	150 000	15 000
Net Cash Flow for ***Year 5***	40 000	*n/a*	14 000
Net Cash Flow for ***Year 6***	20 000	*n/a*	25 000
Net Cash Flow for ***Year 7***	140 000	*n/a*	*n/a*
Total Cash Flows	**-9 000**	**-95 000**	**31 000**

Note:

a. Each project has a different lifespan. Project C might be a new practice management system that will have benefits for 6 years, as opposed to Project B which might be a new computer system that has a shorter life span.
b. The last year of net cash flows for each project reflects a higher amount – this is because many capital items can be sold at the end of their useful lifespan. This could be scrapping old office furniture or selling a used vehicle.
c. The net cash flows do not necessarily reflect the actual life span – one practice may plan on using an item of machinery for 3 years, while another plans to use the same item

for 4 years. This means that the first practice would calculate net cash flows over a 3-year period, while the other would use a 4-year period.

So why is it important to forecast the net cash flows for each year? Simply put, because R100 today is worth more than R100 in the future. This is because:

- The R100 could be put in a savings account to generate interest
- The risk of receiving R100 in the future is much greater
- Inflation means that one can purchase less at a later stage

Put another way – would you rather someone give you R1 million right now, or in another 5 years? How are future cash flows discounted?

If I wanted to receive R1,000 in 4 years' time at an interest rate of 6%, I would have to invest R792. If this was flipped around, we could say, then, that in 4 years' time, R1,000 will equal R792 at today's rates.

Not so long ago, finance people would use pages and pages of tables like the one below. Now, of course, it's much simpler using either a spreadsheet program or the internet. In other words, at today's prices Project A would have a negative cash flow of R103,283.

Clearly Projects A and B are money wasters, but even Project C may not be a wise investment with such a low net cash flow. In fact, it might be a better investment to place the R80,000 in a savings account at the bank.

5. Conclusion

Capital investment within the veterinary industry needs to move towards financial rationalisation, where decisions are made based on the cash inflows that a project yields.

The same sluggish economy that is making this necessary for vet practices is making their customers seek less pricey consults, resulting in it becoming increasingly difficult to generate direct cash flows from new machinery and equipment.

Present value interest factor of R1 per period at i% for n periods, PVIF(i,n).

Period	1%	2%	3%	4%	5%	6%	7%	8%	9%	10%
1	0.990	0.980	0.971	0.962	0.952	0.943	0.935	0.926	0.917	0.909
2	0.980	0.961	0.943	0.925	0.907	0.890	0.873	0.857	0.842	0.826
3	0.971	0.942	0.915	0.889	0.864	0.840	0.816	0.794	0.772	0.751
4	0.961	0.924	0.888	0.855	0.823	0.792	0.763	0.735	0.708	0.683
5	0.951	0.906	0.863	0.822	0.784	0.747	0.713	0.681	0.650	0.621
6	0.942	0.888	0.837	0.790	0.746	0.705	0.666	0.630	0.596	0.564
7	0.933	0.871	0.813	0.760	0.711	0.665	0.623	0.583	0.547	0.513
8	0.923	0.853	0.789	0.731	0.677	0.627	0.582	0.540	0.502	0.467
9	0.914	0.837	0.766	0.703	0.645	0.592	0.544	0.500	0.460	0.424
10	0.905	0.820	0.744	0.676	0.614	0.558	0.508	0.463	0.422	0.386

Using the inflation rate of 5% as a benchmark, the net cash flows for Project A would be:

	Project A		Present Value Factor		Present Value
	R				R
Initial Investment	-700 000	x	1.00	=	-700 000
Net Cash Flow for Year 1	246 000	x	0.95	=	234 192
Net Cash Flow for Year 2	155000	x	0.91	=	140 585
Net Cash Flow for Year 3	50 000	x	0.86	=	43 200
Net Cash Flow for Year 4	40 000	x	0.82	=	32 920
Net Cash Flow for Year 5	40 000	x	0.78	=	31 360
Net Cash Flow for Year 6	20 000	x	0.75	=	14 920
Net Cash Flow for Year 7	140 000	x	0.71	=	99 540
Total Cash Flows	-9 000				-103 283

When the exercise is repeated for Projects B and C, this is the revised summary of all the Projects:

	Project A (R)	Project B (R)	Project C (R)
Initial Investment	-700 000	-100 000	-80 000
Net Cash Flow for Year 1	234 192	-61 880	19 992
Net Cash Flow for Year 2	140 585	-45 350	17 233
Net Cash Flow for Year 3	43 200	-25 920	14 688
Net Cash Flow for Year 4	32 920	123 450	12 345
Net Cash Flow for Year 5	31 360	n/a	10 976
Net Cash Flow for Year 6	14 920	n/a	18 650
Net Cash Flow for Year 7	99 540	n/a	n/a
Total Cash Flows	-103 283	-109 700	13 884

On a broader level, practices need to be run on sound business principles to maintain and improve profitability in a rapidly changing veterinary landscape. An increasing amount of attention is being paid to performance – the opex component – and it is time that capex decisions are made with sound financial reasoning.

The ABCs of Veterinary Dentistry

'P' is for Periodontal Pockets

Do you know how to help patients with pockets? Get the lowdown on subgingival cleaning, laser gingivectomy and locally applied antimicrobials and sealants.

By Jan Bellows, DVM, DAVDC, DABVP, FAVD

The gingival sulcus is a normal shallow space between the marginal gingiva and the tooth. Its depth is generally 0.5 to 1 mm in cats and 1 to 5 mm in dogs, depending on the specific tooth and the size of the patient.

A pocket is a pathologically deepened gingival sulcus that occurs secondary to coronal movement of the gingival margin (pseudopocket), apical movement of the gingival attachment (periodontal pocket) or a combination of both. The clinical or absolute pocket depth is the distance from the gingival margin to the base of a pocket (measured in millimeters).

Gingival recession refers to the displacement of the gingival margin apical to the cementoenamel junction. Periodontal pockets and pseudopockets can occur together with gingival recession.

In this article, I will help you diagnose pockets and determine the optimal treatment.

Suprabony and infrabony pockets

Suprabony pockets, also referred to as supra-alveolar and supracrestal pockets, occur above the crest of alveolar bone (Figure 1). The lateral wall of the suprabony pocket consists of epithelial tissue. When the suprabony pocket is less than 5 mm in a medium or large dog, representing stage 2 periodontal disease, treatment includes the removal of supra- and subgingival plaque and calculus and closed root planing. If the 5-mm pocket represents stage 3 or 4 periodontal disease, consider extraction. Treatment may also include locally applied antibiotics. For suprabony pockets greater than 5 mm without gingival recession, coronal repositioned flap surgery can be performed by a practitioner with advanced training in periodontal surgery.

Infrabony pockets, also referred to as intra-alveolar pockets, occur when the pocket floor (epithelial attachment) is apical to the alveolar bone (Figure 1). The lateral wall will consist of epithelial tissue and bone. Radiographically, infrabony pockets appear as vertical bone loss along the root surface. However,

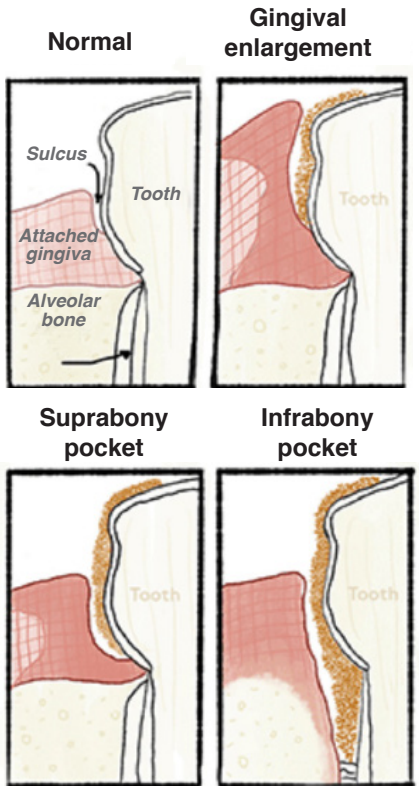

Figure 1: Illustrations of normal and abnormal gingival conditions. (Illustration by Roxy Townsend)

radiographs generally cannot be used to diagnose pockets since they are soft tissue defects.

Infrabony defects can be further classified by the number of walls remaining around the tooth—information that can help inform treatment decisions. An infrabony defect is shaped like a box without a top. The bottom of the box is the base of the pocket. One of the box's sides is the tooth root. The three remaining sides of the box are the potential walls of the defect.

There is a direct relationship between the prognosis of the therapy and the number of intact walls. Three-wall defects that have progressed to stage 3 periodontal disease (25% to 50% support loss) have the best prognosis for new attachment after advanced periodontal surgery, bone grafts and stringent home care. Two-wall defects lag behind in terms of treatment prognosis, and one- and no-wall defects carry the worst prognosis. For patients with stage 2 periodontal disease (less than 25% support loss) easier treatment options can be effective (see below).

Gingival enlargement and resultant pseudopockets

Gingival enlargement is an increase in the size or thickness of the gingiva (Figure 1). Gingival hyperplasia, a histopathologic term, is an increased number of normal cells in normal arrangement. Gingival hypertrophy is an increase in the size of individual cells. Gingival hyperplasia and hypertrophy can be accurately diagnosed only microscopically. When viewed clinically without histologic confirmation, this condition is correctly referred to as gingival enlargement.

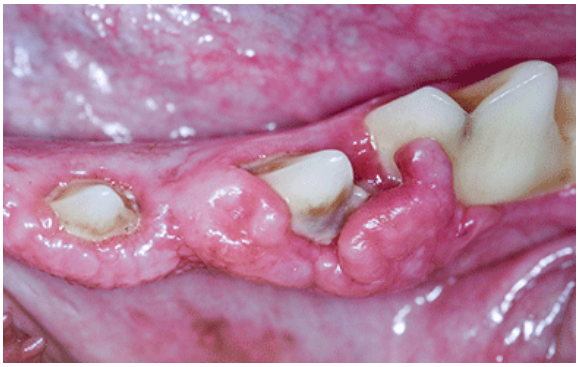

Figure 2A: Gingival enlargement resulting in pseudopockets. (All images courtesy of Dr. Jan Bellows).

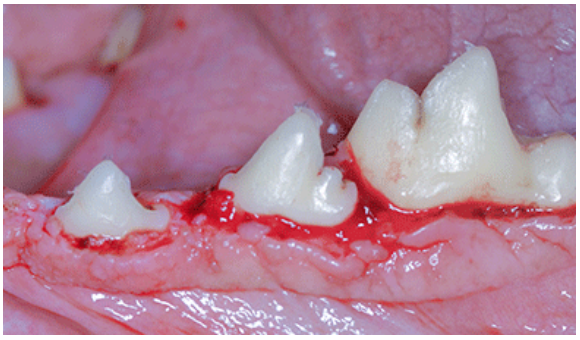

Figure 2B: A gingivectomy surrounding the left maxillary third and fourth premolars and first molar is performed, eliminating the pseudopockets

The specific cause of gingival enlargement is unknown, but there may be a genetic predisposition in boxers, rottweilers, Great Danes, collies, Doberman pinschers, Dalmatians and golden retrievers. Cyclosporine, phenytoin and calcium channel blocker medications, including amlodipine, have also been implicated (Figure 2A). Elimination of these medications, coupled with dental scaling, polishing and removal of the enlarged gingiva (preserving at least 2 mm of attached gingiva), usually results in a cure in cases caused by medication (Figure 2B).

Gingival enlargement can lead to increased pocket depths secondary to augmented gingival height versus

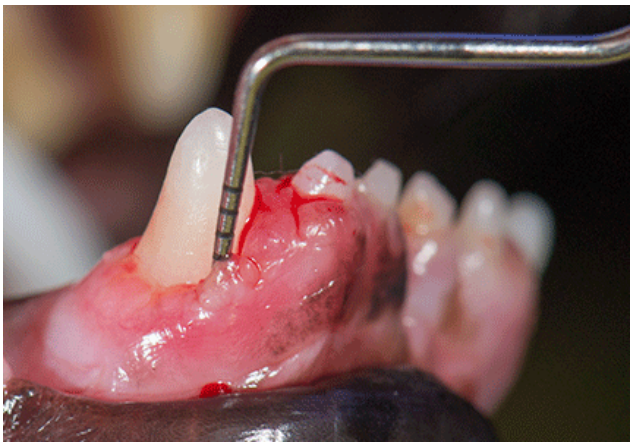

Figure 3A.: Gingival enlargement secondary to amlodipine.

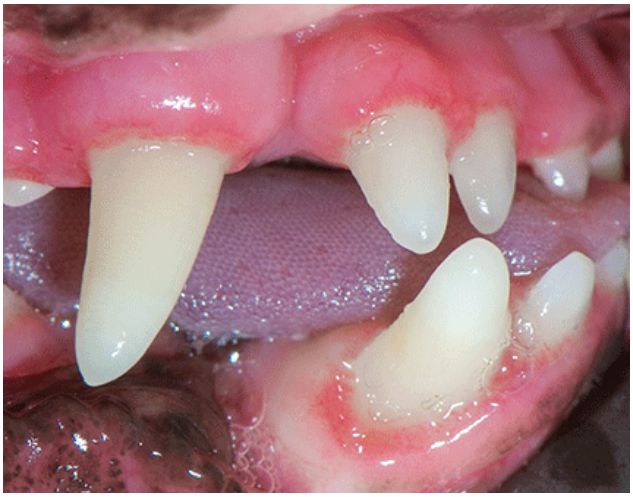

Figure 3B: Gingival enlargement resolved after discontinuation of medication and laser-assisted gingivectomy.

Periodontal nomenclature

An **alveolectomy** is the removal of some or all of the alveolar bone.

An **alveoloplasty** is a form of alveolectomy performed to restore physiological contours or achieve smooth contours of the alveolar bone.

An **apically positioned flap** is moved apical to its original location.

An **envelope flap** is retracted away from a horizontal incision; there is no vertical incision.

Closed periodontal debridement involves the removal of damaged, infected, inflamed or necrotic tissue from periodontal pockets and dental deposits from the tooth surface without the creation of a flap; this includes gingival curettage (or excisional new attachment procedure) and root planing.

A **coronally positioned flap** is moved coronal to its original location.

Gingival curettage refers to the removal of damaged, infected, inflamed or necrotic tissue from the soft tissue lining of a periodontal pocket.

Gingival enlargement is a clinical term referring to the overgrowth or thickening of gingiva in the absence of a histological diagnosis.

A **gingival flap** contains gingiva.

Gingival hyperplasia is a histological term referring to an abnormal increase in the number of normal cells in a normal arrangement resulting in clinical gingival enlargement.

Gingival recession refers to root surface exposure caused by apical migration of the gingival margin or loss of gingiva

Gingivectomy refers to removal of some or all gingiva surrounding a tooth.

Gingivoplasty is a form of gingivectomy performed to restore physiological contours of the gingiva.

A **mesiodistally** or **distomesially positioned flap** is moved distal or mesial to its original location along the dental arch; this flap has also been called a **laterally positioned flap**.

Open periodontal debridement is the removal of damaged, infected, inflamed or necrotic tissue from periodontal pockets and dental deposits from the tooth surface after flap creation; this includes the removal of affected gingiva and granulation tissue upon flap creation and management, root planing, and osseous resective procedures such as an alveolectomy and alveoloplasty.

A **periodontal flap** contains gingiva and alveolar mucosa.

Root planing refers to the removal of dental deposits from and smoothing of the root surface of a tooth; it is described as closed when performed without a flap and open when performed after flap creation.

attachment loss (Figure 3A and 3B). The resultant pseudopocket accumulates plaque and calculus, which, if left untreated, may progress to attachment loss. Surgical treatment, including gingivectomy and gingivoplasty, is performed using a scalpel blade, laser or radiosurgery to sculpt the gingiva and decrease or eliminate the pseudopockets. What to do when you find a pocket

The goal of periodontal therapy is to decrease the size of or eliminate pockets in cases of early and moderate periodontal disease by removing subgingival plaque and calculus, using locally applied antimicrobials, performing gingivectomy, or extracting the affected teeth. Extraction is indicated when more than half of the root is not supported by the periodontium.

Subgingival cleaning: ultrasonic scaling.

Bacteria-coated calculus left on the root surface contributes to the progression of periodontal disease. In order for the ultrasonic scaler to therapeutically débride a periodontal pocket, it needs to contact every part of the accessible root surface. Using ultrasonic thin periodontal tips specifically manufactured for root surface use, place the scaler tip's side parallel to the long axis of the tooth, similar to positioning a diagnostic periodontal probe. To avoid iatrogenic damage, decrease the power and increase the amount of water

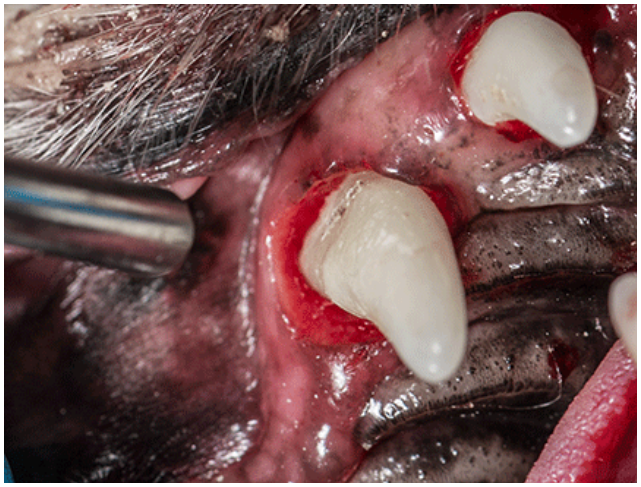

Figure 4: An air/water syringe is used to show plaque and calculus not removed during scaling and polishing

irrigation to remove subgingival plaque and calculus. After you've completed ultrasonic tooth scaling, use an air/water syringe to gently blow the gingival margin away from the tooth and examine the tooth surface for remaining plaque and calculus to remove (Figure 4). You can then use water from the air/water syringe to lavage unattached debris from the sulcus or pocket.

Subgingival cleaning: curette-assisted root planing.

The goal of root planing is to make the root less

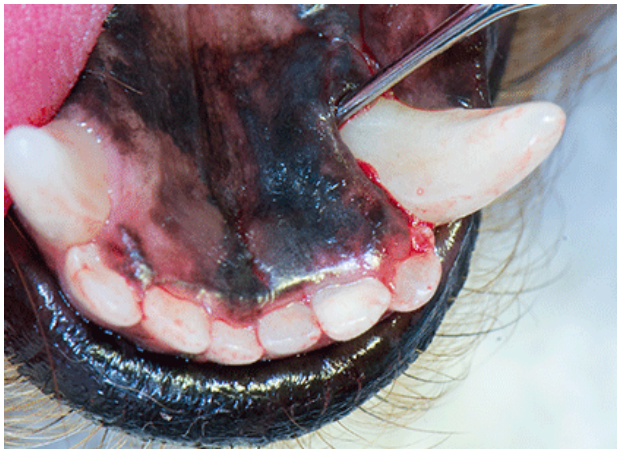

Figure 5. Subgingival root planing.

supportive of bacterial colony formation, plaque and calculus (Figure 5). Insert the curette with the face of the blade flush against the tooth. When the instrument reaches the bottom of the pocket, the working angulation of the instrument (between 45 and 90 degrees) is established. Place the instrument against the tooth, pulling coronally and repeating the process until all subgingival calculus is removed.

Locally applied antibiotics.

Applying local antibiotics is thought to reduce pocket depth by aiding in tissue shrinkage, connective tissue remodeling and soft tissue attachment. Diligent home care is essential for maintenance.

There are two locally applied antibiotic products approved for dental conditions in small animals: Clindoral (TriLogic Pharma) and Doxirobe Gel (Zoetis). In cases of early periodontal disease, the biodegradable insertion of either of these products allows for the sustained release of therapeutic levels of the antimicrobial for several weeks at the injection site. However, neither product is a substitute for scrupulous pocket débridement and subsequent home care, and neither one should be applied to unclean root surfaces.

Clindoral is a periodontal pocket filler containing 2% clindamycin hydrochloride in a biodegrading, mucoadhesive gel matrix that releases clindamycin to the dried periodontal pocket or sulcus over a period of seven to 10 days after a single application (Figures 6A-6C). As the product warms to body temperature, it increases in viscosity two- to threefold to form a soft, pliable matrix the consistency of thick jam. Any liquid product that is part of the cleaning (e.g. fluoride, chlorhexidine) should be applied before applying Clindoral, and tooth sealants should be applied after. The pet owner should abstain from wiping or brushing the pet's teeth or giving dental treats for seven days after application.

Doxirobe Gel is provided in a two-syringe system. Syringe A contains the polymer delivery system:

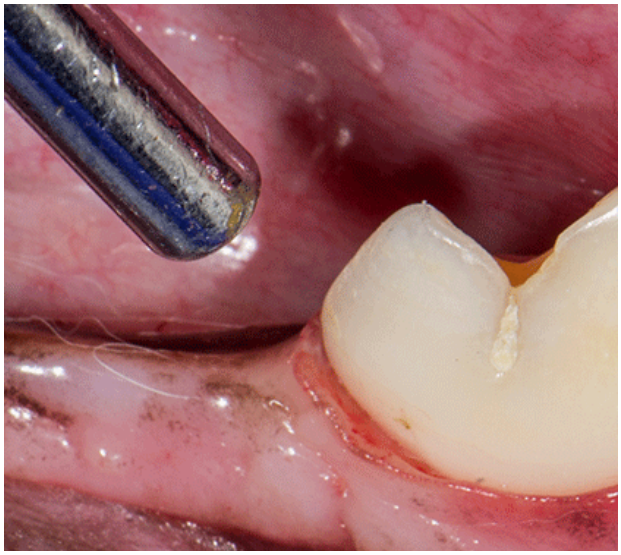

Figure 6A: An air/water syringe is used to dry a 4-mm periodontal pocket on the distal root of a dog's right mandibular first molar.

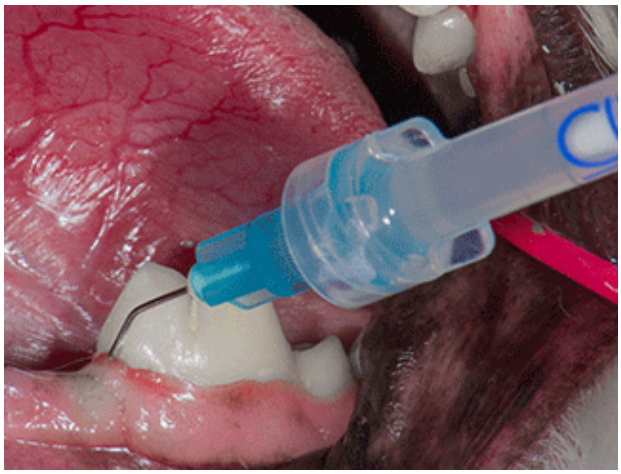

Figure 6B: Clindoral application.

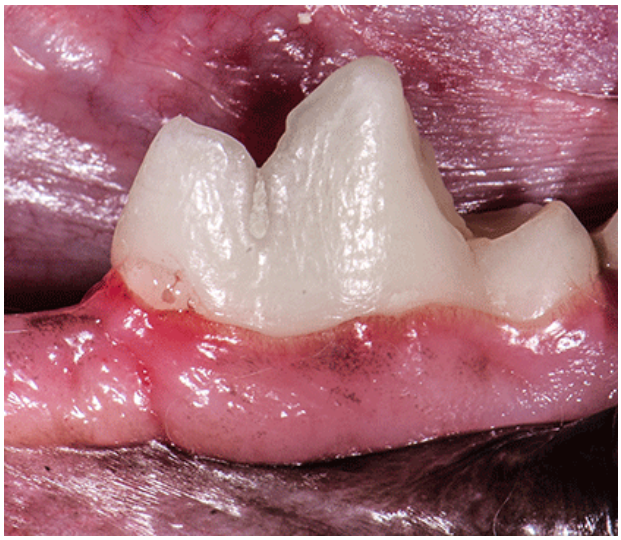

Figure 6C: The appearance of extruded medication from the pocket confirms a complete fill.

N-methyl-2-pyrrolidone and poly (D,L-lactide). Syringe B contains the active ingredient: doxycycline hyclate. Once combined, the product is a flowable mix equivalent to 8.5% doxycycline. When applied subgingivally, doxycycline is slowly released from the polymer, providing a local antimicrobial effect similar to Clindoral, particularly toward gram-negative anaerobic bacteria involved in periodontal disease. As with Clindoral, clients should avoid wiping and tooth brushing for one week.

Diode laser

Lasers may have a place in periodontal therapy. Diode laser energy transmitted through a thin fiber placed into a periodontal pocket is absorbed by the melanin and hemoglobin that are present in periodontal disease. For humans, dental protocols include the débridement of the hard side of the pocket (tooth and root surface) with ultrasonic scalers and hand instrumentation.

Measure the laser fiber to a length of 1 mm short of the pocket depth and use the energized fiber tip in light contact with a sweeping action that covers the entire epithelial lining, from near the base of the pocket upward (Figures 7A-7C and 8). Clean the fiber tip often with damp gauze to prevent the buildup of debris.

More randomized controlled clinical trials are needed to measure the benefit of using lasers as an adjunct to nonsurgical periodontal therapy.

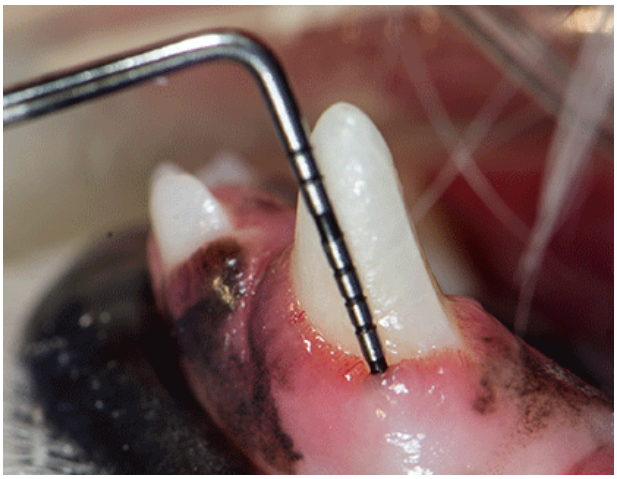

Figure 7A: A 6-mm periodontal pocket affecting a dog's left mandibular canine.

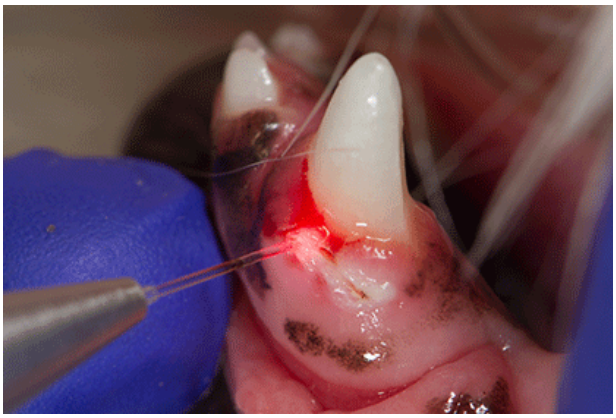

Figure 7B: Laser gingivectomy of the dog's left mandibular canine.

Locally applied sealants

In human dentistry, a dental sealant is a thin, plastic coating painted on the chewing surfaces of teeth to prevent caries. In veterinary dentistry, caries are rare, so sealants are applied to help prevent periodontal disease (Figure 9). Currently, there are two commercially available veterinary dental sealants that have been clinically proven to prevent the reattachment of plaque and calculus: SANOS Dental Sealant and OraVet Plaque Prevention Gel.

Figure 7C: The dog's decreased pocket depth following the laser gingivectomy.

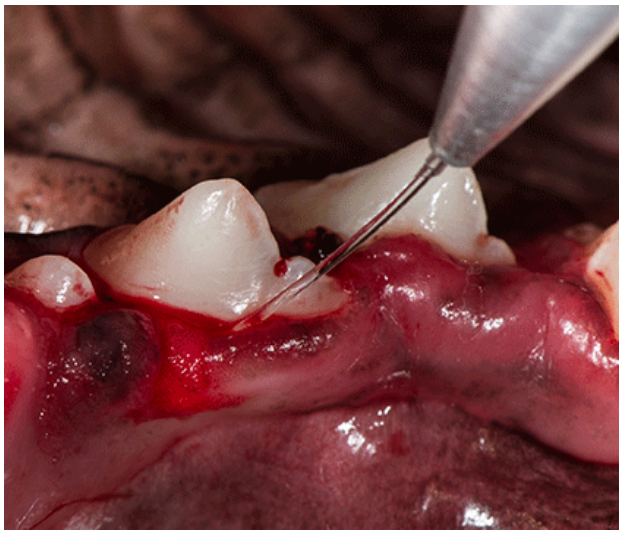

Figure 8: Locally applied diode laser energy into a dog's 4-mm pocket.

SANOS Dental Sealant has been accepted by the Veterinary Oral Health Council and is applied by a veterinary professional during the oral hygiene procedure. SANOS is a hydrophilic polymer that seals the subgingival sulcus or small pocket from the accumulation of plaque and tartar. The hydrophilic design of the sealant is uniquely engineered to attract water and allow oxygen to pass through to create an unfavorable environment for anaerobes. Reapplication is recommended at six-month intervals. Home care products are OK to use with SANOS, though withholding dental diets, dental chews, water additives and gels for seven days after a professional cleaning is recommended.

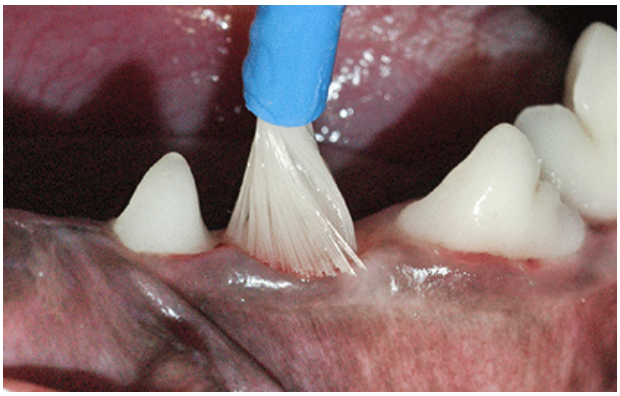

Figure 9. Dental sealant application.

OraVet Plaque Prevention Gel is a hydrophobic wax that binds electrostatically to tooth enamel, creating a barrier that helps prevent plaque-forming bacteria from attaching. OraVet is applied professionally during the oral hygiene procedure and then weekly by the client thereafter. It has been clinically proven to significantly reduce the formation of plaque and calculus in dogs and cats.[1-3] There are no restrictions on withholding treats or at-home dental care after application.

Periodontal disease is the most common malady affecting dogs and cats. With 42 teeth in dogs and 30 in cats, you have a great opportunity to make a difference in the lives of your patients and their caregivers.

References available online: www.vet360.vetlink.co.za

Is Tramadol the New Placebo?

A recent study challenges the use of tramadol for canine osteoarthritis.

Budsberg SC, Torres BT, Kleine SA, et al. Lack of effectiveness of tramadol hydrochloride for the treatment of pain and joint dysfunction in dogs with chronic osteoarthritis. J Am Vet Med Assoc. 2018 Feb 15;252(4):427-432.
Summarised by Amy Van Gels, DVM

A placebo has no therapeutic action. However, the improvement in clinical signs (or development of side effects) associated with the administration of a placebo—the placebo effect—is a well-known phenomenon that affects human patients and caregivers alike. Physicians and veterinarians are not immune to its effects.

Too easily, the improvement exhibited by a few patients can seemingly prove the efficacy of a treatment and promote further use in other patients. To avoid this bias, clinicians rely on evidence-based medicine and the use of placebo-controlled clinical trials to ascertain whether the effects of a medication are indeed beneficial.

In the last 10 to 15 years, tramadol has been widely used to control pain despite a lack of scientific evidence to support its use. Pharmacologically, dogs are known to produce insufficient amounts of the metabolite of tramadol that is responsible for analgesia, and the bioavailability of this metabolite drops significantly within one week of regular use. Clinically, few studies have been performed evaluating the efficacy of tramadol in veterinary species. That is, at least, until February of this year when a clinical trial was published that examined whether or not tramadol is effective for the management of pain associated with canine osteoarthritis.

What they did

Forty dogs with radiographically confirmed osteoarthritis of the elbow or stifle were included in a randomized, blinded, placebo-controlled, crossover study. All dogs received each of the following treatment regimens for 10 days, with at least a week of washout between treatment periods:

- Tramadol: 5 mg/kg every 8 hours (morning, midday and night)
- Carprofen: 2.2 mg/kg every 12 hours (morning and night, with a placebo midday)
- Placebo: lactose powder every 8 hours

The dogs received these treatment regimens in random order, and all medications appeared identical. To evaluate treatment response, force-plate analysis and pain scoring (using the Canine Brief Pain Inventory) were performed before each treatment period (baseline) and on the last day of each treatment regimen. A force plate measured vertical impact (VI) and peak vertical force (PVF) to measure the arthritic limb's weight-bearing ability. To calculate the pain score, the owner assessed the severity of the dog's pain and the degree to which pain interfered with daily activities.

What they found

Thirty-five dogs completed the study. Force-plate readings (VI and PVF) improved significantly from the baseline while the dogs received carprofen but not while they received tramadol or the placebo. The extent of the improvement seen with carprofen was also significantly greater than that seen with tramadol or the placebo.

Based on a reduction in pain scores, significantly more dogs improved while taking carprofen (42%) than while taking tramadol (24%) or the placebo (21%). There was no significant difference in pain scores between the tramadol and the placebo regimens.

Take-home message

In this study, the effects of tramadol were similar to the placebo. Although some patients may improve while taking a placebo, the improvement cannot be attributed to its action. For a medication to be considered effective, it must produce a significant benefit as compared to the placebo. In this study, carprofen proved therapeutic for the pain and dysfunction of canine osteoarthritis, whereas tramadol did not.

So, if an arthritic patient improves while taking tramadol, is it just the placebo effect? The results of this study suggest that it is.

Study shows Tramadol has no effect on osteoarthritis pain scores

Commonly prescribed drug not associated with improvement when compared with NSAID; experts vary on whether it still has a place in veterinary pain management.

By Kristi Reimer Fender

When researchers attempted to demonstrate measurable effects of tramadol on osteoarthritis pain, they came up empty. Researchers from the University of Georgia have found that tramadol is ineffective in alleviating signs of pain associated with osteoarthritis in dogs, according to a release from the Morris Animal Foundation (MAF), which funded the study. The research team published their results in the Feb. 15 issue of the Journal of the American Veterinary Medical Association.

"The data shows conclusively that tramadol is not an effective drug in treating the pain associated with arthritis in the dog, despite its common recommendation," says Steven Budsberg, DVM, MS, DACVS, professor of surgery and director of clinical research at the University of Georgia College of Veterinary Medicine, in the MAF release. "This use of tramadol is a classic example of failing to acknowledge and control for bias when evaluating a potential treatment."

The team at the University of Georgia, led by Dr. Budsberg, compared the use of tramadol with both a placebo and the nonsteroidal anti-inflammatory drug carprofen in client-owned dogs in a randomized, blinded, placebo- and positive-controlled crossover study, according to the study abstract.

Dogs with osteoarthritis of the elbow or knee were assigned to receive each of the three treatments in a random order, with each treatment arm lasting 10 days. Improvement was measured using vertical impulse, peak vertical force and Canine Brief Pain Inventory scores to assess gait and pain levels. The results showed no improvement when tramadol was given compared to either baseline or placebo. Carprofen was associated with significant improvement in results.

We reached out to several veterinary pain experts who contribute to dvm360 and the Fetch dvm360 conferences for their thoughts on the study. As experts often do, they diverge in their assessment of whether tramadol has a role in veterinary practice going forward.

OPINIONS

Opinion No. 1: 'Why would you use it?' - Dr. Michael Petty

Michael Petty, DVM, CVPP, CVMA, CCRT, CAAPM, owner of Arbor Pointe Veterinary Hospital and Animal Pain Center in Canton, Michigan, comes down squarely on the "don't use it" end of the spectrum. He says veterinary pain experts have known for a number of years that orally administered tramadol doesn't work well for the treatment or prevention of acute pain, "and those of us with pain practices have suspected for several years that tramadol doesn't work for chronic pain either," he continues

"This is based on personal experience—not always the best measure—and on some pharmacokinetic studies showing that the active metabolite is not detectable in many dogs receiving tramadol, even those receiving several hundred milligrams per dose," he says. "The study by Budsberg and his colleagues is a good one and underlines that tramadol is not a drug that can be depended on for chronic pain issues."

Dr. Petty says tramadol is a serotonin reuptake inhibitor, which can help with mood, and this could account for what some people see as a positive result when giving it. But it also has a history of causing serotonin syndrome, even at small doses and upon the first administration to a particular dog or cat. "You have a drug that is a controlled substance, has the potential for human abuse, has no studies showing it works in either acute or chronic pain, and might kill your patient," Dr. Petty concludes. "Everyone who wants to prescribe it to a patient needs to imagine the courtroom scenario where you're trying to defend your decision to use it on an animal that came to harm."

Opinion No. 2: 'My patients benefit' - Dr. Dani McVety

Like Dr. Petty, Dani McVety, DVM, founder and CEO of the Lap of Love Veterinary Hospice network, says the study results are not unexpected. "I looked at the study, but I didn't really have to," she says. "It's truly no surprise to me or anyone else who's been using tramadol for years that it's not the perfect pain medicine."
But she says hospice practitioners occasionally need to push the boundaries on medical comfort measures, and for her, tramadol will continue to have a role in managing veterinary pain.

"When I prescribe tramadol, I tell my clients that it's not used directly for pain reduction, but more like a glass of wine," she says. "Sometimes you need one; sometimes you need two or three to get calmed down a bit. But if we're leaning on a very high dose for more than one to two nights, we have a quality of life issue and need to have a separate conversation." In other words, Dr. McVety uses tramadol in a limited capacity to get a very specific reaction from the pet: calmness. "Sometimes just reducing the emotional wind-up that occurs with pain is helpful to our clients and their pets," she says. "This is why we will not stop using this medication, combined with adequate pain relief."

Opinion No. 3: 'The jury is still out' - Dr. Ralph Harvey

Ralph Harvey, DVM, MS, DACVAA, is associate professor of anaesthesiology at the University of Tennessee College of Veterinary Medicine. He says trying to understand all the nuances of how living things experience pain—and how various drugs affect that experience—is like trying to answer the question "What is truth?"

"I have a great deal of faith in Budsberg and the validated models he uses," Dr. Harvey says. "The work he has done is meaningful and useful, while not surprising. But many veterinarians far more well-known and expert than I am continue to advocate for the use of tramadol. And veterinarians continue to have the impression that it's beneficial."

When Dr. Harvey gives talks on pain management, he asks participants to raise a hand if they've heard experts say there's little to no evidence supporting the use of tramadol in veterinary patients. Many hands go up. "Then I ask how many of them are using tramadol anyway," Dr. Harvey says. "And many hands go up or stay up." In the pyramid model of evidence-based medicine, Dr. Harvey continues, double-blind placebo-controlled studies such as Dr. Budsberg's rank higher than the bottom level of expert opinion and clinical impression, which can be clouded by bias and wishful thinking. But still, he believes the jury is out on whether tramadol has value in mitigating the experience of pain in veterinary patients.

The problem, he proposes, lies in thinking of tramadol as an analgesic. "We do better to think of it as an emotion-modifying drug—at least in those animals that produce the right metabolite," he says. "Dogs are extremely variable in their ability to produce that mu-receptor-binding metabolite. Some dogs do; others don't." Dr. Harvey refers to the work of psychologist Ronald Melzack, who several decades ago presented a theory differentiating between two aspects of pain. The discriminative aspect of pain, Melzack says, is highly localized, specific and sharp—it's what you would experience if you touched a hot stove. The affective aspect of pain is not how it feels but how it makes us feel—it's the suffering or emotional component of pain.

"The affect of pain is less distinct and slower to come across, and it degrades engagement with life," Dr. Harvey says. "It's what Freud was talking about when he said a man with a toothache cannot be in love." Of course, no validated assay exists for the emotional experience of animal pain, so it's extremely difficult to provide evidence that a drug like tramadol is efficacious in relieving it. Another confounding factor is that tramadol is best used as a complementary therapy, Dr. Harvey says, which muddies the waters in terms of knowing which drug is causing which effect.

"But absence of evidence is not evidence of absence," Dr. Harvey says. "Our greatest limitation in evaluating pain is our ability to recognize and quantify it, especially when it comes to validated models for the suffering aspect of pain. This is a new frontier we are only just beginning to explore." Issues surrounding tramadol use—its status as a controlled substance, the potential for human abuse and diversion, the risk of adverse effects such as serotonin syndrome, and patients' varied ability to metabolize the drug—must all play a role in an individual veterinarian's decision to use it or not. But is this study the last word on the subject? No, Dr. Harvey says. "My impression is that, while I have great respect for this study, the metric is limited to the sensory rather than the emotional component of pain," he says. "But Dr. Budsberg has elevated the conversation, and this is a rich area for continued discussion."

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