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Vol 07 | Issue 01 | February 2020

CPD Article

GDV: It Doesn't Have to Make Your Stomach Turn

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Urinary Tract

Managing Recurrent UTIs in Pets

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Key Points for Managing Cutaneous Mast Cell Tumours in Dogs

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Editor's Note

To start of 2020 , I've included an article with some tips for a recession - with our probable downgrading by Moody's - it might be time to start following some of this advice.

Included is an article by Dr Abdur Kadwa, an anaesthesiology resident at OP, on some of the expanded uses for lidocaine in small animals, supported by two journal scans on the same topic.

The CPD article on canine GDV is a nice complete summary to the diagnosis, surgery and management of such a case.

This edition contains smaller articles highlighting relevant topics; recurrent UTI and mast cell tumours in dogs and tips for the dermatological examination.

Hope you enjoy the read.

Liesel

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South African Veterinary Association
Suid-Afrikaanse Veterinêre Vereniging

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Vet360 is a source of current, relevant educational material for practising veterinary professionals in Southern Africa.
Published Bi-monthly in hard copy and electronically on www.vet360.vetlink.co.za.

Distribution: Southern Africa, 3 500 copies. Copyright reserved.

Editor: Dr Liesel van der Merwe BVSc (Hons) MMedVet (Med) Small Animals.
Layout and design: Heinrich van Rijn
Publisher and Owner: Vetlink Publications
Other Publications by Vetlink: Vet360 Mobile App, Livestock Health and Production Review, Hooo-Hooo, Equine Health Update

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The Editor, PO Box 232, GROENKLOOF, 0027
Tel: (012) 346 1590, 082 575 6479. Fax: 086 671 9907
Email: lieselvdmvet@gmail.com
(Dr Liesel van der Merwe)

Madaleen Schultheiss

The Next Recession is Coming: Here is What to do Now

Matthew Salois, PhD

The 'R' word is an economic inevitability, but that doesn't mean you should panic. Take these five steps to prepare your veterinary practice for the next downturn.

There's no crystal ball to tell us when the next recession will occur or how severe it will be. But economists do rely on a set of key economic indicators to help gauge the performance of the economy and what direction it's heading in. These key indicators—include unemploymentand consumer sentiment. We do know there will be another recession eventually. They're a normal part of the economic cycle.

Whether the next downturn starts in months or years, there's no reason to panic. But there's always good reason to prepare. Forward-thinking business owners can actually make recessions work in their favour.

Five ways to shore up your practice now

The actions you take in a strong economy can set you up to reap dividends when a downturn arrives. Here are important steps you can take now to safeguard both your practice and your personal finances once a recession hits.

1. **Reduce the debt on your books.** In a recession, you'll have lower sales and less available cash. That calls for deft financial management. If your business is heavily in debt, you may be more vulnerable. Do what you can to reduce your debt burden now by consolidating loans or refinancing at a lower interest rate. Studies have shown that many businesses that failed during the Great Recession had much higher debt-to-asset ratios than those that survived and thrived.

2. **Strengthen and build your business**. Now is the time to be aggressive in seeking new clients and building stronger relationships with current ones. It can be tempting to take it easy when times are good, but the exact opposite is the wiser course.

Building during boom times helps insulate against a recession. Work extra hard to help existing clients understand the importance of compliance with preventive care guidelines now, when the healthy economy means they're more likely to prioritise veterinary care. Similarly, now is not the time to cut back on work hours if you can avoid it.

3. **Invest in digital technology.** It's tempting to think of battening down the hatches during uncertain economic times, but improving your technology now can put you in a better position to manage uncertainty. If you've been considering experimenting with telemedicine, a client-facing app, or texting or analytics platforms, committing now can put you in a better position during the inevitable downturn.

4. **Examine your decision-making process.** In the last recession, companies that decentralised decision-making fared better than those that clung to authority. Leaders who passed authority further down the chain of command found that their employees remained more committed and involved. Although it might make you feel uneasy, work on delegating various elements of your practice oversight. At the very least, seek more input from employees at all levels.

5. **Avoid layoffs if possible, even when the recession hits.** Look beyond headcount reduction to strengthen your practice's finances. Layoffs hurt productivity and morale. If you need to cut payroll costs, consider alternatives such as furloughs, reduced hours, or reduction or elimination of performance pay. Above all, be transparent with staff in advance about your need to reduce expenses. You might be amazed by the ideas they come up with, given the chance.

Resources to bolster your practice against recession

The AVMA has numerous tools to help you strengthen your practice before and during a recession. These include a market share calculator, marketing and social media materials, human resources materials covering everything from team building to payroll, and even a purchasing tool (AVMA Direct Connect) that includes real-time price comparisons for consumer pharmacies.

The newly redesigned AVMA website includes a dedicated practice management section and a personal finance section, too. Visit avma.org/PracticeManagement and avma.org/PersonalFinance. The AVMA also has a growing library of business and financial CE on AVMA Axon. Check the financial health section at Axon for timely guidance on both practice and personal finances, including courses we offered at October's AVMA Economic Summit.

Economic indicators to monitor

AVMA's economists recommend that veterinarians get in the habit of tracking four key economic indicators for signs of recession. You can do this very quickly on a daily basis. Just five to 10 minutes with your morning coffee will keep you informed.

1. **Unemployment rate.** Recently, the U.S. unemployment rate has been hovering near its 50-year low, which should be reassuring. What matters is change over time. If the unemployment rate rises quickly, that almost guarantees a recession has begun. (Unemployment in South Africa is currently at 30% - an all time high - .ed)

2. **Yield curve inversion**. Long-term investments normally earn higher interest than short-term. In a yield curve inversion, this is reversed. For example, a three-month U.S. Treasury bond may pay a higher rate of return than a 10-year bond. This means investors have low confidence in the economy. A yield curve inversion usually shows up well in advance of a recession, so there's no need to panic. It can take as long as two years for a recession to develop after this happens.

3. **University of Michigan Index of Consumer Sentiment.** *Consumer spending drives our economy. This monthly index tracks how confident consumers are that the economy is healthy. Look for long-term changes rather than one-month fluctuations—for example, a 15% year-over-year drop lasting for several months*

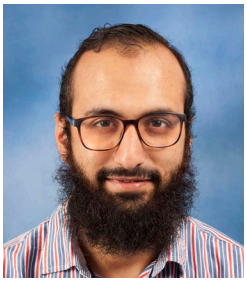
4. **Manufacturing sector Purchasing Managers Index (PMI)**. *Each month, the Institute for Supply Management surveys its members, then aggregates the data on a scale of 1 to 100. If the PMI is 50 or above, manufacturing expanded that month. Below 50 indicates that the sector contracted. Manufacturing no longer drives the U.S. economy, so a dip in the PMI is not as significant as it used to be. Steep downturns are a sign of trouble, but not a guarantee that recession has arrived.*

Remember: Recessions are normal, so don't overreact to either positive or negative financial news. Stay informed, but don't dwell on the negative. A relentless focus on streamlining your practice and achieving your goals is the best way to weather any downturn.

Matthew Salois, PhD, is chief economist and director of the Veterinary Economics Division at the AVMA.

Local Anaesthetics

A Review and New Applications



Dr. Abdur Rahmaan Kadwa, BSc, BVSc
Resident in Veterinary Anaesthesia
Department of Companion Animal Clinical Studies
Faculty of Veterinary Science

In 1905, the discovery of procaine standardised its use as a local anaesthetic for almost 50 years. Its side effect profile was far from ideal. It had a short duration of action, low potency requiring large volumes to be administered which has increased its risk for systemic toxicity compared to modern local anaesthetics, poor tissue penetration and a proclivity for allergic reactions.[1,3] The development of lidocaine in the 1940's heralded a new dawn for local anaesthetics. In comparison to procaine, the duration of action of lidocaine was approximately 4 times longer, and demonstrated a more favourable safety profile.[1]

Pharmacology

Neurons have a resting membrane potential of approximately -90mV as a result of the distribution of charged ions within the extracellular and intracellular fluids. Upon application of a stimulus to the neuron, the propagation of an impulse along the length of the neuron is facilitated by the rapid influx sodium ions. These ions enter the neuron via voltage-gated sodium channels, raising the membrane potential to approximately +50mV. Depending on the membrane potential, these sodium channels exist in three conformations: resting, activated or inactivated.[6]

Local anaesthetics exert their effects by primarily interacting with voltage-gated sodium channels. Local anaesthetic drugs enter the neuron in an unionised state via the sodium channels or by diffusing through the cell membrane. The molecule dissociates within the cytoplasm and the ionised form of the molecule blocks the sodium channel from the inside of the neuron. (Fig. 1) The rapid influx of sodium ions into excitable cells, neurons, cardiac muscle is thus prevented, thus reversibly inhibiting the development and propagation of an action potential. Ion channels are preferentially blocked in an 'active' state; thus, stimulation of a nerve can hasten the onset of action of a local anaesthetic – a phenomenon known as 'use-dependant' or 'frequency-dependant' blockade.[2,3,6]

The chemical structure of a local anaesthetic drugs contains a benzene ring on one side and tertiary amine at the other end. The nature of the chemical group that links either end of the molecule dictates its metabolism. (Fig. 2) Ester-based drugs like procaine and benzocaine are locally broken down by plasma cholinesterases. In comparison, amide-based drugs such as lidocaine, ropivacaine and bupivacaine primarily undergo hepatic metabolism. Consequently, amide-based drugs are more likely to be cumulative due to their slower elimination.[2,3]

Three physicochemical properties of local anaesthetic molecules must be considered to better understand the unique characteristics of the individual drug. (Table 1)

1. Acid Dissociation constant

Considering that a drug can only enter a cell in its unionised form, the pKa (acid dissociation constant) will dictate the proportion of unionised drug available depending on the local environment. Local anaesthetics are weak bases with a pKa of 7.6-9.1. Hence, most agents will exist in the ionised form at physiological pH. The less the unionised fraction, the longer the onset of action. This effect of local tissue pH becomes especially relevant in injured tissues which have a lower than normal pH. Commercially available formulations are acidic, therefore adding bicarbonate may reduce the onset of action and pain on injection.[2,3]

2. Lipid Solubility

The potency of local anaesthetic drugs is related to its lipid solubility. Highly lipid soluble molecules penetrate cell membranes more efficiently and thus their effects are more pronounced. Furthermore, lipid solubility is also related to the drug's propensity to display side-effects.

3. Degree of protein binding

Lastly, the degree of protein binding to sodium channels dictates its duration of action. Local tissue binding also increases the duration of action by forming a local anaesthetic depot.[3]

Applications

Perhaps the most common application of lidocaine is its use as a local anaesthetic. Its application has expanded to a plethora of regional blocks of superficial nerves, infiltration into surgical wounds via soaker catheter, blockade of body cavities, intravenous regional anaesthesia, desensitisation of the larynx before intubation and most simply, the splash block.[4]

Due to its favourable toxicity profile, lidocaine can be administered intravenously for reasons other than local anaesthesia. Its affinity for sodium channels results in it being classified as a class Ib anti-arrhythmic drug.[4] It reduces the degree of depolarisation of cardiac myocytes by limiting the influx of sodium ions into the cell during early depolarisation; it also exhibits similar effects on the Purkunje fibres and thus may be administered for the treatment of ventricular arrythmias.[7]

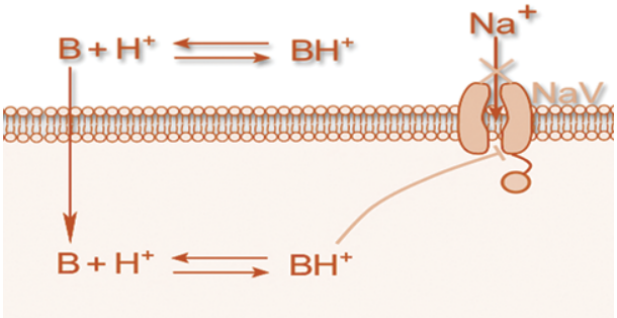


Figure 1. Local anaesthetics blocks the sodium channel on neuronal membranes from the inside

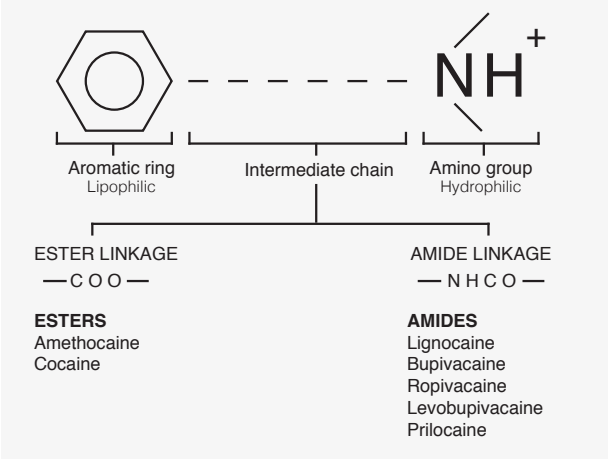


Figure 2: Representation of the structure of local anaesthetics, which affects their metabolism

In addition to its anti-arrhythmic activity, intravenously administered lidocaine provides visceral analgesia in a variety of species when injected intravenously .This analgesia is evidenced by the reduced requirements for inhalational anaesthesia. The mechanisms behind the analgesia are not completely understood, but are thought to be mediated by various receptor interactions in the dorsal horn of the spinal cord.[2,4]

Lidocaine also mediates anti-inflammatory effects by inhibiting the production of inflammatory mediators, reduction of leukocyte margination and platelet aggregation. Furthermore, lidocaine has the ability of both inhibiting the production of free oxygen radicals and scavenging them if they have already been produced.[2,7] Lastly, the anti-inflammatory effects of lidocaine is thought to contribute to its prokinetic

Drug	pKa	Protein binding (%)	Relative lipid solubility	Speed of onset	Duration of action
Procaine	9.0	6	1.7	Slow	Short
Lidocaine	7.7	64	43	Rapid	Intermediate
Bupivacaine	8.1	95	346	Slow	Long
Ropivacaine	8.1	94	115	Slow	Long

Table 1: Summary of the physicochemical characteristics of local anaesthetics (Reproduced in part from BSAVA Manual of Canine and Feline Anaesthesia and Analgesia)

effects. Lidocaine seems to exhibit prokinetic effects on intestines which have already been compromised, more so in cases of reperfusion injury.[2,4]

The suggested intravenous dose of lidocaine in dogs:
- 1mg/kg bolus over 5 minutes.
- Do not exceed 4mg/kg per hour.
- Administer at 0.3-0.6mg/kg/h as a constant rate infusion.
- Intravenous use not recommended in cats.

Ropivacaine and bupivacaine are long-acting amide-group local anaesthetics. Their use intravenously is not recommended due to a high potential for toxicity, bupivacaine more so than ropivacaine. They posses relatively similar physochemical properties when compared to each other. The onset of action of ropivacaine and bupivacaine is slightly longer than lidocaine due a higher pKa; their duration of action exceeds lidocaine. Bupivacaine may be slightly longer acting than ropivacaine due a higher degree of protein binding. Due to these factors, ropivacaine and bupivacaine are used exclusively for local anaesthesia.

Toxicity

Consequent to their mechanisms of action, local anaesthetic toxicity is manifested via the central nervous and cardiovascular systems. Symptoms of toxicity are dose dependant, initially affecting the central nervous system and then the cardiovascular system at higher doses.[2,4]

Toxicity usually occurs as a result of unintentional intravascular injections during perineural or neuraxial blocks. Peak plasma concentration will determine symptoms experienced Intravascular injection will cause the fastest rise in plasma concentration, whereas the amount of drug absorbed from the periphery depends on the total dose administered, the degree of vascularity of tissues and metabolic pathway of the drug in question. Therefore, rapid intravenous administration of a sub-toxic dose can cause toxicity symptoms. Additionally, different species also display varying sensitivities to the local anaesthetic drugs.[2,4]

In animals, the first sign of toxicity is usually sedation, followed by muscle fasciculations, seizures, cardiac conduction disturbances and finally complete cardiovascular collapse. Toxicity can be successfully treated by a lipid emulsion (example: Intralipid® 20%, Fresenius-Kabi). The mechanism by which lipid emulsion work for local anaesthetic toxicity is not known, it is suspected to provide a 'lipid-sink' which draws in the drug.[2,4]

Several local anaesthetic drugs, namely the ester-based compounds and prilocaine have been associated with methaemoglobinaemia. The metabolites these drugs cause oxidation of iron molecule with the heme ring of haemoglobin. Felines are thought to be especially prone to this phenomenon.[2]

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Drug	Peak effect (minutes)	Duration of effect (hours)	Toxic dose		Maximum safe dose	
Lidocaine	2-5	1-2	Dog Cat	22mg/kg IV 11 mg/kg IV	Dog Cat	10 mg/kg 6 mg/kg
Bupivacaine	5-10	~2	Dog and cat	4mg/kg IV	Dog and cat	2mg/kg
Ropivacaine	5-10	~2	Dog	4mg/kg IV	Dog	3 mg/kg

Table 02: Reproduced in part from BSAVA Manual of Canine and Feline Anaesthesia and Analgesia

GDV: It Doesn't Have to Make Your Stomach Turn

Bronwyn Fullagar, BVSc, MS, DACVS

Whether you perform surgery yourself or refer your patient to a specialty veterinary facility, aggressive stabilisation can make all the difference.

Just before closing time, a large-breed dog arrives at your clinic with a history of nonproductive retching, lethargy and a progressively distending abdomen. You know gastric dilatation-volvulus (GDV) is likely, but where do you begin? Should you refer the patient straightaway? Will you need to perform surgery yourself?

If this scenario makes your palms sweat and your adrenals squeeze, fear not. GDV is a true medical and surgical emergency, but with a logical diagnostic and treatment approach and a focus on thorough preoperative stabilisation, a good outcome can be achieved in the majority of cases. Even if you intend to refer the case to a specialist or emergency facility, the early stabilisation procedures you perform can make a big difference to your patient's success, and your clients will appreciate your help in their decision-making process as they grapple with a very stressful situation.

Triage: What's the patient's status?

Patients that present with a history suggestive of GDV should be brought immediately to the treatment area and assessed as soon as possible by a veterinarian. In the meantime, the owners should be asked to sign a consent form authorising initial stabilisation procedures and diagnostics. Remember that although middle-aged, large- and giant-breed, deep-chested dogs are the poster children of GDV, dogs of any breed between 10 months and 14 years of age-and even cats-may be affected.

Patients with GDV are often hypersalivating with a painful abdomen, and a tympanic quality can be auscultated on abdominal percussion. Patients may present in a critical condition with an unstable cardiovascular status. Heart rate, respiratory rate, temperature, blood pressure and pulse quality should be evaluated immediately.

Stabilisation: Improve perfusion and tissue oxygenation

The first priority in treating patients with suspected GDV is to stabilise their cardiovascular status. The sooner you treat shock and restore oxygen delivery to the tissues, the less likely it is that the patient will require resection of necrotic gastric wall and the better the overall prognosis. Although it can be tempting to perform radiographs immediately, this step should be postponed until after initial resuscitation.

Use initial physical exam findings to guide your stabilisation efforts and be aware that patients may present in varying degrees of shock. Those presented earlier in the disease process will have signs consistent with hyperdynamic, hypovolemic shock due to their blood volume being restricted to the caudal half of their body by the enlarged stomach compressing the caudal vena cava. These dogs will be tachycardic and tachypnoeic, with normal femoral pulses, pale mucous membranes and a slow capillary refill time.

As the syndrome progresses, dogs will develop injected mucous membranes and weak femoral

pulses-signs of endotoxaemic shock. Eventually, patients will decompensate to a point where they are hypotensive and bradycardic with white mucous membranes; at this stage they often present laterally recumbent in lacteral recumbency.

Fluid resuscitation is the most important component of GDV patient stabilisation. Place two venous catheters in the **cranial half of the body** (cephalic or jugular veins can be used). Select the largest size catheters possible to allow rapid delivery of fluids. Provide crystalloid fluid therapy (e.g. lactated Ringer's solution, Plasmalyte or 0.9% sodium chloride) initially at a dose of 40-90 ml/kg over the first 30 to 60 minutes. **As a rule of thumb, a large-breed (30-kg) dog will require 1.5 to 3 L crystalloids, while a giant-breed (60-kg) dog will require 2.5 to 5.5 L.** Alternatively, you can use colloid fluids (e.g. hydroxyethyl starch) at a dose of 10-20 ml/kg combined with crystalloids at 10-40 ml/kg. Titrate fluids to effect until you note improvements in heart rate, respiratory rate, mucous membrane colour and CRT, and blood pressure.

Patients with GDV can be painful, so administration of opioid analgesia is helpful. Pure mu agonists (fentanyl, methadone, hydromorphone) are preferred over agonist-antagonists (buprenorphine) or partial agonists (butorphanol), as these can interfere with the effects of pure mu agonists administered perioperatively. **Nonsteroidal anti-inflammatory drugs (NSAIDs) are always contraindicated in cases of GDV.**

Gastric decompression is an important part of stabilisation as it relieves pressure on the caudal vena cava, thus allowing blood to flow from the caudal half of the body back to the heart (improved preload). It also helps to improve gastric perfusion and patient comfort. Decompression can be performed either via orogastric intubation or trocharisation. Although orograstric intubation is more effective in decompressing the stomach, it can be challenging in awake patients and is generally reserved for those that are sedated or recumbent.

Take care to protect the airway; intubation may be required to avoid aspiration pneumonia. Premeasure a large-bore, smooth tube to the last rib and lubricate it. Note that often the tube cannot be passed past the lower esophageal sphincter due to the torsion, and care must be taken not to force the tube as esophageal perforation is possible.

If orogastric intubation is not possible or not safe, you can perform trocharisation over the region of greatest gastric tympany, usually over the right lateral abdomen caudal to the last rib. Clip and aseptically prepare the skin, insert a 14- to 18-ga over-the-needle catheter, and remove the stylette. You may need to repeat trocharisation if surgery is delayed as the stomach will continue to fill with gas. (Trocharisation often facilitates the passing of a stomach tube- .Ed)

An electrocardiogram should be performed on GDV patients prior to surgery. Up to 70% of dogs with GDV develop cardiac arrhythmias,[3] which are frequently ventricular in origin.[1] Arrhythmias may occur up to 72 hours postoperatively. Patients with ventricular tachycardia, R on T complexes, multiform ventricular premature complexes, or clinical hypotension related to their arrhythmia should be treated with lidocaine (2 mg/kg slow intravenous bolus, then constant rate infusion [CRI] of 25-80 µg/kg/min).See Journal Scan and local and anaesthetic article on the use of lidocaine in these patients

Diagnostics: GDV, GD or something else?

A right lateral abdominal radiograph is the view of choice to diagnose GDV and is important to differentiate dogs with gastric dilatation from those with true dilatation and volvulus. In the latter, a soft tissue band will be present between the distended pylorus and the fundus-the so-called "double bubble" or "Popeye arm." Dogs with gastric dilatation without volvulus will have a distended stomach without the soft tissue fold between the two regions.

The radiograph should also be evaluated for free peritoneal gas, which may indicate gastric perforation, a poor prognostic indicator. Note that patients that received gastric trocharisation prior to radiographs may develop some scant free peritoneal gas in the absence of perforation. Thoracic radiographs may be indicated for patients in which aspiration pneumonia is suspected or for elderly patients in which GDV may be secondary to neoplastic causes.

Initial bloodwork should include packed cell volume and total proteins, blood glucose and venous blood gas with electrolytes if available. In patients that will undergo surgery, complete blood count and serum biochemistry are indicated to assess platelet number, leukogram, liver and kidney function, and albumin levels. Coagulation testing (PT and aPTT) can help to identify patients that are coagulopathic or trending towards disseminated intravascular coagulation (DIC) preoperatively.

Decision-making: 'Should I pursue surgery for my dog?'

Ultimately, patients with GDV require surgery after medical stabilisation. The decision to proceed with surgery can be difficult for owners, and family veterinarians play an important role when they're able to provide accurate information about the pet's prognosis to help with the process. Historically, GDV was thought to carry a poor prognosis, but the most recent studies report improved rates of survival of 73% to 90%.[2-4]

The variable most consistently associated with a poor prognosis is gastric necrosis,but unfortunately

there is no way of definitively determining whether gastric necrosis is present until surgery is performed.[1-5] However, several prognostic factors have been identified that can offer insight into whether your patient is likely to have a successful outcome.

- In general, dogs that present with clinical signs of less than six hours' duration and those that "walk in" to the clinic do better than those that present laterally recumbent.
- Dogs that present with sepsis, gastric perforation or DIC have a worse prognosis.[1,2,4,5]
- In one study, dogs with an increased amount of time between presentation and surgery had a decreased mortality rate, which speaks to the importance of aggressive preoperative stabilisation.[3]
- Blood lactate has been evaluated in several studies as an indicator of gastric necrosis. Normal canine lactate is <2.5 mmol/L, and increased lactate indicates poor tissue perfusion. If a blood lactate meter is available, measuring trends in lactate (rather than absolute values) can reflect the success of your stabilisation efforts. A change (decrease) in lactate of greater than 42.5% after fluid resuscitation was associated with a survival rate of 100% in one study.[6]

Surgery: Derotate, assess viability, pexy

Once you've made the decision to pursue surgery, the next consideration is whether to refer the case to a specialty or 24-hour facility or perform surgery at your own clinic. There are a number of factors that affect this decision, but referral is strongly recommended in all cases where gastric necrosis is suspected. Gastrectomy greatly increases the technical difficulty of the surgery, surgical stapling equipment available in specialty practices improves the efficiency and safety of the procedure, and postoperative care is significantly more intensive in patients that have undergone gastrectomy. Of course it's not possible to know for sure preoperatively that a gastrectomy will be required, but using the prognostic indicators above can give a reasonable idea of which cases will be more complicated.

1. Anaesthesia

Anaesthesia for GDV patients should prioritise maintaining blood pressure and tissue oxygenation and avoiding arrhythmogenic agents. Therefore, alpha-2 agonists (dexmedetomidine, medetomidine), acepromazine and ketamine should be avoided. If possible, patients should remain in lateral recumbency for as long as possible during preparation for surgery to avoid additional pressure on the caudal vena cava, and preoxygenation is beneficial.

A common protocol is premedication with a benzodiazepine (e.g. midazolam or diazepam) and an opioid (e.g. hydromorphone or fentanyl), induction with propofol or alfaxalone, rapid intubation and endotracheal tube cuff inflation, and maintenance with isoflurane and oxygen. A fentanyl CRI of 5-10 µg/kg/h can be helpful to reduce the minimum alveolar concentration of isoflurane required. A lidocaine CRI can be administered concurrently with fentanyl-this has the dual effect of treating cardiac arrhythmias and providing analgesia. Perioperative antibiotics are indicated in all cases of GDV, and a first-generation cephalosporin (e.g. cefazolin) or ampicillin-Beta Lactam is appropriate. Administer the first dose at anesthetic induction and then every 90 minutes intraoperatively.

2. Step-by-step surgical procedure

Be sure to clip a wide area-the caudal half of the thorax and the entire abdomen to the pubis should be prepared aseptically. The surgeon stands to the patient's right side. Perform a ventral midline celiotomy from the xyphoid to the fourth mammary glands or prepuce. Be prepared to encounter a haemoabdomen, as avulsion of the short gastric arteries from the greater curvature of the stomach has often occurred. In most cases, haemorrhage is self-limiting or can be addressed after gastric derotation. In patients with GDV, the greater omentum will be draped over the stomach upon entering the abdomen. Occasionally the stomach has derotated itself, in which case you will encounter the ventral surface of the stomach and spleen first, without omentum covering them.

In most cases, the stomach has rotated 180 to 270 degrees. To derotate the stomach, it may be necessary to first decompress it, which can be performed either by having an assistant pass an orogastric tube or by trocharising the stomach with a needle intraoperatively. Then use your right hand to reach the pylorus, which is usually located dorsal to the esophagus on the patient's left side. With your left hand, form a fist and push the distended fundus (malpositioned on the dog's right side) dorsally while you pull the pylorus ventrally and to the right. The spleen will usually derotate with the stomach.

3. Evaluation of abdominal cavity

After derotation, perform complete exploration of the abdomen. Residual hemorrhage from the short gastric arteries may need to be controlled with ligatures. Palpate the stomach carefully for a gastric foreign body-gastrotomy is required in this case. Assess the colour of the spleen, splenic engorgement is expected and the spleen should return to a dark purple colour following derotation. If the spleen is black in colour or thrombosis of the splenic artery has occurred (or both), a splenectomy is indicated.

4. Gastric wall assessment

Next, assess the gastric wall. This is a subjective assessment; accuracy improves with surgeon

experience. When gastric wall colour, thickness, peristalsis and bleeding on incision are evaluated in combination, accuracy of assessment is about 85%.[7] The most common region affected by ischemic injury is the fundus along the greater curvature. Be sure to evaluate the stomach all the way to the lower esophageal sphincter. If the gastric wall remains black without improvement following reperfusion, if the serosal surface is pale greenish or grey, if there is absence of the normal mucosal "slip" or the wall is very thin, or if the serosal surface does not bleed when a partial thickness incision is made, partial gastrectomy is required. Gastrectomy can be performed by either a cut-and-sew technique with an inverting suture pattern, the use of surgical stapling equipment, or invagination of the ischemic gastric wall. The procedure is well described elsewhere.[8]

5. Gastropexy

The next step is gastropexy. Incisional gastropexy is technically simple and highly effective, with reported rates of GDV recurrence almost 0%.[9] If you have performed a gastrectomy, lavage the abdomen with warm sterile saline and suction it, then exchange gloves and instruments for a sterile set. Gastropexy takes place between the pylorus and the patient's right abdominal wall, so it's easiest if the surgeon moves to stand on the patient's left side. The pexy site on the stomach is located 2 to 3 cm oral to the pylorus, on the ventral surface, between the greater and lesser curvatures.

The pexy site on the body wall is located in the right transverse abdominis, caudal to the last rib, parallel to the skin incision and approximately one-third of the distance from ventral to dorsal. In some larger dogs it may be necessary to place the pexy slightly more cranial, over the last ribs, but take care not to pass the insertion of the diaphragm on rib 11 to avoid an inadvertent thoracotomy!

It is helpful to have an assistant stand on the patient's right side, grasp the right abdominal wall along the linea alba with towel clamps and elevate it toward the ceiling for better visualisation. A description of one way to perform the procedure is below:

- Place two partial-thickness (seromuscular layer only) stay sutures of 2-0 PDS 4 to 5 cm apart at either end of the proposed pexy site on the stomach (leave about 3 cm between the pexy site and the pylorus). Knot the stay sutures to the stomach and leave the needles attached, with a long suture tag.
- Make a partial-thickness (seromuscular) incision in the gastric wall between stay sutures using a new No. 15 scalpel blade. The submucosa can be visualized, but do not enter the gastric lumen.
- Suture each 2-0 PDS stay suture to the right transverse abdominis at the site of proposed pexy. Leave the needles attached to the sutures.
- Make a full-thickness incision in the transverse abdominis between the stay sutures.
- Suture each seromuscular layer of gastric wall to the cut edge of the transverse abdominis, using a simple continuous appositional pattern (2-0 PDS). Suture the dorsal edge first.

Postoperative care: Continue support and resuscitation; monitor for complications

Patients will require 24-hour care postoperatively, so transfer to a referral facility may be necessary. Continue fluid resuscitation and address electrolyte imbalances. Continue analgesia with pure mu agonists until the patient is able to tolerate oral analgesia. NSAIDs are contraindicated. You can continue to provide a lidocaine CRI to provide analgesia as well as to treat cardiac arrhythmias. Continuous ECG is helpful to screen for ventricular arrhythmias, which may persist for 48 to 72 hours postoperatively. Discontinue antibiotics within 24 hours of surgery unless gastric necrosis was present.

Patients can be fed within 12 to 24 hours of surgery, either via nasogastric (NG) feeding tube or orally. Small meals of a bland or gastrointestinal diet are ideal. Prokinetic therapy (e.g. metoclopramide or cisapride) and intermittent suctioning of the NG tube may be necessary in cases of ileus, and an antiemetic (e.g. maropitant) is helpful to prevent vomiting secondary to ischaemic gastritis. H_2 antagonists (e.g. famotidine) or proton pump inhibitors (e.g. omeprazole, pantoprazole) can be administered to treat gastric ulceration.

Major complications of surgery following GDV include peritonitis, sepsis and DIC.[8] Peritonitis is most often due to ongoing gastric necrosis that was not adequately assessed initially and repeat surgery is required.

Prophylactic gastropexy: Prevention is better than cure!

Did you know that Great Danes have a 42% of developing GDV in their lifetime?[10] Other large and giant breeds have a lifetime risk of 4% to 37%.[11] Prophylactic gastropexy in predisposed dogs reduces their mortality rate by 29 times,[3,12] and incidence of GDV after incisional gastropexy is nearly 0%.[12]

Gastropexy can be performed at the time of spay or neuter and is recommended once dogs are close to adult size. Pexy can be performed either via a traditional "open" approach (as described above) or via laparoscopy or laparoscopic-assisted techniques. Minimally invasive approaches result in less postoperative pain and a faster return to normal activity than open approaches and are equally as effective.[12,13] Next time you meet a puppy of a predisposed breed or are performing abdominal surgery for another reason on a large- or giant-breed dog, consider

recommending prophylactic gastropexy-it may just save a life.

Approach your next case with confidence

GDV is a true medical and surgical emergency, but most patients can do well with aggressive early stabilis ation and prompt surgical care. Taking the time to stabilise your patient prior to surgery will greatly improve postoperative outcomes, whether you plan to perform surgery yourself or transfer the patient to a referral facility. Palpating as many normal stomachs as possible and performing gastropexy in a prophylactic setting first will improve your technical expertise and confidence if and when you are required to perform surgery on a clinical GDV case.

Client education plays a major role in both the prevention of GDV through prophylactic gastropexy and rapid identification of the condition, which allows for timely veterinary care. Treating patients with GDV can be challenging, so don't hesitate to seek advice from your local emergency or specialist veterinary facility.

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View complete list of references online: www.vetlink.co.za

Anaesthetic (ASA) scoring system

An ASA risk is a 1-to-5 score adapted for animals from human medicine's American Society of Anesthesiologists. The system is based on the patient's overall health, not the procedure being performed.

ASA Classifications

CLASS 1: Minimal risk with a normal healthy patient with no underlying disease.
CLASS 2: Slight risk with slight to mild systemic disease: neonates, geriatrics, obesity.
CLASS 3: Moderate risk, obvious systemic disease. Anaemia, moderate dehydration, fever, low-grade heart murmur or cardiac disease.
CLASS 4: High risk with severe, systemic, life-threatening disease. Severe dehydration, shock, uraemia, toxaemia, high fever, uncompensated heart disease, uncompensated diabetes, pulmonary disease, emaciation.
CLASS 5: Extreme risk, moribund; patient will probably die with or without surgery. Advance cases of heart, kidney, liver or endocrine disease; profound shock, severe trauma, pulmonary embolus, terminal malignancy.
CLASS E: Emergency. Can be attached to each class in case of emergency surgery.

Assigning an ASA score is relatively subjective, but it should help to identify patients who needs more pre-operative testing, pre-operative stabilisation, a more experienced anaesthesia technician and / or more advanced intra-operative monitoring.

A Spanish study1 evaluated over 2,000 dogs that underwent anaesthesia. A death rate of just over 1 % (26 patients) was recorded. A direct correlation was found between the patients' ASA score and the death rate:

- 0.3 % of fatalities involved ASA 1 and 2 scores.
- 1.7 % of fatalities had ASA 3 scores.
- 13 % of fatalities were ASA 4 or 5 patients.

About 25% of the fatalities happened intra-operatively while the remainder occurred in the post-operative period. This emphasises that patients should be monitored closely after extubation. Walking away from patients as soon as they're extubated is so last century.

Journal Scan : Effect of Intra-operative CRI of lidocaine on short-term survival of dogs with septic peritonitis: 75 cases (2007 – 2011)

Bellini L, Seymour CJ. Journal of the American Veterinary Medical Association, 2016, 248:422 - 429.
Summarised: By Dr Liesel van der Merwe BVSc MMedVet (Med) Small Animals

Why they did it?

Distributive shock, characterised by tachycardia, hypotension and a decrease in systemic vascular resistance secondary to the severe inflammatory response frequently develops in patients with septic peritonitis. This makes these patients poor surgical candidates due to their risky anaesthesia. Survival rates for dogs with septic peritonitis vary from 50 – 80%. Death is caused by multiple organ dysfunction. Endotoxins released during sepsis cause hyperinflammation which activates the coagulation cascade and causes leukocyte and platelet adhesion to the endothelium, disrupting microcirculation, which, in humans, is correlated to a poor outcome.

Inhalation anaesthetics exacerbate haemodynamic instability by decreasing systemic vascular resistance in a dose dependent manner , and the intraoperative use of opiods will reduce the requirement of the inhalation anaesthetic. However opiods can cause a vagally mediated bradycardia - which is compensated for by a baroreceptor mediated response to increase systemic blood pressure. In patients with sepsis, this compensatory mechanism in insufficient which can impair tissue perfusion.

Lidocaine supresses in vitro platelet and leukocyte aggregation and p-selectin expression, which are sensitive indicators of sepsis. Lidocaine has analgesic and anti-inflammatory, analgesic and anti-endotoxic properties as an IV infusion at 33 – 50 ug/kg/min in rats, rabbits and horses. In experimentally induced septic peritonitis in dogs IV infusion of lignocaine decreased mortality rates.

What they did

A retrospective study of hospital records over a 5-year period of 75 dogs which matched the inclusion criteria. Dogs were included in the study if they had a septic peritoneal effusion and underwent a laparotomy and also if a perforation of a gastrointestinal or uterine wall was detected on an exploratory laparotomy in the absence of an effusion.

Two groups were evaluated: Group O (n=33) which only received opiods and group L (n=42) which received opiods and lidociane (at the discretion of the anaesthetist). The lidocaine was given at 2mg/kg IV bolus and then a CRI of 50µg/kg/min during surgery.

The data for each dog was divided into preoperative, intra-operative and post-operative periods.

- Variables of the pre –operative period were: heart rate, respiratory rate, body temperature, type of IV fluids administered and time from admission to anaesthesia induction.
- Variables for the intra-operative period were: ASA status immediately prior to anaesthesia, type of opiod administered, type and amounts of IV fluids number of IV boluses, and number of dogs receiving vasopressors and inotropes, heart rate, duration of hypotension, body temperature at extubation and anaesthesia duration.
- Variables for the post-operative period, defined as 48 hours, were: types of analgesia drugs used, patients receiving lidocaine CRI, type and amount of IV fluids, and number of dogs receiving cardiovascular support (inotropes and vasopressors).

Limitations of the study include a fairly small sample size, non-random nature of the administration of lidocaine, lack of scoring system for the severity of sepsis and the retrospective nature of the study.

What they found

None of the pre-operative outcomes assessed differed significantly between the groups. Only the number of IV boluses of synthetic colloids administered differed, being higher in the L group. The group L dogs did receive more IV boluses of a synthetic colloid - but this was not associated with the short term odds of survival post operatively. The heart rate and incidence of hypotension was the same in each group during the intraoperative period. In humans small blood vessel perfusion is a better predictor of survival than mean arterial pressure.

The proportion of dogs which remained alive at 48 hours after surgery was significantly (p=0.036) greater for group L (n = 35/32, 83.3%) than group O (n = 20/33, 60.6%). When survival was compared to the ASA status prior to surgery all dogs in group O which were classified as ASA V died within 48 hrs whereas only 5/16 (31.3%) dogs in group L , classified

preoperatively as ASA score V died (p< 0.001). There were no significant differences in the groups with preoperative ASA scores of IV and III. Results of a multivariate logistical analysis showed that a lidocaine infusion significantly increased the odds of survival at 48hrs after surgery (OR 8.77) when administered during the intra-operative period.

Some dogs received addition CRI of lidocaine at 30µg/kg/min for analgesia for 6 – 12 hours post operatively.

These patients showed a decreased odds of survival (OR , 0.13) 48 hour post op.

Take Home Message

Results of this study indicated that a CRI of lidocaine at 50ug/kg/min can be administered with an opiod as part of a balanced anaesthetic protocol during the intraoperative period in dogs with septic peritonitis and improves the short term (48hrs) survival rates.

Journal Scan: Evaluation of Lidocaine treatment and risk factors for death associated with gastric dilation and volvulus in dogs: 112 cases (1997 – 2005)

Buber T, Saragusty J *et al.* Journal of the American Veterinary Medical Association, 2005; 230: 1334-1339.
Summarised by Dr Liesel van der Merwe BVSc MMedVet (Med) Small Animals

Why they did it?

In GDV rapid accumulation of gas in the stomach, gastric displacement increased intra-gastric pressure and decreased venous return occur. GDV is characterised by cardiogenic shock.

The most serious complications of GDV are ischaemic reperfusion injury and resultant SIRS, including hypotension, acute renal failure, DIC and cardiac arrhythmias. The reported mortality is high (15 – 28%) and gastric necrosis and high serum lactate concentration are predictors of post-operative complications and death underlining the importance of ischaemic hypoperfusion. Initial treatment goal is reversal of shock by increasing venous return and reperfusion of ischaemic tissue. However reperfusion will in turn cause tissue damage by reactive oxygen species ("free oxygen radicals") circulation which were formed in the ischaemic tissues. When ROS interact with cells they cause lipid peroxidation, often leading to cell death. Possible positive effects of lidocaine in reducing ischaemic perfusion injury include scavenging oxygen radical species, decreasing release of superoxide from granulocytes, decreasing neutrophil activation and tissue migration and endothelial damage.

What they did

The case files of all surgically treated dogs presenting for GDV in the allotted timespan (n=112) were evaluated. Dogs were divided in 2 groups: those pre-surgically treated with lidocaine (n=51), and a non-treated group (n=47).

In the lidocaine treated group - 2 protocols were used - an IV bolus at 2mg/kg followed by a CRI of 50ug/kg/min for at least 3 hours (n=8/51), or a CRI at 50ug/kg/min without the initial IV bolus (n=43/51). DIC was describes as a platelet count < 150 000cells /ul and 2 of ; > 25 % prolonged PT/PTT of evidence of petechial. Cardiac complictaions were defines as VPCs, Ventricular tachycardia , atrial fibrillation and idioventricular rhythm. ARF was diagnosed when dogs had a creatinine > 2mg/dl after 24 hours of fluid administration.

What they found

Gastric decompression prior to surgery was performed in 68% of dogs and did not improve survival rate.
Overall mortality rate was 26.8%. Gastric wall necrosis was detected in 27/108 (25%) and was a significant risk factor for death with a mortality of 59.3% (p<0.001) compared with a mortality of 16% in the remaining dogs (81/108). Unresected suspected gastric necrosis (n=6/27) had a 100% mortality rate versus 47 % in the patients (n=21/27) where surgical resection was performed.

Coagulopathy (DIC) and cardiac arrhythmias, neither a risk factor for death were diagnosed in 18/112 (16.1%) and 51/112 (45.5%) patients respectively. (The definition of DIC in this study is quite broad - this accounting for the no-influence on survival - a better term is coagulopathy - vdM). Arrythmias are a result of myocardial ischaemia, due to hypo-perfusion, lactic acidosis and electrolytre imbalances. Myocardial depressant factor is also produced by the pancreas in ischaemic conditions. A study has shown that

dogs with GDV had significant increases in cardiac troponins after 24 and 48 hours.

ARF was diagnosed in 9/112 (9%) was a significant (p=<0.005) risk factor for death resulting in a 66.6% mortality versus 23.3% in dogs without ARF. Bacterial translocation , endotoxaemia, sustained reduction of peripheral perfusion and ischaemic injury all contribute to ARF.

The mean duration of lidocaine CRI was 20.8 hrs (3 – 72) . There were no differences in prevalence of complications between the treated and untreated group. There was a significant overrepresentation of German shepherd dogs (28.6%) and Great Danes (17%). Dogs with a rectal temperature > 38°C were significantly more likely to survive (40.9%) than those with a temperature of <38° (14.9%) (p=0.019). Low body temperature reflects the severity of venous obstruction caused by the dilatation and rotation of the stomach and consequent shock.

Mortality rate was significantly higher in dogs with a timelag of ≥ 5 hours from commencement of clinical signs to admission. It is likely that severity of gastric wall necrosis, accumulation of radical oxygen species and resultant ischaemic injury correlates with the duration of the GDV and vascular obstruction.

Take home message

The mortality rate in this study is similar to that of previous studies. No significant differences in mortality rates and prevalence on postoperative complications were found between the two groups. Ischaemic reperfusion injury has a major role in GDV complications and post-operative monitoring and management is imperative.

Lidocaine treatment was initiated in all dogs prior to surgical repositioning of the stomach but AFTER initiation of medical treatment and gastric decompression. It is thought that by this time reperfusion injury would have already become established.

Additionally the treatment protocols without the initial IV bolus may have been insufficient. The onset of action of lidocaine after an IV bolus is usually 2 minutes, with a duration of action of 10 – 20 minutes. If a lidocaine CRI is started without the initial IV bolus, it may take up to an hour to reach its therapeutic concentrations and this protocol was used in 84% of patients. This may explain why there was not a positive effect on survival in this study as the drug reached effective concentrations too late in the ischaemic injury cascade.

CPD Questions

AC/0028/20

GDV: It Doesn't Have To Make Your Stomach Turn

Web-based: www.cpdsolutions.co.za - choose Onlinevets/VET360. SMS number available through website.
*CPD reserved for Vet360 subscribers (R360 per annum) Call 012 346 1590 for assistance SMS code a90165

The CPD questions are only based on the GDV article, however, there is important information contained in both Journal scans as to the management of this condition in dogs .

01. Which one of the statements listed below regarding shock in GDV syndrome is CORRECT?

a. Hyperdynamic shock occurs due to blood being restricted to the caudal half of the body
b. Injected mucous membranes and weak femoral pulses are signs of hyperdynamic shock
c. Hyperdynamic shock occurs due too low systemic blood pressure
d. Weak femoral pulses are found in hyperdynamic shock
e. Dogs presented later in the disease process will have signs of hyperdynamic shock.

02. Which one statement regarding fluid therapy in a GDV patient is INCORRECT?

a. Fluid resuscitation can be estimated at about 5 – 10% of patient mass.
b. Catheters should only be placed into the cranial part of the body
c. Colloids can be used at 10 – 20ml/kg for resuscitation
d. The initial dose of crystalloids is a maximum of 40 ml /kg over the first 30 – 60 minutes.
e. Rapid IV delivery is required - free flow is preferable to fluid pumps in large breed dogs

03. Which one of the statements below is INCORRECT?

a. Patients with GDV can be painful,
b. Pure mu agonists (fentanyl, methadone, hydromorphone) are preferred
c. Agonist-antagonists (buprenorphine) or partial agonists (butorphanol) are less effective than pure Mu agonists

d. Butorphanol can interfere with perioperative anaesthesia
e. Non-steroidal anti-inflammatory drugs (NSAIDs) are indicated as part of analgesia

04. Which one of the following statements is INCORRECT?

a. Gastric decompression relieves pressure on the caudal vena cava,
b. Gastric decompression improves cardiac pre-load (improved preload).
c. Reperfusion injury is a result of decompression
d. Orogastric intubation can generally be performed in the awake patient
e. Thocharization can be performed, usually over the right lateral abdomen caudal to the last rib.

05. Which one of the following statements regarding imaging in GDV is CORRECT?

a. A left lateral abdominal radiograph is the view of choice to diagnose GDV
b. It is important to radiographically differentiate dogs with gastric dilatation volvulus from those with splenic torsion
c. In GDV a soft tissue band will be present between the distended pylorus and the fundus-the so-called "double bubble" or "Popeye arm", "boxing glove".
d. Free peritoneal gas, is an expected radiographic sign in cases with GDV.
e. Thoracic radiographs are not indicated.

06. Which one of the statements below regarding prognosis in GDV is INCORRECT?

a. The variable most consistently associated with a poor prognosis is gastric necrosis
b. The presence of gastric necrosis can be determined by an increased lactate level on presentation.
c. Dogs that present with clinical signs of less than six hours' duration do better
d. Dogs with increased time span and thus aggressive preoperative stabilization between presentation and surgery had a decreased mortality rate
e. A decrease of > 42.5% in lactate after fluid resuscitation was associated with a survival rate of 100% in one study

07. Which one of the anaesthetic protocols below is most correct for patient with GDV?

a. Domitor pre-med with morphine analgesia and propfol and inhalation maintenance
b. ACP premed with morphine for analgesia, IV antibiotics and ketamine induction and inhalation maintenance
c. A benzodiazepine pre-med with fentanyl CRI analgesia , alfaxan induction and maintenance with propofol
d. A benzodiazepine pre-med with fentanyl CRI analgesia ,IV antibiotics, propofol induction and maintenance with inhalation anaesthesia
e. A benzodiazepine premed with fentanyl analgesia and ketamine induction with inhalation maintenance

08. Which one of the statements below regarding assessment for gastric wall necrosis is INCORRECT?

a. When gastric wall colour, thickness, peristalsis and bleeding on incision are evaluated in combination, accuracy of assessment is about 85%.
b. The most common region affected by ischemic injury is the pyloric region along the lesser curvature.
c. If the gastric wall remains black without improvement following reperfusion made, partial gastrectomy is required.
d. If the serosal surface does not bleed when a partial thickness incision is made, partial gastrectomy is required.
e. If the serosal surface is pale greenish or grey, and if there is the normal mucosal "slip" is absent, partial gastrectomy is required.

09. Which one statement listed below regarding gastropexy is most CORRECT?

a. Incisional gastropexy is technically simple and highly effective, with reported rates of GDV recurrence only 15%.
b. The pexy site on the stomach is located 2 to 3 cm oral to the pylorus, on the ventral surface, between the greater and lesser curvatures.
c. Gastropexy takes place between the pylorus and the patient's right abdominal wall, so it's easiest if the surgeon moves to stand on the patient's right side.
d. The pexy site on the body wall is located in the left transverse abdominis, caudal to the last rib
e. The pexy site is parallel to the skin incision and approximately two – thirds of the distance from ventral to dorsal.

10. Which one of the statements below regarding post-operative care in patients with GDV is most CORRECT?

a. Continue analgesia with a partial mu agonist until the patient is able to tolerate oral analgesia
b. Continue analgesia with a pure mu agonist until the patient is able to tolerate oral anti – inflammatory therapy
c. Continue antibiotics for 3 days after surgery
d. Patients should only be fed within 12 - 24 hours after surgery, either via nasogastric (NG) feeding tube or orally.
e. H_2 antagonists or proton pump inhibitors are never indicated.

IMV Imaging Insights

Thrombosis and Thromboembolism in Small Animal Patients

Focus on an Ultrasound Diagnosis

Dr Kirstin Dinham BVSc MRCVS in collaboration with the IMV Imaging Clinical Team

Background, Causes, Clinical Findings

Thrombosis and thromboembolism are well recognised conditions in dogs and cats. Though rare in dogs, accounting for less than 0.0005% of hospital admissions in a ten-year study period,[1] aortic thrombosis has been associated with many conditions that predispose the patient to a hypercoagulable state, such as neoplasia, hyperadrenocorticism, hypoadrenocorticism, hypothyroidism, diabetes, cardiac disease, endocarditis, protein losing nephropathy, spirocercosis and recent corticosteroid administration.[2]

Aortic thromboembolism in cats is relatively commonly encountered, and although it can be caused by any condition causing a hypercoagulable state, cardiac disease (HCM) is the most common underlying cause[3]. The primary thrombus usually forms in the left atria and can become quite sizeable, with the resulting thrombo-emboli tending to lodge in the aorta or its branches. When this occurs at the aortic trifurcation (the 'saddle') both pelvic limbs will be affected, which is the most common presentation in cats.

The clinical signs associated with aortic thromboembolism are typically caused by acute ischaemia of the tissue supplied by the occluded artery, and classically include the 'five "P's"'; pulselessness, pain, pallor, paresis and poilkilothermia[4]. Differential diagnoses for acute loss of limb function should also include spinal cord disease, peripheral neuropathies and acute intracranial disorders.

In both dogs and cats however, one limb only may be affected. This can be more of a challenge diagnostically and may result in underdiagnosis. Thrombosis or thromboembolism should always be considered in cases of unilateral limb lameness (usually hind limb), particularly when associated with signs of ischemia. Non-appendicular locations such as renal, cerebral, mesenteric or splenic arteries can also be affected.[4]

Diagnostics

With any case with suspected thromboembolism, a full work up to screen for underlying causes is important for patient prognosis and appropriate treatment. Increased suspicion of a thromboembolism can be obtained by acquiring a blood sample to measure serum glucose or lactate in the body part(s) distal or peripheral to the suspected thrombus and comparing it to a central sample obtained proximal to the suspected thrombus[5]. The glucose levels from the sample distal or peripheral to the thrombus will typically be lower than those from the central sample whereas blood lactate level will be higher in a distal or peripheral sample as compared to a central sample.

Imaging can be used to obtain a definitive diagnosis of thromboembolism and it is important in order to rule out other causes of vascular occlusion such as neoplastic invasion or severe vasculitis. Many imaging techniques, such as radiographic or computed tomographic (CT) angiography, scintigraphy and magnetic resonance imaging (MRI) can be considered for directly diagnosing thromboembolism, especially when additional sites of thromboembolism are suspected. However, ultrasound machines with doppler capability are widely available and are a practical, cost effective, non-invasive and rapid diagnostic modality. Ultrasonographic assessment also provides valuable information on the extent and localization of the thrombus, real time evaluation of blood flow compromise, the presence of collateral circulation, and allows for sequential monitoring for dissolution.

One can also perform a comparative doppler blood pressure reading in the affected versus non affected limb(s).

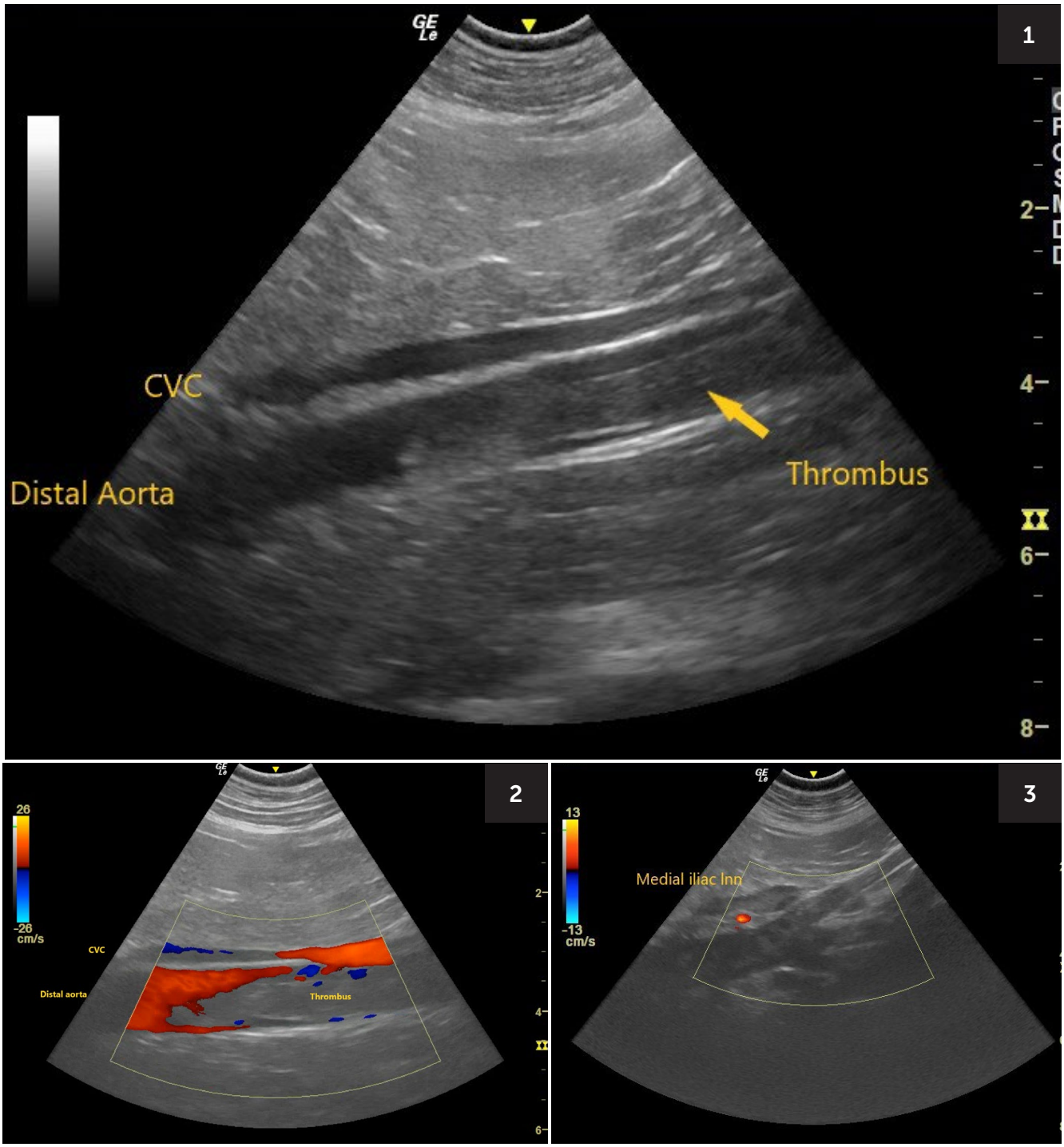

Figure 1. Aortic Thrombus in a canine patient. Sagittal plane B-mode image acquired from the right of the patient, showing the distal aorta and caudal vena cava (CVC). There is an isoechoic, well-demarcated soft tissue mass within the aortic lumen consistent with a thrombus (arrow).

*Figure 2. The same image as in Figure 1 now with the colour doppler function activated, showing the presence of flow cranial to the thrombus, with no flow present across or caudal to it. *Note: Colour doppler has not been fully optimized in this case.*

Figure 3. Image acquired caudal to those of Figures 1 and 2. No doppler flow was detected at the area of the aortic trifurcation, but the isoechoic thrombus was visible extending into the branches of the external and internal iliac arteries.

Finding Thrombi on Ultrasound

Visualisation of a thrombus within the aorta or its branches requires a probe capable of imaging at higher frequencies of 6 – 10 MHz. A micro-convex or linear probe can be used, and machine doppler capability is important.

The great vessels have a typical ultrasonographic appearance; tubular structures with an anechoic lumen and thin hyperechoic walls. When interrogated with doppler they demonstrate the presence of laminar flow.

Thrombi are visible as echogenic areas within the blood vessel lumen, that can be adherent to an area of the blood vessel wall. A wide variety of echogenicities have been described for thrombi, ranging from hyperechoic, isoechoic or hypoechoic to the adjacent muscle.[1] This has been potentially attributed to the age and composition of the thrombus.[6] More acutely, a thrombus may be less echogenic and more difficult to visualise than a chronic thrombus.[7] A thrombus can appear homogenous or heterogenous, the latter because of thrombolysis and recanalisation.[7] Lack of or decreased blood flow distal to the clot can be assessed with spectral, power or colour doppler.

Tips for Optimising Colour Doppler in the Great Vessels

- Colour Doppler (rather than power Doppler) should be used to assess large vessels.
- Ensure your colour box is as small as possible to increase doppler sensitivity and maintain as high a frame rate as possible.
- B-mode and colour Doppler frequency need to be optimised for the depth the vessel lies below the skin.
- Set appropriate color gain by holding your probe in the air and decreasing the colour gain until all speckling disappears.
- If your colour appears to 'bleed out' from the blood vessel walls, decrease the colour gain or increase your pulse repetition frequency (PRF) (also known as velocity scale or scale) or lastly, adjust the threshold.
- Similarly, if the colour does not appear sensitive enough, increase the colour gain or decrease your PRF.
- If aliasing is present, increase your PRF.
- Higher PRF's are used for higher flow vessels, and lower PRF's are used for lower flow vessels.
- A perpendicular angle between the ultrasound beam and the vessel is not optimal for Doppler detection, use the steer function or try and orientate to 45 – 90° if possible.

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Veterinary Dermatology Exam
Watch For These Red Flags

Jennie Tait, AHT, RVT, VTS
(Dermatology – Charter Member)

One the most challenging things about veterinary dermatology is that a lot of conditions look very similar. That's because there are only a limited number of reaction patterns in the skin. How are you supposed to tell what's what?

A dermatologic exam is one part of the puzzle and should include your entire patient from stem to stern:

Eyes
You're looking at the sclera, conjunctiva and periocular region. Many atopic animals will have conjunctivitis or episcleritis, which could end up causing self-induced trauma, blepharitis, erythema, hypotrichosis and excoriations. What else do you think of when I say "periocular"? Demodicosis should come to mind.

Ears
This includes palpating the ear canals and examining the concave and convex surfaces, as well as margins of the pinnae. If crusting is noted on the ear margins, *Sarcoptes scabiei* should be on your differentials list and a pinnal-pedal reflex should be checked. Ear margin dermatitis is also seen in vasculitis, drug reactions, solar dermatitis and immune-mediated diseases like lupus and pemphigus foliaceus.

Submandibular and prescapular lymph nodes
Generalised lymphadenopathy accompanies any type of mange but will be more localised when dealing with infections.

Appearance of nasal planum
Not many conditions will cross over from skin to the nasal planum, so if crusting is present, consider mucocutaneous pyoderma or an immune-mediated condition like lupus or pemphigus foliaceus. If there is a loss of pigment in the nasal planum, along with a smoothing out of the normal cobblestone appearance, you'll be leaning toward discoid lupus erythematosus.

Muzzle and lip margins
Hypotrichosis on the dorsal muzzle is a subtle hallmark of hypothyroidism and may or may not be accompanied by facial oedema, causing a tragic expression. If there is crusting of the lip margins, this could be mucocutaneous pyoderma or an immune-mediated disease. Some immune-mediated skin diseases also have oral lesions, so take a peek while you're there. Cats can also have oral lesions with eosinophilic granuloma complex and may have an accompanying indolent ulcer on the upper lip or a swollen ("pouty") lower lip.

Cranial locations
Examine under the collar, at the ventral neck fold and down both forelegs. Erythema and evidence of barbering are often seen on the cranial aspect of forelimbs at the flexure sites of elbows in patients with environmental allergies. Ask the owner if they've seen their furry friend do any "corn cobbing" or nibbling down the legs. Continue to palpate lymph nodes as your exam progresses caudally.

Other classics for environmental allergies include erythema of the interdigital spaces, caudal surfaces of the carpus, axillae, inguinal region and ears. When these areas are inflamed, they create the perfect conditions for secondary bacterial and fungal infections. Performing cytology is necessary for appropriate treatment. Even after 20 years of working in dermatology, I am still surprised by findings on cytology—you can never be sure what's going on until you examine your samples under the microscope!

Patients with atopy often have secondary infections on the ventrum, while patients with adverse cutaneous food reactions will often have secondary lesions over the dorsum.

Food-allergic patients also often have perianal erythema with or without adverse gastrointestinal signs that can include halitosis, burping, borborygmus, flatulence, scooting, anal gland issues, pica, vomiting and abnormal stools (very hard, soft, diarrhea, voluminous, multiple bowel movements per day). Unfortunately, many sensitive patients have a combination of food and environmental allergies.

Coat

A moth-eaten appearance can indicate folliculitis, which can be a result of a bacterial infection or even demodicosis. Take a good look at the hairs. If follicular casts are evident, your differentials could include vitamin-A–responsive dermatoses, primary seborrhoea, sebaceous adenitis, demodicosis or even dermatophytosis. Is the coat brittle or dry? Is it greasy? Does it epilate easily? Any conditions involving the hair follicle or sebaceous gland could be involved.

Are there areas of alopecia? Are there lesions? If these are symmetric, consider an internal disease process such as allergies or immune-mediated, metabolic or endocrine disorders. Asymmetric lesions move your differentials to infections, ectoparasites or neoplasia. Are there any comedones (blackheads)? Comedones are also seen with endocrine disorders as well as vitamin-A-responsive dermatoses, demodicosis and dermatophytosis. They can also be seen in animals that are genetically predisposed, like schnauzers or Mexican hairless.

Distribution patterns

Look for patterns—many conditions have hallmark patterns. Examples: Extremities are affected in vasculitis; face and feet in demodicosis; ear margins, hocks and elbows for sarcoptes; dorsum for cheyletiellosis; and head and paws for pemphigus.

Scaling and dander

Are flakes fine or large? Very large scaling is seen in ichthyosis, while smaller scaling can occur in seborrhea, follicular dysplasia or following chronic inflammation. Scaling with pruritus is seen with cheyletiellosis, *Demodex injai* or cutaneous T-cell lymphoma. Scaling without pruritus can be seen with dermatophytosis or keratinization disorders. Bear in mind that there will be increased scaling for the first couple of months in a patient just started on thyroid supplementation.

Appearance of skin

Thin or atrophied skin can be seen with Cushing's syndrome or in patients on long-term corticosteroids. Myxoedema can be seen with hypothyroidism. Thickened or lichenified skin is seen in patients with chronic inflammation. Hyperpigmentation is also seen as a footprint of inflammation. On the opposite end, hypopigmentation is seen with vitiligo and, more concerning, with lupus.

Are there papules, pustules or epidermal collarettes? All of these are seen with pyoderma but also in immune-mediated skin diseases. Pustules that are a result of a bacterial skin infection usually only span one hair follicle. Larger pustules spanning more than one follicle are seen with immune-mediated disease. Bacterial infections usually affect the trunk and may extend down legs, while immune-mediated diseases will affect the head and paws followed by the trunk. Always check mucocutaneous membranes. If affected, your differentials can include harsher forms of pemphigus with a poorer prognosis.

Paws

Don't forget to examine every paw, including claw beds, nails and the interdigital spaces. Crusting of foot pads can be seen with many things, including pemphigus, lupus, vasculitis, zinc-responsive dermatosis and hepatocutaneous syndrome (often with fissuring). Swollen footpads in cats are seen with plasma cell pododermatitis. If more than one claw is affected in a dog, consider symmetric lupoid onychodystrophy. Crusting of ungual folds is often seen in cats with pemphigus.

Hyperkeratosis of footpads and the nasal planum can be seen with distemper, zinc-responsive dermatosis, hepatocutaneous syndrome, pemphigus and lupus. This can also be seen with idiopathic nasodigital hyperkeratosis and hereditary nasal parakeratosis of Labrador retrievers. Breed predilections play a big role in narrowing in on a differential diagnosis as well as age and response to previous treatments.

It's a lot, but if you put all the pieces of the puzzle together (patient signalment, history, physical findings and diagnostics), you can't go wrong.

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Managing Recurrent UTIs in Pets

Kristi Reimer Fender, News Channel Director

Internist and Fetch dvm360 conference speaker Dr. Michael Wood offers practical insights into this frustrating veterinary condition.

When it comes to urinary tract infections (UTIs) in pets, the good news is that most dogs with simple uncomplicated UTIs will recover from the episode and go on to live a happy healthy life, says Michael Wood, DVM, PhD, DACVIM (SAIM), of the University of Wisconsin-Madison School of Veterinary Medicine. The not-so-good news? About 25% of pets that develop a UTI in their lifetime will experience recurrence.

Recurrent infections are a problem for a couple of major reasons,

How do you:

1. Manage your patient in a way that minimizes antibiotic resistance, both in the individual and more broadly in the population? T
2. Decide whether bacteria in the urine should be treated or not?

Your basic game plan

Step one when faced with a UTI, is to **know your local resistance patterns**. "Veterinarians have generally not done a very good job in this area," Dr. Wood says.

Yes, you can look up published data on antibiotic resistance in veterinary patients, but this data may or may not match what's happening in your immediate patient population. The best approach is to **keep track in your own hospital by recording culture and sensitivity results for every patient, building a database over time.** This will help you know which antibiotics are likely to be more effective and less effective in your own patients when an empiric choice is needed. Antibiotics with resistance rates greater than 20% should be used cautiously if at all empirically.

Step two is to understand a specific patient's individual risk factors for resistance. For cats, risk increases with the number of antibiotics used in the last three months and the number of days the cat has been hospitalised-more days means increased likelihood of resistance. In dogs, prior use of antibiotics is a risk factor, especially fluoroquinolones. Dogs are also at higher risk of developing resistance if they've been hospitalised for three days or more or if they consume a raw meat diet.

Step three is to perform a urine culture. The challenge, of course, is obtaining the urine sample. While cystocentesis will provide the most reliable results, free catch is also acceptable-you just have to interpret the results accordingly. With a free catch urine sample, you need to see more than 100,000 colony-forming units/ml to be confident you have a true infection rather than a contaminated sample.

While you're waiting for culture and sensitivity results, empiric therapy is justified if the patient has had limited previous antibiotic use, if you know the likely pathogen, if you know local susceptibility, and if the patient is showing clinical signs. According to the International Society for Companion Animal Infectious Diseases (ISCAID), appropriate first-tier empiric antibiotics are amoxicillin, amoxicillin-clavulanate, and trimethoprim sulfa. But this is where it's important to know your local resistance rates, Dr. Wood says. Your own first, second and third choices may differ accordingly.

Second-tier antibiotic choices, which should be prescribed based on culture and sensitivity results, are fluoroquinolones, third-generation cephalosporins and nitrofurantoin; the latter is best for maintaining urine sterility but does have side effects that should be discussed with the owner. An important exception outlined by ISCAID is pyelonephritis. Here a fluoroquinolone is an appropriate first-line treatment because of its ability to penetrate into kidney tissue where other water-soluble antibiotics are less effective.

What about NSAIDs?

ISCAID proposes that nonsteroidal anti-inflammatory drugs (NSAIDs) may be effective as an antibiotic alternative to treat UTI in veterinary patients based on human studies. The thinking is that they provide the patient relief from clinical signs while the body clears the infection on its own. However, Dr. Wood says, in the human studies, people who use NSAIDS in this manner are shown to experience clinical signs one day longer than those treated with antibiotics, and increased rates of pyelonephritis were detected. Given the challenges of diagnosing pyelonephritis in our veterinary patients, Dr. Wood doesn't use NSAIDs to treat UTI with one exception: If a patient with known recurrent UTIs presents showing clinical signs, NSAIDs can be used as an effective symptom reliever while you're waiting for culture and sensitivity testing results.

Three kinds of recurrent infections

Recurrent bacteriuria-defined as the detection of bacteria more than three times in a year or twice in six months-falls into one of four categories.

1. **Persistent UTI**: When a urine culture is positive seven to 10 days after the beginning of treatment, this means bacteria has become resistant, the patient's immune system is compromised, or the antibiotic cannot achieve a high enough concentration to wipe out the infection either for endogenous or exogenous reasons. All require further investigation to manage the infection appropriately.

2. **Relapse**: This means the urine is initially cleared of bacteria, but bacterial reservoirs remain, allowing recolonisation of the bladder. This condition can be seen in patients with urolithiasis, prostatitis and pyelonephritis and may call for a longer course of treatment or an antibiotic that better penetrates the area of the infection.

   Another example, though unproven in cats in dogs, is intracellular bacterial communities where bacteria remain quiescent within the urothelium only to proliferate again when the urothelium is turned over.

3. **Reinfection:** This occurs when the UTI is cleared by an antimicrobial, but abnormal host defenses (for example, low urine-specific gravity) prime the host for another infection. "You can treat each of these infection episodes as an uncomplicated infection with five to seven days of an antibiotic," Dr. Wood says.

Bacteria without clinical signs

Persistence, relapse and reinfection are true UTIs that require treatment if the patient is experiencing clinical signs. But what do you do with subclinical bacteriuria, where you have a positive urine culture but clinical signs are absent?

In people, asymptomatic bacteriuria is rarely treated. In fact, doctors restrict screening of diabetics, people with spinal cord injuries and immunocopromised patients for bacteriuria because they assume it will be present-and it may actually be protecting the patient from a more virulent organism.

ISCAID agrees with this approach, stating that if a veterinary patient is not showing clinical signs, it should not be treated. But Dr. Wood says this approach is not always practical in clinical practice.

"It depends on how much you trust the owner to recognise subtle clinical signs," he says. "Are they likely to notice slight changes in frequency, urgency or volume?" Dr. Wood says if he's confident an owner would pick up on these things in their pet, he doesn't treat subclinical bacteriuria.

However, he warns, "This is by no means a foolproof method and hence must only be considered with a strong understanding of the patient's overall health and after discussing the pros and cons of not treating the bacteriuria with owners."

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CBD: Forget the Legal Issues Does it Work in Veterinary Patients?

Carla Johnson, DVM

Veterinarians are being bombarded these days by questions, requests, lofty claims, unrealistic expectations and patchy scientific information about cannabidiol (CBD) products for pets. Public demand is outpacing the research as clients increasingly gain access to these products and, on the advice of a neighbour or internet article, begin using them on Max and Maizy. It's a bit of a free-for-all.

Scientific input from reliable sources is simultaneously vague and overwhelming, largely because this is uncharted territory. Researchers are still investigating the endocannabinoid and phytocannabinoid systems in people and animals, along with the phytopharmacology of the cannabinoids themselves. All of this needs to be unravelled before the veterinary profession has clarity, and while the unravelling is happening, demand is growing faster. Existing research gets a spotty summarisation in most veterinary lectures on CBD;for one, there's never enough time to explain it all, and two, the story is not complete.

Even so, research data is not what most veterinarians want to hear. They want to know about clinical usage. Unfortunately, the data is not yet translatable into reliable clinical information for all the claims made. While over 23,000 scientific papers on cannabinoids in humans, lab animals and companion animals in 24 different species have been published, the jury is still out on exactly what CBD products are effective for and what they are not.

Plus, despite a relatively good therapeutic safety index, they can cause harm, so caution is necessary. If they do cause problems, and veterinarians have been involved in recommending them, they can be held legally responsible for the outcome, and this includes both civil and criminal liability.

Legal and regulatory issues aside (because those change almost daily-and you should definitely stay in touch with veterinary legal experts in your state for specifics), what exactly do we know about the science and clinical value of CBD so far?

Several experts spoke at a recent 2019 Fetch dvm360 conference about their experience and knowledge of CBD, including some case-based information on its use in veterinary medicine. Robert Silver, DVM, MS, CVA, chief medical officer for Rx Vitamins and a practicing small animal veterinarian in Colorado, is seeing some trends and passed along what he recommends so far. Stephen Cital, RVT, RLAT, SRA, VCCS, CVPP, VTS-LAM, co-founder of the Veterinary Cannabis Academy and director of education and development for ElleVet Sciences, added his expertise as well.

Dosage

Prior to recent studies, the generally accepted therapeutic dose range for CBD in animals has been 0.1 to 0.5 mg/kg twice daily, but you can go as high as 5 mg/kg twice daily, according to Dr. Silver and Cital. A Cornell study shows that dosing as high as 8 mg/kg is safe,but this is not very cost-effective or practical, says Cital, who notes that dosing has been updated to 0.1 to 2 mg/kg twice daily based on now-available canine and feline data.[1]

Also, these experts say, not all patients will respond the same to standardised dosing. It is speculated that animals may need to have an endogenous "deficiency" in the endocannabinoid system to respond to these products.

As far as we know at this time, Dr. Silver says, cats and dogs should be treated the same way.

Route

The most commonly accepted route of administration at this time is oral, these experts say. Although there

are many forms of CBD and routes to choose from (concentrates, topicals, transdermals, vaporizers, nebulisers, suppositories, capsules or tablets, soft chews, powders, biscuits and so on), these are all still being studied in animals. Since bioavailability will be different for different routes, the recommendations for each will be different, and there are no guidelines yet.

Cital also warns that any transdermal or inhalation route will bypass first-pass metabolism through the liver. So "inhalers or vaporizers may be the new wave, but be very careful using this route," he says, noting the lack of evidence in companion animals as well.

Pain management

Lower doses of CBD are generally adequate for neuropathic pain, but higher dosages are often necessary for conditions causing chronic pain and inflammation such as osteoarthritis (OA), says Dr. Silver. Cital suggests starting with 1 to 2 mg/kg twice daily as support for either form of discomfort and titrating to effect. NSAIDs and cannabinoids can act synergistically, so dual use may lower the necessary dose of either.

Dr. Silver has been finding 0.5 mg/kg twice daily to be effective, and he reports that other veterinarians also find lower doses to work well for painful patients. "It's worthwhile to start at this lower dose, which may provide a successful outcome, in order to reduce the cost and amount of hemp extract to be administered," he says. The use of CBD together with opioids may allow a reduction in opioid doses, as CBD indirectly stimulates opioid receptors, producing an opioid-sparing effect, he says.

Cancer

Cannabinoids appear to be able to fight cancer, possibly through the induction of cancer cell death, anti-angiogenesis and some anti-metastatic properties, these experts say. Cital reports that an *in vitro* study looking at three different canine cancer cell lines was just completed by Joseph J. Wakshlag, DVM, PhD, at Cornell University, with results expected out by 2020. Dr. Wakshlag is also working on a quality of life *in vivo* study.

While Dr. Silver cautions that many claims surrounding CBD and cancer are, so far, not based in evidence, he also says researchers have discovered the presence of receptors on tumour cells for cannabinoids. He knows of a veterinary oncologist who was treating a lingual mass using non- tetrahydrocannabinol (THC) CBD as a sole treatment (0.5 mg/kg twice daily). Six weeks later the mass was reduced to nearly nothing. Another anecdote involved a dog named Olive with an appendicular fibrosarcoma whose owners did not want to amputate. Non-CBD-containing nutraceuticals were used first, with success, for one year, and then the tumour started to grow again. The patient was started on a 1:1 CBD:THC product, and within 90 days the tumour had shrunk dramatically. In another account, a dog with an undifferentiated nasal carcinoma with bone lysis was treated with a non-THC CBD product (0.4 mg/kg twice daily), and the tumour shrunk significantly over six weeks of treatment. The dog was still in remission 14 months later.

Tolerance

All humans and animals have been shown to develop tolerance to certain cannabinoids, such as THC, with chronic use, these experts say. This means if you're using them successfully for cancer treatment and then stop, the cancer may recur. If it does, as is the case with some other chemotherapeutic drugs, it has usually developed tolerance to CBD products, according to pet owners and oncologists who've observed this phenomenon anecdotally. It's important to warn clients about this. "If you get a tumour response and stop, it will come back, and it will come back resistant," Dr. Silver warns.

It's also speculated that secondhand smoke may create THC tolerance in animals, Dr. Silver said, but further studies are needed.

Seizures

THC itself is psychotropic, so it is not considered an anti-seizure drug-it has been reported to actually cause seizures. Therefore, any THC in a CBD product could theoretically make a seizure patient worse, but studies are incomplete. Cital notes that the manufacturer of the FDA-approved CBD medication Epidiolex conducted a 56-week-long study of rats and dogs at high doses of both THC and CBD (roughly 25 mg/kg twice daily) and were unable to induce any seizures in dogs.

However, Dr. Silver warns, "we are in the infancy of use [of CBD products] adjunctively with epileptic medications." He describes a Colorado State University study of CBD for refractory epilepsy (2.5 mg/kg twice daily) in which some dogs experienced a 40% reduction in seizures, not a very impressive result, Dr. Silver says.[2] A new study at a higher dose (4.5 mg/kg twice daily) is taking place now, but it's too early to draw valuable conclusions. Still, he says, for uncomplicated seizures that aren't frequent or are well-controlled with anti-convulsant drugs, veterinarians and pet owners are finding that 0.5 to 1.0 mg/kg of CBD twice daily can control seizures and, in some cases, allow for reduced doses of anti-epileptic drugs.

Michelle Carnes, MS, DVM, DACVIM, a veterinary neurologist who gave an update on seizure management at Fetch dvm360 in 2019, does not recommend using CBD products in seizure patients at

this time. She says the oral bioavailability of many CBD products is too poor, and CBD is a potent inhibitor of cytochrome P450 with a long half-life in dogs, which could potentially interfere with other medications.

"There are still no extensive studies in dogs," she says, describing the same CSU study that found little improvement in canine epilepsy when CBD was used as an adjunctive drug.[2] She reports that popular thinking surrounding use of CBD in animals with epilepsy originates from the treatment of seizures with Epidiolex in children with severe, difficult-to-treat forms of epilepsy.

"This drug is extremely expensive and has now been reclassified as a schedule 5 drug," Dr. Carnes says. It would not be affordable, or easily available, for veterinary patients at this time, even if it was found to be effective. "That aside, if we take the average human CBD product, using the current recommended dose in humans (5 to 10 mg/kg), and extrapolate this to dogs, the cost would be $444 per month for a 20-kg dog."

Dr. Carnes is not opposed to CBD, but at this time her conclusion is that it is "expensive, unregulated, and the purity is not [adequate]" for use in dogs with epilepsy, adding that it may be a long time before it's found useful.

Metabolism

CBD is metabolised by the cytochrome P450 system in the liver, which means drug competition problems are possible. According to Dr. Silver, this process has not been studied well, but rising alkaline phosphatase (ALP) levels with CBD use have been consistently noted in studies.[1,2] He says the CSU study found that concurrent use of CBD appeared not to change phenobarbital or potassium bromide blood levels, but this was only a pilot study.[2] Larger studies are taking place now.

Dr. Carnes delivered an anecdotal "metabolic" warning in her lecture, describing a case study, her own case, in which (unbeknownst to her) the owner gave CBD to his difficult-to-control epileptic dog for eight months. The dog was already on a moderate dose of phenobarbital. He had no seizures for those eight months, but his phenobarbital levels went from 30 to 50 µg/ml in that time. The dog became ataxic and weak, his liver values went "through the roof," and the dog died of liver failure shortly thereafter. Dr. Carnes warns veterinarians to "remind clients that CBD is still a drug!"

Cital reports that, despite these concerns, veterinary pharmacologist Dawn Boothe, DVM, MS, PhD, DACVIM, DACVCP, has personally reported no drug interactions or adverse effects associated with CBD and seizure medication combination therapies in her work at Auburn University. He also notes that "not all CBD products are alike." They may contain varying levels of cannabinoids, terpenes and contaminants that may either help or endanger a patient. When clients are using CBD products on pets without guidance, anything can happen. It's truly "buyer beware," Cital says.

Adverse effects and toxicity

Diarrhea seems to be a common adverse effect for CBD, but so far studies don't seem to be showing any long-term adverse effects associated with bloodwork or urine testing.[3] However, these experts say, the study lengths might be too short and the parameters too narrow at this time, with researchers needing more time for analysis. More recently a 12-week-long study in dogs and cats was published noting no statistical changes or concerns on physical examination, complete blood count or serum chemistry profiles.[4] THC toxicoses, on the other hand, are a major problem, and veterinarians need to at least advise clients to avoid harming their pets. "Too often in Colorado people are getting adult medical marijuana and giving it to pets," says Dr. Silver. "Human dosages of these drugs will send pets to the ER. Hemp products do contain THC and can cause typical signs of THC toxicity, but they are milder effects."

Here's one important message that's consistent from all specialists: No human edibles! "There is a huge risk of xylitol toxicity," Dr. Silver says. Cital reports that products may also include grape and raisin extracts without careful labeling. Cital also notes again that toxic contaminants in poorly produced products could also be dangerous to pets.

Future hopefuls

There's lots of enthusiasm for the therapeutic use of hemp products, even those with THC, for medicinal human and animal use. Cital notes a long list of potential future usages, including eye drops for glaucoma, tumour injections, stimulation of bone growth, safe sedation for puppies and young animals, anaesthesia induction, inhalers for lung cancer (CBD may have pulmonary cytoprotective properties), pain relievers from certain parts of the plant, antifungal and antimicrobial bedding for animals, treatment for chronic cystitis in cats, use for chronic dermatitis in dogs and cats, and treatment of inflammatory bowel disease, among others. Another use for hemp products is biowaste cleanup, they're able to absorb pollutants from the environment and can be used to reduce the effects of greenhouse gases.

How to choose a CBD product

In the absence of clear guidelines, what should veterinarians look for in a CBD product? A National

Animal Supplement Counsel (NASC) Seal of Quality Assurance is a good start. The NASC is a trade group for nutraceuticals that is taking the lead on trying to regulate the safety of commercial cannabis and hemp products, Dr. Silver says. Both Dr. Silver and Cital recommend that a certificate of analysis indicating potency, per-dosing unit, all ingredients, and the presence of mycotoxins, metals or pesticides can help you determine if a product is reasonably safe. Sadly, many manufacturers do not provide this information or offer only a limited version. Hopefully this will improve over time, these experts say.

Science

Why does this herb have such biomedical value? At least part of it, Dr. Silver reports, is that the body makes its own (endo)cannabinoids as part of the nervous system, or at least as a partner to it. There are cannabinoid receptors in the brain, heart, lungs, liver, spleen, intestinal tract, muscles, bone, reproductive system and circulatory system, among others. There is some evidence to suggest that the endocannabinoid system is responsible for the "runner's high" in people. "It's really the largest system in the body, but we didn't even know about it until recently," Dr. Silver says.

Conclusion

Many veterinarians have adopted a "Well, it can't hurt" attitude when it comes to CBD. Some of us have been reluctant to discuss these products at all. Neither approach appears to be the correct one. We as veterinarians should be careful recommending these products and urge our clients to be cautious, taking a position of "harm reduction."

"If we cannot answer client questions at this time, we can at least become more knowledgeable until we can," Dr. Silver says. Remember, anything that sounds too good to be true usually is, but CBD products may just turn out to have some astonishing medicinal values. We just can't tell you what they are yet.

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5. Dr. Carla Johnson practices emergency medicine at Berkeley Dog and Cat Hospital in Berkeley, California, and general practice at Cameron Veterinary Hospital in Sunnyvale, California. Her non-veterinary loves are writing, dressage with her Iberian warmblood mare, watercolor painting, yoga and running with her dog Tyson. Try as she might, her curly-coated Scottish fold Hootie refuses to go jogging with her.

Key Points for Managing Cutaneous Mast Cell Tumours in Dogs

Michael O. Childress, DVM, MS, DACVIM (Oncology) VETERINARY MEDICINE

Do canine mast cell tumours drive you crazy? If so, I suspect that you probably aren't alone. The clinical behavior of these tumours runs the gamut from relatively benign to highly malignant, making it challenging to decide on the best treatment approach for an individual tumour. Surgical removal is the mainstay of treatment for most mast cell tumours, while chemotherapy and radiation therapy are valuable adjunctive treatments (or alternatives) to surgery in some cases. When deciding on which course of therapy is best for dogs with cutaneous mast cell tumours, here are five key points to keep in mind:

This article is the first in a planned series by veterinary oncologist Dr. Michael Childress in which he will discuss five important facets of common types of tumours that general practice veterinarians should know.

1. The most important step in managing dogs with cutaneous mast cell tumour is determining whether the tumour is surgically curable.

This seems inherently logical, but rational thinking can be subverted by unreasonable expectations of surgical benefit harboured by pet owners and veterinarians alike. Most veterinarians are aware that histopathologic grade is the factor most associated with survival after surgical excision of a mast cell tumour. Unfortunately, this factor cannot be assessed until after the tumour is removed, so veterinarians must rely on other clinical features to decide whether to attempt surgery.

Small, solitary, slow-growing tumours on areas of the body amenable to wide surgical excision (such as the trunk) are usually excellent candidates for surgical removal. On the other hand, tumours that are large, rapidly growing or located at sites where wide excision is challenging may not be. These latter tumours are often high-grade cancers that cannot be cured with surgery.

Other clinical features that are more common in high-grade mast cell tumours include bleeding or ulceration of the tumour, regional lymph node enlargement and the presence of systemic signs of mast cell degranulation, such as vomiting, melena, hematemesis, coagulopathies, generalised pruritus, hypotension and anaphylaxis. Avoidance of surgery in these cases may be prudent because ...

2. Surgery does more harm than good for some dogs with cutaneous mast cell tumours.

Approximately 40% of surgically resected high-grade mast cell tumours will recur locally even if "clean" margins are identified on the histopathology report. Managing locally recurrent mast cell tumours is extremely challenging, as these tumours often grow more rapidly and extensively than the original tumour. Heparin and tissue proteases released by neoplastic

Figure 1: Recurrent high-grade mast cell tumour in a dog. This tumour arose following incomplete excision of a high-grade tumour via midfemoral amputation. The amputation stump is largely replaced by tumour. Significant tumour-associated subcutaneous hemorrhage is readily apparent. Images courtesy of Dr. Michael Childress.

mast cells at the surgical site can cause haemorrhage, surgical wound dehiscence or both (see Figure 1).

It's not clear whether dogs that undergo surgical removal of high-grade mast cell tumours experience a survival advantage compared with dogs treated palliatively with chemotherapy or radiation therapy. Therefore, the choice to pursue surgery in dogs with clinically aggressive mast cell tumours should be made carefully.

An incisional biopsy to confirm the tumour grade may be useful to plan surgery in these cases, as not all mast cell tumours with clinically aggressive features will be high-grade. Dogs with large but low-grade tumours may still benefit from carefully planned aggressive surgery.

3. The contemporary two-tiered histopathologic grading system for cutaneous mast cell tumours is probably more clinically useful than the previous three-tiered system.

The histopathologic grading system proposed by Patnaik in 1984 divided mast cell tumours into low (grade 1), intermediate (grade 2) and high (grade 3) grades, with increasing biological aggressiveness associated with increasing grade number.[1] This system predicted the clinical behavior of grade 1 and grade 3 tumours very well, but it fared poorly with grade 2 tumours. The more recent two-tiered system proposed by Kiupel in 2011 circumvents this problem by classifying mast cell tumours as either low-grade (grade 1) or high-grade (grade 2), eliminating the intermediate-grade designation altogether.[2] In the report initially describing this system, dogs with low-grade mast cell tumours survived more than two years after surgical tumour removal, while dogs with high-grade mast cell tumours survived less than four months.

Subsequent reports have confirmed the prognostic relevance of the two-tiered system, although it does fail to predict outcome for about 5% to 10% of dogs with low-grade mast cell tumours that ultimately succumb to tumour-related death.[2,3] Importantly, the two-tiered system is applied consistently by pathologists, who agree on tumour grade in more than 95% of cases when using it.[2] This compares with rates of agreement of approximately 60% to 70% when using the three-tiered system.[2,4]

4. Dogs with multiple cutaneous mast cell tumours usually have a good prognosis.

One of the most frustrating syndromes in dogs with mast cell tumours is that of multiple cutaneous tumours. This syndrome vexes pet owners and veterinarians alike, who may worry that the presence of multiple mast cell tumours implies a systemic, life-threatening disease. However, many—if not most—of these dogs appear to have a favorable prognosis. Veterinarians do still need to be mindful of the clinical features associated with aggressive tumours (see point 1) when assessing dogs with multiple cutaneous mast cell tumours, as dogs with multiple large, rapidly growing, ulcerated tumours probably do have a life-threatening disease (see Figure 2).

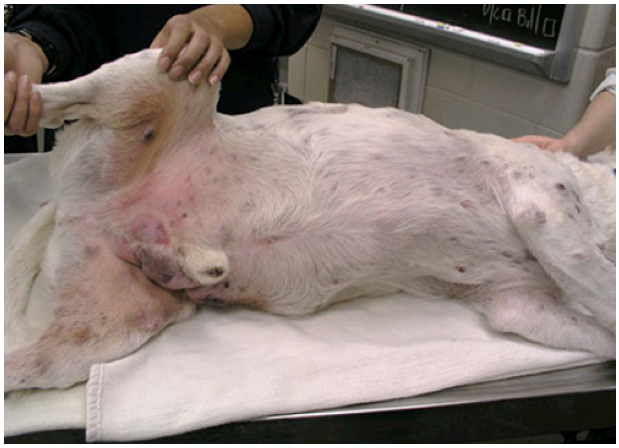

Fig 2. Dog with aggressive multiple mast cell tumours. This dog has numerous rapidly growing, ulcerated or discoloured cutaneous tumours. Note also the preputial swelling, which was associated with metastatic mast cell tumour in the superficial inguinal lymph nodes.

Most dogs with multiple cutaneous mast cell tumours have an indolent disease characterised by the sporadic appearance of small, slow-growing dermal tumours over many months to years. This condition is a cosmetic nuisance but essentially harmless. However, some dogs with multiple cutaneous tumours, have a life-threatening disease.

Most dogs with multiple mast cell tumours tend to present with small, slow-growing tumours. In such cases, surgical removal of one or two representative tumours is recommended to establish their histopathologic grade (they are almost always low-grade). If a low histopathologic grade is confirmed, watchful waiting is prudent for these cases,

accompanied by batched surgical removal of several tumours at a time if new tumours continue to form (and they often do). There is no evidence that any medical therapy prevents new tumours from forming in these cases, which can be upsetting to pet owners. However, reassuring these owners that the disease is not life-threatening (think of it as "mast cell acne") can help to ease their concerns.

5. The most useful chemotherapy drugs for cutaneous mast cell tumours are toceranib (Palladia—Zoetis), vinblastine and prednisone.

Chemotherapy is indicated for dogs with mast cell tumours that cannot be removed surgically or dogs that have undergone successful surgical removal of high-grade tumours, which carry a significant risk for metastasis.

- In the first of these settings, chemotherapy is given with palliative intent, with the goal of temporarily shrinking the tumour and improving the dog's quality of life.
- In the second, chemotherapy is given to prevent or delay metastasis, thereby extending survival. Toceranib, vinblastine and prednisone are the drugs that most reliably achieve either one of these outcomes.

Because its spectrum of side effects does not overlap with that of toceranib or vinblastine, prednisone can be combined with either drug, which may result in additive or synergistic anti-tumour activity. Combinations of toceranib and vinblastine, however, have proven too toxic to recommend their use at this time. Other drugs whose use for treating mast cell tumours is reported include lomustine (CCNU), cyclophosphamide and chlorambucil. However, these drugs appear to be less effective than the aforementioned three, and they probably should not be used in the first-line treatment setting.

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5. Dr. Michael O. Childress is associate professor of comparative oncology at Purdue College of Veterinary Medicine

The ABCs of Veterinary Dentistry

'U' is for Ulcers—What a Pain!

Treating these painful lesions in dogs and cats requires figuring out what's causing them.

Jan Bellows, DVM, DAVDC, DABVP, FAVD

An ulcer is a tissue defect that has penetrated the border between epithelium and connective tissue. Its base is located deep in the submucousa or even within muscle or bone. An oral ulcer is a break in the mucous membrane with loss of surface tissue and necrosis of epithelial tissue. It is a deeper breach of epithelium than an erosion or excoriation, involving damage to both epithelium and lamina propria (Figure 1). Oral ulcers in dogs and cats are painful to the patient and often challenging to the veterinarian who has to figure out what to do about them to eliminate suffering.

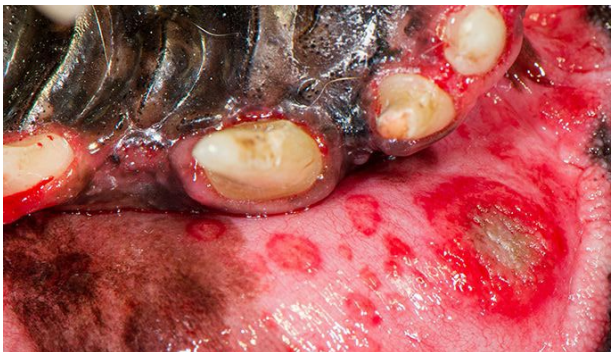


Figure 1. Superficial and deep ulceration in a dog's mouth. All images courtesy of Dr. Jan Bellows.

First things first: Identification

Clients rarely notice their dog's or cat's oral ulcerations. If the lesions were on the tip of their pet's nostrils, they'd be on your office doorstep in a minute. More often it's the veterinarian or technician who finds ulcers either during an exam room check or when the patient is under anaesthesia.

The most commonly affected oral tissues include the oral mucousa, the palatal mucousa, the lip margins and the vestibules (areas between the teeth, lips and cheeks) (Figures 2A-2C).

Some dogs and cats with oral ulcerations show excessive drooling (Figure 2D), halitosis and a history of pain when eating. Unfortunately, most do not show any clinical signs, suffering silently.

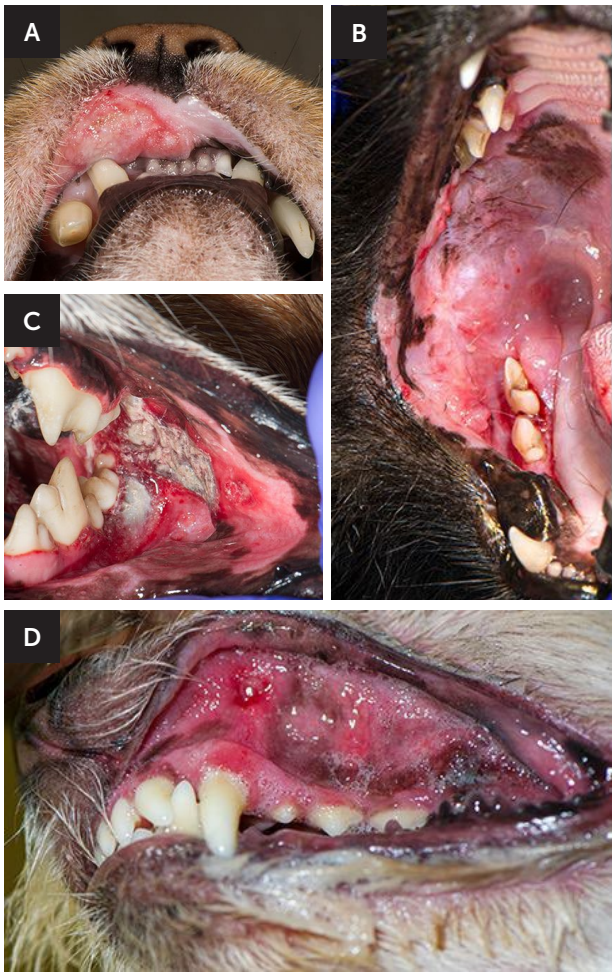


Figure 2 (A) Rodent ulcer affecting a cat's lip. (B) Diffuse ulcerations involving a cat's soft palate and right vestibule. (C) Marked oral ulceration involving a dog's left vestibule (D) Ptyalism secondary to oral ulcers.

Oral ulcers arise from either inside or outside causes.

Inside (organic internal medicine) causes. Internal causes of oral ulcers include viruses, bullous mucocutaneous diseases, azotemia and neoplasia (Figure 3).

Outside (other than organic) causes. Contact ulcers occur secondary to direct mucousal interaction with an irritant, allergen or antigen (Figure 4). Contact mucositis with ulceration is most commonly observed where the labial, buccal or lingual mucousa touches a prominent tooth surface in susceptible dogs or, more rarely, in cats. They have also been referred to as kissing ulcers, kissing lesions and chronic ulcerative paradental stomatitis (CUPS) lesions.

Ulcers are wounds. Their persistence depends on their etiology and the animal's ability to self- repair. Treatment of oral ulcers involves eliminating the cause, allowing re-epithelisation to occur. Topical medicaments with zinc ascorbate and zinc gluconate

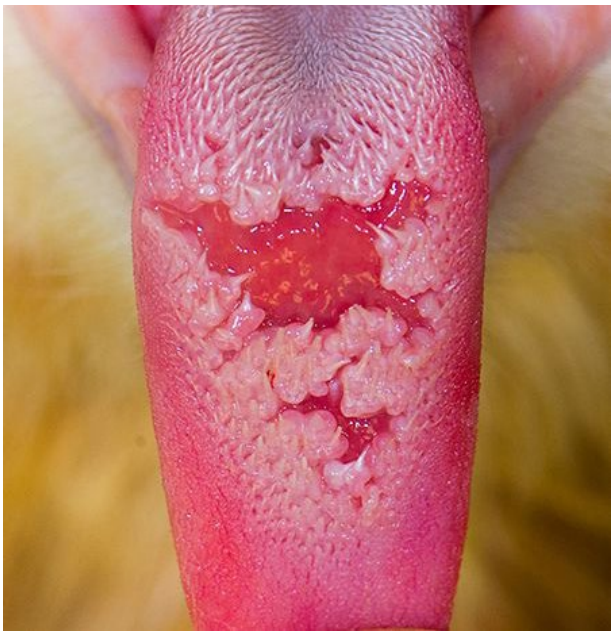


Figure 3A. Marked ulceration on a cat's tongue secondary to calicivirus and feline leukemia virus.

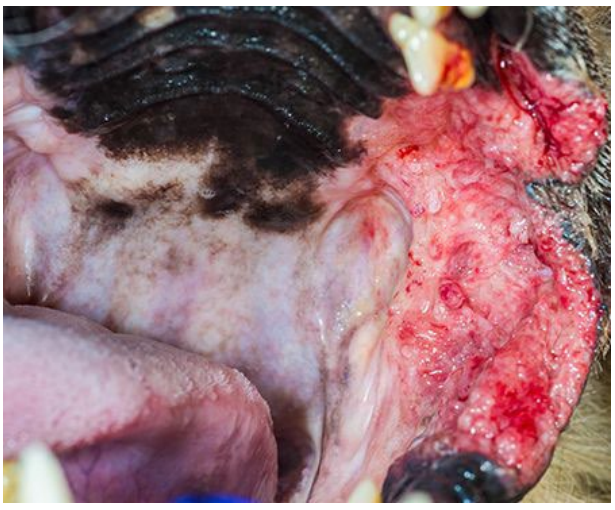


Figure 3B. Vestibular ulceration secondary to squamous cell carcinoma

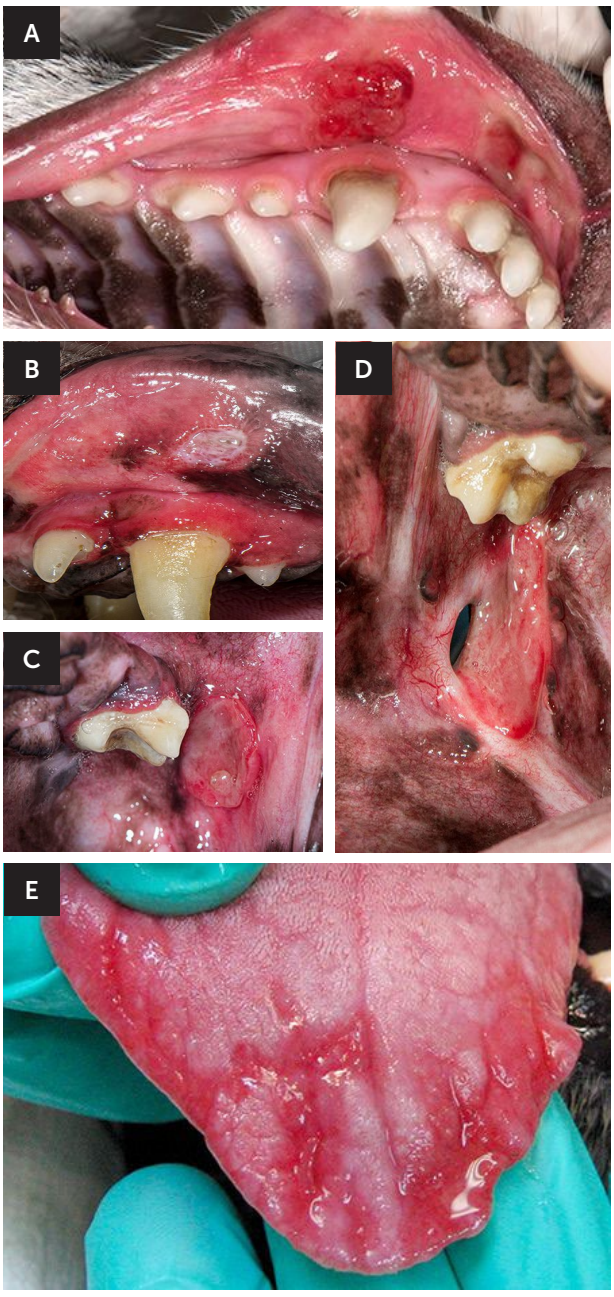


Figure 4(A) Contact mucousitis with ulceration affecting a dog's maxillary canines and incisors (B) Pseudomembrane formation over ulcer secondary to a hypersensitive reaction to plaque. (C) Contact ulceration caused by a dachshund's left maxillary first molar. (D) Contralateral side in the same dog as in Figure 4C where ulceration has eroded through all layers of the mucosa, submucosa and epidermis. (E) Ulcers on a dog's tongue after contact with bleach.Treatment

(such as Maxi/Guard Oral Cleansing Gel-Addison Biological Laboratory) help stimulate collagen production, which is part of the healing process. The antimicrobial properties of zinc ascorbate help control infection. Other therapies include tooth extraction to eliminate mechanical irritation, short-term use of systemic anti-inflammatories and antimicrobials, and CO_2 laser.

In cases of ulcers caused by plaque-laden tooth contact with the alveolar mucousa (Figure 5A), initial treatment involves dental scaling and polishing followed by scrupulous home care administered twice daily. Daily application of OraVet Plaque Prevention Gel (Boehringer Ingelheim) is advised to create a barrier between the tooth and mucousa. Unfortunately, this does not usually eliminate the ulcers, necessitating extraction of teeth (Figures 5B, 5C).

Figure 5A. Marked ulceration secondary to plaque hypersensitivity in a Maltese dog.

Figure 5B. Full-mouth extractions to remove the plaque-laden teeth from contacting sensitive mucous membranes.

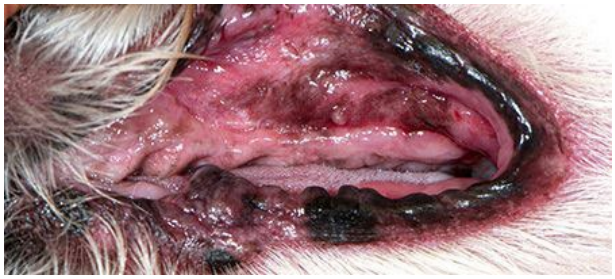


Figure 5C. Resolution of ulceration after full-mouth extraction

The use of a CO_2 laser to photovaporise oral ulcers has been met with favorable results. The process decreases pain and the bacterial load, leaving a "Band-Aid" char covering exposed tissue. The laser is set between 3 and 6 watts in continuous mode, and the ulcer is slowly circumscribed by gradually focusing on the lesion until the entire ulcer is "painted" with light energy (Figure 6).

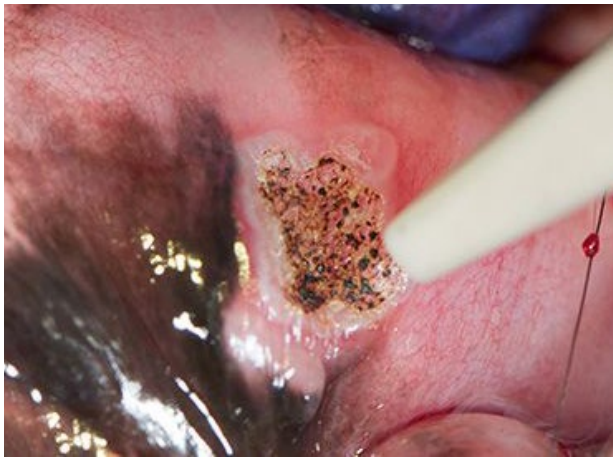


Figure 6A. CO_2 laser treatment of a localized ulcer.

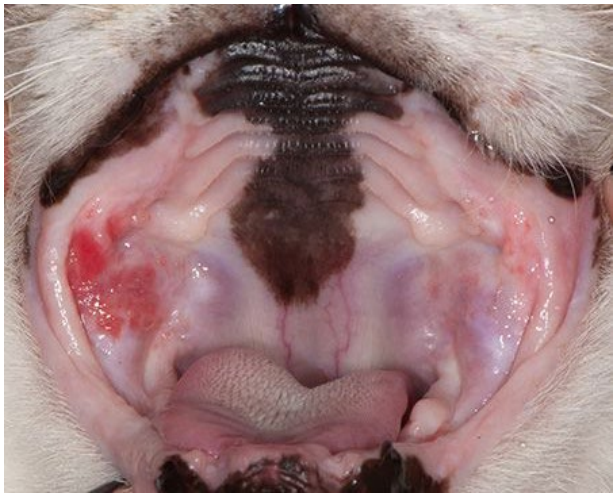


Figure 6B. Refractory caudal ulceration in a cat after full-mouth extraction.

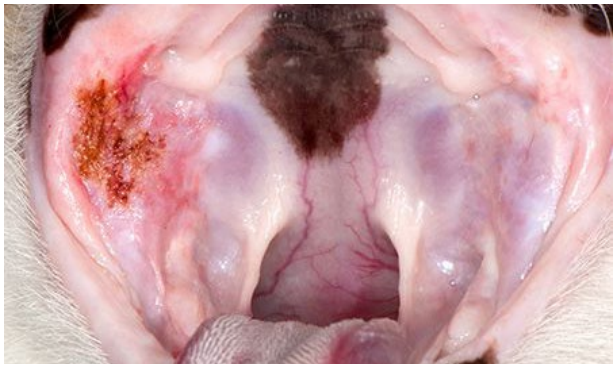
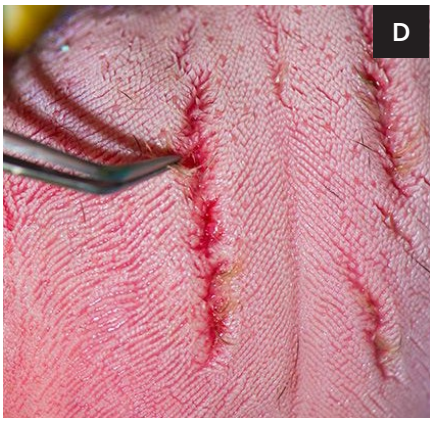
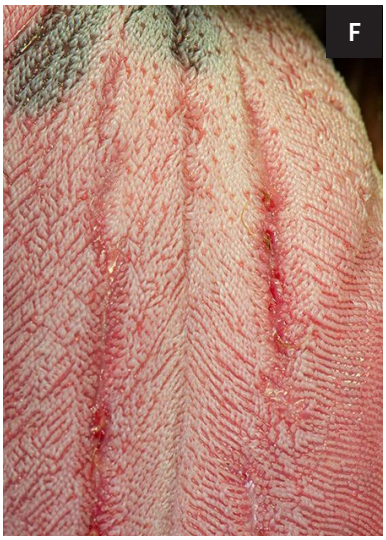
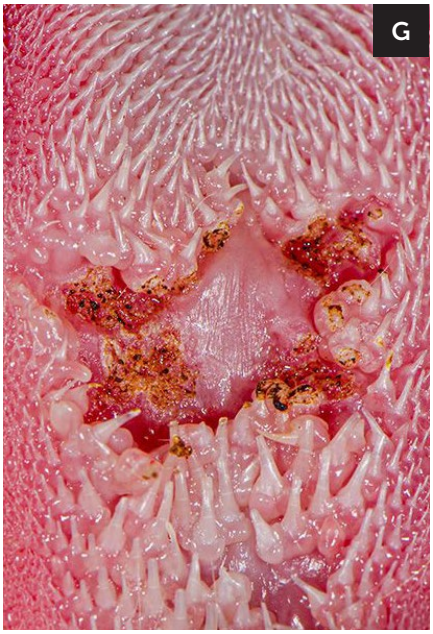


Figure 6C. Laser tissue ablation on the affected side in the same patient as in Figure 6B







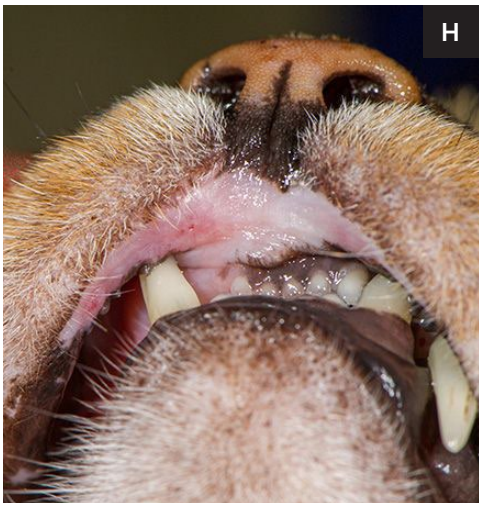


Figure 6D. Tongue ulceration secondary to hair foreign bodies.

Figure 6E. The CO2 laser used to help treat the ulcers in the same patient as in Figure 6D after hair foreign body removal.

Figure 6F. Appearance of patient shown in Figure 6D one month later at a follow-up laser treatment.

Figure 6G. Laser treatment for resolving ulceration of a cat's tongue (same patient as in Figure 3A).

Figure 6H. Resolution of the rodent ulcer in the patient shown in Figure 2A after systemic steroid treatment and local CO2 laser tissue ablation.

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