Continuing Professional Development | Business Success | Clinical Skills

vet 360

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CPD Article

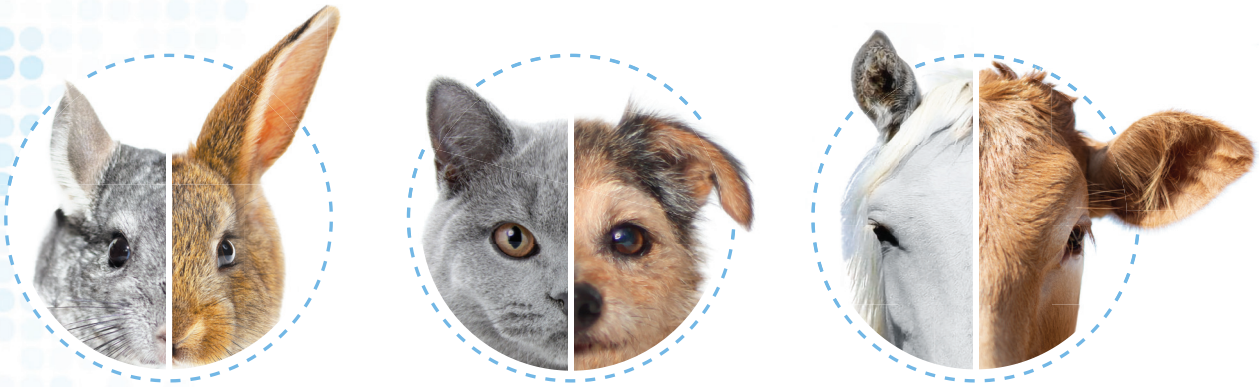
Managing Immune-mediated Haemolytic Anaemia

Anaesthesia

Anaesthesia & Management of Peri-Anaesthetic Hypotension for Critical Patients

HYPOCHLOROUS ACID

ADVANCED INFECTION MANAGEMENT



- Non-cytotoxic – shown to have beneficial effects on fibroblast and keratinocyte migration at low concentrations;[1,2]
- Kills bacteria, fungi, viruses, spores and amoeba – killed 99.99% of test pathogens within 2 minutes of exposure;[4]
- And improves wound healing, reduces pain, exudate, odour and scar formation.[3,5]

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Editor's Note

I can't believe it's already nearly the end of the year. I'm loving the green buds on the trees and the spring flowers. Being outside is really a joy. This edition continues with the anaesthetic management with a proceedings article on anaesthesia and hypotension. We also have the second half of the IMHA article and I did some research and summarised two recent articles on blood groups and also the ACVIM consensus statement on treatment of IMHA. This collection is our CPD "article". I would urge vets to utilise the free access journals. Much more information is freely available than before. The Journal of Veterinary Internal Medicine is free access and often has helpful articles for the GP - whereas some are definitely high on the specialist range.

I would be interested to hear what the vets in practice (you) are wanting to see published in the Vet360. I often publish according to what I have recently seen or new info I have recently come across which I think could be helpful.

I would also like to thank Dr Paolo Pazzi, senior lecturer at the Department Of Companion Animal Clinical studies and MMedVet and ECVIM Small Animal Specialist, for agreeing to help as a subeditor - assisting with content evaluation from September onwards.

I hope you enjoy the edition

Regards,

Liesel

Index:

PREVIOUS EDITION: July 2020

- Common Dilemmas in Epilepsy
- Peri-Operative Hypothermia: Foe More Than Friend
- Understanding the Use of Antibody Titres in Veterinary Practice

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Anaesthesia and Management of Peri-Anaesthetic Hypotension for Critical Patients

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Fetch DVM360 Conference Proceedings 2020

General anaesthesia involves the careful and judicious use of compounds that induce sensory deprivation to noxious stimuli, muscle relaxation, and in most cases, unconsciousness. In critically ill patients, there often is a delicate balance between loss of consciousness and cardiovascular and respiratory compromise, requiring careful monitoring techniques to ensure patient safety. Fortunately, many animals that present to you in an emergency setting will be young, healthy animals that may require anaesthesia to repair minor trauma. Other cases, however, will present to you with potentially life-threatening critical illnesses, making anaesthesia more challenging and somewhat risky. Many anaesthetic agents induce some degree of cardiovascular and respiratory depression. The goal of this presentation is to describe anaesthetic protocols for both healthy and unhealthy animals

The Physical Examination

The physical examination is one of the most important aspects of preparation prior to inducing anaesthesia. In critically ill patients, a careful physical examination should be performed just prior to inducing anaesthesia, as clinical status may have changed dramatically since initial presentation, often changing your choice of anaesthetic protocols.

Questions to ask yourself include: Is the patient's airway patent? Does the animal require mechanical or assisted ventilation? Is the animal morbidly obese or have an intraabdominal

mass that will change efficacy of ventilation once the patient is placed in dorsal recumbency? Does the animal have a sucking chest wound, rib fractures, pleural effusion, or potential for pneumothorax? Are there pulmonary contusions that may be affected by large volumes of intravenous fluids? Are the animal's respirations effective or ineffective? Is the animal in a state of circulatory shock? Is there a normal circulating blood volume? Is the heart beating efficiently, or is there a cardiac murmur or dysrhythmia? What is the clinical status of the animal? Is there an adequate blood pressure? Is there any sign of ongoing haemorrhage or severe electrolyte loss?

In most cases, the answers to these questions can be obtained from a thorough and systematic physical examination, starting with the basic ABC's

of emergency medicine. The animal must have a patent **Airway**, to deliver oxygen into the lungs while **Breathing**, and the oxygen can be delivered to tissues during the process of normal **Cardiac function** and **Circulation**. Once the ABC's have been evaluated and stabilized, other diagnostics can then be performed.

Bloodwork and Electrolytes Abnormalities

Following physical examination, bloodwork should be performed to evaluate the patient's oxygen carrying capacity, renal and hepatic function, and coagulation status. Every effort should be made to normalise values prior to inducing anaesthesia. However, in many cases, until the underlying problem is definitively addressed, patient clinical status may not improve despite very aggressive efforts. These types of cases present the most challenge for the veterinary practitioner in deciding how to induce general anaesthesia without potentially doing more harm.

Patients with traumatic uroabdomen should be stabilised prior to inducing general anaesthesia. Every effort should be made to decrease serum potassium to less than 7 mmol/L before any anaesthesia is induced. Treatment protocols include administering regular insulin (0.25 units/kg IV) and dextrose (2 gm dextrose IV per unit of insulin, followed by 2.5 – 5% dextrose CRI to prevent hypoglycaemia), intravenous sodium bicarbonate (0.25 – 1.0 mEq/kg), or calcium gluconate (0.5 – 1.0 ml/kg 10% solution IV). The electrocardiogram should be monitored closely for the appearance of atrial standstill and inappropriate bradycardia.

Secondly, drainage of intraabdominal fluid can be accomplished by placement of an intraabdominal catheter attached to a closed collection system. A red rubber tube or an Argyle chest tube can be placed using a local anaesthetic such as lidocaine (0.5 – 1.0 mg/kg). Once secured, the drain can be left in place until definitive repair of the urinary tract can be performed at the time of surgery.

Anaemic or haemorrhaging patients should have a combination of crystalloid and colloidal support. In cases of haemoabdomen and gastric dilation-volvulus, synthetic haemoglobin can be administered as a bolus or as a slow trickle (1 – 2 ml/kg/hour) to provide both colloidal support and improve oxygen carrying capacity.

Preanaesthetic Agents

There are several rationales for using pre-anaesthetic medications. One of the most important reasons for using premedications is to decrease the total amount of anaesthesia required to induce and maintain general anaesthesia. The use of a balanced anaesthetic approach provides many benefits for the patient, particularly those that are critically ill. Anticholinergic drugs such as atropine and glycopyrrolate increase heart rate by inhibiting vagal stimulation. Glycopyrrolate though, has less of a chance of inducing tachyarrhythmias. Atropine reduces respiratory secretions and can cause second-degree heart block. Atropine crosses the blood-brain and placental barriers, while glycopyrrolate does not. This has important implications when providing anaesthesia for the periparturient dam in need of a C-section, anaesthesia that can potentially affect the outcome of the neonates.

Opioids, used in combination with a phenothiazine tranquilizer such as acepromazine, provide neuroleptanalgesia. Morphine provides excellent analgesia without inducing severe cardiovascular compromise. Potential complications of morphine and other opiate drugs include bradycardia (which can be reversed or prevented by using an anticholinergic agent), and hypoventilation. Morphine administration can also induce vomiting and ileus in ambulatory patients. In recumbent patients, though, the use of morphine is justified by decreasing doses of induction agents and inhalant drugs required to maintain general anaesthesia.

Butorphanol, a mu antagonist, kappa agonist, also can be used as a premedication when used in combination with a phenothiazine tranquilizer such as acepromazine. Used alone, however, butorphanol's sedative effects are fairly unreliable and short-lived. Additionally, due to its receptor affinity, using butorphanol early in the course of anaesthesia may prevent more potent drugs such as morphine and fentanyl from providing adequate analgesia in the early post-operative time period, depending on the length of surgery.

For these reasons, this author does not routinely use butorphanol, favoring more potent and more reliable opioids such as morphine and fentanyl. Fentanyl, a pure opioid agonist, is potent opioid with a very short duration of action. It should be used in very critical patients for analgesia, then as part of an induction protocol. When used as a premedication, induction of general anaesthesia should occur shortly thereafter, within approximately 20 minutes, or the drug should be given as a constant rate infusion until general anaesthesia is induced.

Phenothiazine tranquilizers, namely acepromazine, should be given to healthy animals as part of the premedication protocol. Acepromazine's antagonism of alpha-adrenergic activity can potentially induce vasodilation with subsequent hypotension, so should be used with caution. Other untoward side effects that have been reported include reduction of seizure threshold in predisposed animals. Thus, its use is relatively contraindicated in such patients. A potentially beneficial side effect of acepromazine is decreasing catecholamine-induced dysrhythmias.

Alpha-2 agonists such as xylazine and medetomidine induce intense peripheral vasoconstriction, AV nodal block, bradycardia, and decrease in cardiac output. For these reasons, alpha-2 agonists should never be administered to emergency and critical care patients for absolutely any reason. The alpha-2 agonists may have their place in healthy animals, but should not be used in emergent settings.

Anaesthetic Induction

Anaesthetic induction should occur rapidly. The most critically ill patients often will benefit from preoxygenation prior to induction. An intravenous catheter should be in place prior to induction, for maintenance of vascular access. Benzodiazepine tranquilizers such as diazepam (0.3 – 0.5 mg/kg IV) or midazolam can be used. Diazepam induces more reliable tranquilization and is less expensive than the more costly midazolam. If given alone, diazepam can potentially induce excitement; therefore, this drug is often used in combination with dissociative agents such as ketamine (5.5 mg/kg IV, 10 – 30 mg/kg IM), opioids such as fentanyl (10 mcg/kg IV), or in combination with etomidate (0.5 – 1.5 mg/kg IV). Ketamine is a dissociative anaesthetic agent that will initially cause a catecholamine-induced increase in cardiac output.

In critically ill patients that have maximised sympathetic tone, however, ketamine can decrease cardiac contractility; therefore, its use is relatively contraindicated. Ketamine, when used pre-intra- and post-operative, can decrease activation of NMDA receptor-mediated "wind-up" and decrease post-operative pain even days after surgery. Its use in combination with other analgesic agents is therefore beneficial, especially in controlling post-operative orthopedic pain. Propofol (4 – 7 mg/kg IV) is another drug that can be used to induce general anaesthesia. Unrelated to other pharmacologic agents, propofol induces rapid anaesthesia. Recovery from Propofol is also very rapid in most cases. Potential untoward effects of this drug include vasodilation and hypotension, and apnoea. In cats, Propofol should not be used on consecutive days due to the potential for development of Heinz body anaemia. In the most critically ill patients, Etomidate can be administered along with Diazepam with minimal effects on cardiovascular status.

Anaesthesia Maintenance and Monitoring

Immediately after successful induction of anaesthesia, anaesthesia should be maintained with an appropriate gas anaesthetic agent. For short procedures in cardiovascularly stable patients, Propofol can be administered as a constant rate infusion (6 – 20 mg/kg/hour). It must be remembered, however, that Propofol has no analgesic properties; therefore, if a painful procedure is to be performed, appropriate analgesia should be administered prior to recovery from anaesthesia. Proper anaesthetic monitoring including pulse oximetry, electrocardiogram, temperature, capnography, and blood pressure should be performed and recorded for even "apparently routine" procedures. In many critical patients, pre-, intra- and post- anaesthetic hypotension is a potential hazard that should be carefully addressed.

First, the level of gas anaesthesia should be decreased. Secondly, a fluid bolus (5 – 10 ml/kg) can be administered IV. All patients under general anaesthesia should have vascular access. If a patient is hypotensive and haemodilution or true anaemia is a concern, synthetic colloids such as Hydroxyethyl starch can be administered as an IV bolus (5 ml/kg). Alternatively, component blood products such as whole blood, packed red blood cells, or fresh frozen plasma can be administered depending on the primary problems at hand.

If decreasing anaesthetic depth and fluid bolus does not sufficiently increase blood pressure, use of a positive inotrope such as dobutamine (2 – 20 mcg/kg/min IV CRI) can be administered. Dopamine at lower doses (2 – 10 mcg/kg/min IV CRI) stimulates cardiac contractility through beta-adrenergic stimulation. Dobutamine, primarily a betaagonist, stimulates cardiac contractility and as such, indirectly increases blood pressure. Dobutamine increases blood pressure more reliably than dopamine. Ephedrine is a synthetic sympathomimetic drug that stimulates both alpha- and beta-adrenergic receptors to stimulate catecholamine release. Bolus injection of ephedrine (0.1 – 0.25 mg/kg IV) has a longer duration of action than dobutamine, thus does not require administration as a constant rate infusion. If none of the above options are successful, vasopressor agents such as dopamine (> 10 mcg/kg/minute IV CRI), epinephrine (0.05 – 0.4 mcg/kg/min IV CRI), and norepinephrine (0.05 – 0.4 mcg/kg/min IV CRI) can also be administered. It is important to remember that agents that induce peripheral vasoconstriction may increase blood pressure but not necessarily improve renal, cerebral, and coronary blood flow.

In any patient with tachy- or bradyarrhythmias such as AV block, bradycardia, or sinus or ventricular tachycardia, attempts should be made to treat the dysrhythmia. Anticholinergic drugs such as atropine or glycopyrrolate should be administered to treat cardiovascularly unstable bradyarrhythmias. In some cases, if the heart rate is 50, and the animal's blood pressure is stable and normal, treatment may not be necessary, However, if the animal is bradycardic and hypotensive, interventions should be implemented. Sinus tachycardia can adversely affect blood pressure by decreasing the amount of time the heart has to fill. Decrease in diastolic filling will result in a decreased cardiac wall stretch available for rebound, the force

necessary for normal myocardial contraction. Therefore, filling the cardiovascular space with fluids, and in some cases, INCREASING anaesthetic depth if the animal is actually feeling the procedure, may be necessary to decrease heart rate. Ventricular dysrhythmias should be treated with a combination of crystalloid/colloid therapy, oxygen, and drugs such as lidocaine (2 mg/kg IV bolus, followed by 50 – 100 mcg/kg/minute IV CRI) or procainamide. Procainamide can contribute to refractory hypotension, and is not the antiarrhythmic of choice in a patient with hypotension.

Post-operative Analgesia

Post-operative analgesia should be performed with the thought that the patient should never ever be allowed to be painful. Constant rate infusions of fentanyl (1 – 7 mcg/kg/hour), morphine (0.1 mg/kg/hour in dogs, 0.01 mg/kg/hour in cats) can be administered with ease. Additionally, at the suggested doses, the drugs cause minimal cardiovascular depression or ileus. Intermittent bolus injections can also occur, provided that the drugs are administered according to an exact schedule, not given when the patient begins acting apparently painful or "PRN".

Local anaesthetic agents can be placed in the wounds prior to wound closure for post-operative analgesia. Additionally, intrapleural lidocaine (1 mg/kg) or bupivacaine (0.5 – 1.0 mg/kg) can be administered via thoracostomy tubes for additional pain relief. Transdermal fentanyl patches are effective in controlling patient discomfort, but do not immediately take effect, thus requiring other types of analgesia until adequate blood levels of fentanyl are reached, usually 16 – 24 hours after placement of the patch.

Some clinicians advocate placing the fentanyl patch on the evening prior to surgery, to ensure adequate blood levels are obtained for the immediate post-operative period. Using combination multimodal approach to therapy such as a constant rate infusion of ketamine (0.5 mg/kg IV bolus at time of anaesthetic induction, then 10 mcg/kg/minute intraoperatively, then 2 mcg/kg/minute post-operatively as a CRI), a nonsteroidal anti-inflammatory drug such as carprofen or ketoprofen, epidural morphine, along with opioids helps to ensure that the patient never becomes painful. At the time of discharge, a combination of opioids and/or Nonsteroidal anti-inflammatory drugs should be considered for continued analgesia in the post-operative recovery period.

It is sometimes difficulty to distinguish between pain, anxiety, and opioid dysphoria. Physical examination parameters such as heart and respiratory rate, pupil size and responsiveness, should all be checked, but are often indistinguishable from pain, anxiety, need to urinate or defecate, or attention seeking behavior. When I suspect an animal may be painful or dysphoric, I perform a thorough physical examination, including palpation of the surgical site. If the patient reacts to surgical site manipulation, I immediately give an additional dose of analgesia. If no apparent pain is present with surgical site manipulation, but the patient quiets when being handled, anxiety or attention-seeking behaviour is diagnosed. I will add an anxiolytic agent to the treatment protocol, provided that hypotension is not a concern. If the patient neither responds to pain nor attention and anxiolytic agents, I make sure that the patient's urinary bladder is empty and there isn't a need to urinate or defaecate. Only after all of these choices have failed will I consider reversing an opioid drug with naloxone.

Conclusions:"What's the bottom line?"

In the emergent situation, there may not be enough time to take a "wait and see if this works" kind of approach. One of the most important things to remember is that gas is poison. Many animals, particularly those that are critically ill, are exquisitely sensitive to the cardiorespiratory effects of inhalant anaesthetic gases. For this reason, when an animal is hypotensive, one of the first things to consider is turning down the anaesthetic vaporizer. If there is concern about an animal waking up during the anaesthesia and surgery, balanced anaesthesia with constant rate infusions of fentanyl or fentanyl and ketamine can be administered, to decrease the total amount of anaesthetic gas required to maintain an adequate plane of anaesthesia without causing hypotension and cardiovascular compromise.

Next, (or sometimes simultaneously), a crystalloid (10 ml/kg) or colloid (5 ml/kg) fluid bolus can be administered, to fill up the vasodilated vascular beds. When a blood vessel dilates, a state of relative hypovolaemia occurs, in which there is inadequate circulating volume to maintain vascular tone and cardiac preload. If there is insufficient myocardial stretch, the force of contraction is limited, and thus, can result in impaired cardiac output and a decrease in systemic blood pressure.

Many anaesthetic agents render the cardiovascular system incapable of compensatory changes such as vasoconstriction, so blood pressure and thus tissue perfusion and oxygen delivery become compromised. If decreasing the anaesthetic depth and administration of fluids does not cause an increase in blood pressure, then positive inotropes (dobutamine and/or ephedrine) and vasopressors (dopamine) can be administered.

Having an "anaesthetic book" that contains charts of all of the necessary drugs, instructions on how to dilute each drug, resulting concentration, and volume of the diluted drug to administer to each patient based on body weight can save a lot of time and quick arithmetic in an emergent situation.

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Managing Immune-Mediated Haemolytic Anaemia

Carla Johnson, DVM

Immune-mediated haemolytic anaemia (IMHA) is far from a simple disease with a straightforward treatment plan. Difficult to treat and confusing to manage over time, this disease has many clinicians referring patients to internists for both short- and long-term management. The difficulties may be due largely to the fact that each patient is so different in how they present, how the disease progresses, how they respond to treatment and how they fare over the long term:

- **How they present:** A patient with IMHA may present clinically normal with abnormal routine lab work, or may come in nearly dead.
- **How they progress:** A dog with questionable bloodwork can crash in a haemolytic crisis within 24 hours after a normal physical exam.
- **How they respond to treatment:** The protocol that keeps one dog alive can appear to be useless for another patient, or a protocol that worked initially can appear ineffective after a relapse.
- **How they do long term:** One dog never relapses, the next never seems to stop relapsing or ever get out of its initial crisis.

At a recent Fetch dvm360 conference, Elisa Mazzaferro, MS, DVM, PhD, DACVECC, a criticalist at Cornell University Veterinary Specialists and adjunct associate clinical professor at Cornell University College of Veterinary Medicine, shared her approach to IMHA cases. A previous article discussed Dr. Mazzaferro's insight on the causes and diagnosis of IMHA.[1] Here, she sheds light on short- and long-term treatment, as well as patient monitoring.

Emergency and short-term therapy

Initial treatment for IMHA depends on the severity of the clinical presentation, the clinician's preference and the owner's cost limitations. Any moderately affected patient is better off being hospitalised than being monitored at home, but this is not always something you can convince owners to do.

First, Dr. Mazzaferro said, identify and remove any inciting cause, if possible. These may include ticks, pennies or other metals (e.g., zinc intoxication); note that zinc-containing ointments such as sunblock and Desitin can also be culprits.

Glucocorticoidsandotherimmunosuppressive agents

"Steroids, steroids, and more steroids" - this is the one and only proven treatment, and sometimes it is

the only immunosuppressive treatment chosen, Dr. Mazzaferro said, adding that "you cannot skip steroids, but you will need to wean them sooner or later and you may need to add a second drug." The current recommended dose for prednisolone is 1 to 2 mg/kg/day, divided **(it is no longer 2-4 mg/kg/day)**. With vomiting animals, you can start with injections of dexamethasone sodium phosphate at about 0.1 to 0.2 mg/kg IV every 12 hours, which is roughly equivalent to the prednisolone dose.

If there is proof of autoagglutination, or in severe cases that require multiple transfusions, Dr. Mazzaferro will start a second immunosuppressive drug and thromboprophylactic therapy right away, but when to do this is not clear from the literature (see Is combination therapy effective?). None of the drugs below have proven efficacy by themselves in patients with IMHA[2]:

- Cyclosporine suppresses T cells and inflammatory cytokines and has a rapid onset; the dose is 5 to 10 mg/kg PO every 12 hours. Side effects include infections, gingival hyperplasia and, occasionally, renal or liver toxicity.
- Azathioprine suppresses T-cell function, and takes one to two weeks to start working; the dose is 2 mg/kg/day PO for five to seven days and then every other day. It can cause vomiting, diarrhoea, pancreatitis and myelosuppression.
- Mycophenolate inhibits T and B cells, and inhibits B-cell antibodies; the dose is 10 mg/kg/day PO or IV. This drug has variable bioavailability, and can cause lethargy, vomiting, diarrhoea, lymphopenia and pyoderma.
- Leflunomide, a newer agent, blocks both T and B cells; the dose is 2 to 4 mg/kg/day PO. Side effects include vomiting, diarrhoea and bone marrow suppression. (Leflunomide is not used commonly — consult with your local internist.)

Is combination therapy effective?

Dr. Mazzaferro noted that "at this time, there are no studies to support combination therapy of other immunosuppressive agents with glucocorticoids versus administration of glucocorticoids alone." According to the American College of Veterinary Internal Medicine guidelines[2] for treating IMHA, a second drug may be added if the patient is very severely affected on presentation, packed cell volume does not stabilise within seven days, the dog continues to be transfusion dependent over seven days or the patient is having severe adverse effects from corticosteroids. The choice of which drug to add may be based on several factors:

- How quickly you want the second drug to start working (i.e., how quickly you want to start weaning prednisolone or how quickly you want the immune system suppressed — maybe faster with a severely affected cocker spaniel, for example)
- What is available
- Cost
- What the internist you consult with recommends

Transfusions

If your patient is symptomatic for anaemia (weak, pale, elevated respiratory rate or heart rate), or the packed cell volume (PCV) is 13% or lower, immediate transfusion is required. This will likely need to be done at a 24-hour care facility using the freshest packed red blood cells (RBCs) available.

Note that cross-matching can be difficult for dogs that are already macroagglutinating, but the RapidVet-H cross-match kit (DMS Laboratories) is not supposed to be affected by this. If the dog has never had a transfusion, it can be done without cross-matching. If the patient is four days or more out from a recent transfusion, you must cross-match as they may have started making antibodies against foreign RBCs already.

Other medical therapies

Several other medications can also be used to treat IMHA. Crystalloids (ideally, intravenous fluids) can help flush out bilirubin, which can be nephrotoxic.

An estimated 50% to 80% of dogs with IMHA develop life-threatening thromboembolic complications. Steroids are pro-coagulable, and the systemic inflammation from haemolysis can trigger clots, consumption of clotting factors and other clotting dyscrasias. To prevent these complications, patients should be started on one of the following anticoagulant drugs:

- Aspirin: 0.5-1 mg/kg/day PO (can be compounded for small dogs) Dr. Mazzaferro noted that this dose is largely ineffective as an antiplatelet drug in dogs, but this is the "recommended" dose.
- Clopidogrel: 10 mg/kg/day PO loading dose on day 1, then 2 mg/kg/day PO
- Rivaroxaban: 0.8 mg/kg/day PO
- Unfractionated heparin: may be useful only if dose adjustments are based on an individual patient's daily anti–factor Xa activity measurements, so it is not widely used or recommended at this time
- Fractionated heparin (enoxaparin): 0.8 mg/kg SC every six to eight hours, with dose adjustments based on anti–factor Xa activity

Gastrointestinal protectants, such as proton pump inhibitors, histamine blockers, anti-emetics and/or sucralfate, may also be considered for these patients. Dr Mazzaferro prefers to start doxycycline empirically at 5 to 10 mg/kg every 12 to 24 hours in all patients with IMHA. She will stop if there are signs of gastrointestinal

upset and restart it later if indicated by the infectious disease panel. Doxycycline can be discontinued if an infectious cause is no longer suspected.

Monitoring and long-term therapy

These are tailored to the individual patient and also guided by clinician preference. Monitoring and long-term treatment can get complicated, but here are some basic guidelines.

- Once the patient is out of the hospital, recheck the PCV/total protein every seven to 14 days initially, then every two to four weeks, and do blood smears and a full complete blood cell count (CBC) as often as owners will allow. Each CBC should have an add-on review by a pathologist.
- Taper the prednisolone when
    1. PCV has risen to a normal level,
    2. there are no longer any reticulocytes or evidence of regeneration, and
    3. all of the spherocytes and abnormal RBCs (including nucleated RBCs) are gone from the comprehensive CBC with a pathologist's review.
- Editor comment - you need to compare the changes in PCV with the degree of regeneration on the blood smear. If there is a gradual increase inPCV but still a very regenerative blood smear then haemolysis is still present and you shouldn't taper. Do not just rely on the PCV. (You can roughly monitor in-house with cytology if you are skilled, Dr. Mazzafero said, but send blood to the lab for confirmation that everything has normalized). Do not taper too hastily. Decrease prednisolone by 25% and then recheck every three to four weeks. If the pathologist's review reveals ongoing evidence of IMHA (spherocytes, reticulocytes, anaemia, abnormal blood cells or recurrence of anaemia), increase the prednisolone dose. If CBCs remain normal with no evidence of reticulocytosis or spherocytosis, lower the dose by another 25% of the original dose every four to six weeks. Wean cortisteroids in total over three to six months, and be aware that relapse can occur at any time. In the face of an altered immune system, regularly check for urinary tract and skin infections, and treat these based on culture and sensitivity results. Stop the anticoagulant therapy when prednisolone is stopped.If using adjunctive cyclosporine, start to decrease it in the same way as the prednisolone, at about 20 weeks out, and check lab work monthly.

Summary

Due to patient variability, there will be many exceptions to these guidelines. More often than not, Dr. Mazzaferro says, you will seek a consultation with or refer to an internal medicine specialist— and you should. With a complicated disease, however, having some simplification allows us to manage a good portion of the care, helps us catch relapses and diagnose the disease on initial presentation, and allows us do our part in co-managing these cases. Maybe we can save more of them.

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

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ACVIM Consensus Statement on the Treatment of IMHA in Dogs

Journal of Veterinary Internal Medicine (2019) 33:1141-1172
Summarised by Dr L. L. van der Merwe BVSc MMedVet (Med) Small Animals
Please Note that the Journal of Veterinary Internal Medicine is Open Access

Results and Recommendations of the Panel

1. Timing of Treatment

- Complete diagnostic tests as far as possible before instituting treatment (to enable proper detection of underlying disease example lymphoma) , where the delay is not life-threatening.

2. Blood typing and cross matching

- Clinicians must be aware that auto-agglutination will affect interpretation of blood typing kits and cross matching results.

3. Blood transfusion and blood products

- Recommend that packed red blood cells (pRBC) not older than 7 – 10 days are administered when a dog with IMHA shows clinical signs of decreased oxygen delivery to the tissues. Whole blood is a reasonable alternative. Older units of blood have shown increased risk of complications and mortality.
- Review of the literature did not identify any studies which showed an association between the number of,or the volume of blood transfusions with mortality in dogs with IMHA.
- There was no recommendation to administer fresh frozen plasma to dogs with IMHA as a tool to prevent disseminated intravascular coagulation.

4. Immunosuppressive treatment

- Prednisolone at 2-3mg/kg/day or 50 – 60mg/m2 for dogs >25kg be initiated after the diagnosis of IMHA is made. The drug may be administered as single dose or divided into a BID treatment.
- Dexamethasone can be administered at 0.2-0.4mg/kg/day if the oral route is not considered viable initially.
- If the starting dose of prednisolone is >2mg/kg/day it should be tapered to <2mg/kg/day within 1-2 weeks of initiating treatment, provided the dog is responding to treatment (*as interpreted by the rate of change of the PCV and the degree of regeneration in the blood smear – LvdM*).
- A second immunosuppressive drug may be introduced from the outset to reduce the dose of corticosteroids. There is weak evidence, mainly based on clinical experience of the panel, to support the suggestion that 2 immunosuppressive agents are used in the following situations:
  - The dog has severe or life-threatening disease at presentation.
  - The PCV does not remain stable and shows an absolute decrease of ≥ within 24 hrs , at any stage during the first 7 days of treatment with prednisolone as described above.
  - The dog continues to be dependent on blood transfusions after 7 days of treatment as described above.
  - The dog develops or is expected to develop severe side effects due to the corticosteroid treatment. This is especially true of dogs > 25kg *(LvdM - large breed dogs show severe muscle catabolism and weakness due to corticosteroid therapy - this is not as evident in small breed dogs*).

- The benefit of adding a second drug has not been established beyond a doubt in the various publications. The panel suggests the following options listed in alphabetical order, if a decision is made to add a second medication.
  - Azathioprine: 2mg/kg or 50mg/m2 PO q24h. After 2-3 weeks, the dosing frequency can be decreased to every second day (q48h).
  - Cyclosporine: 5mg/kg PO q12h. the dose may be adjusted according to therapeutic drug monitoring.
  - Mycophenolate mofetil: 8 – 12mg/kg PO q12h.
  - Leflunomide has the least evidence supporting its use: 2mg/kg PO q24h.
  - There is strong evidence that cyclophosphamide is NOT recommended in dogs with IMHA.
  - IV immunoglobulin has strong recommendadtion as a salvage measure but is not recommended for routine treatment. IVG administered at 0.5 -1 g/kg as a single infusion.

- The panel suggests that the use of three immunosuppressive medications be avoided as 3 drugs are rarely required for management and the risk of adverses effects is increased with the combinations.
- When the PCV has remained stable and >30%

for 2 weeks after starting treatment then it is recommended to start decreasing the dose of prednisolone by 25%. If the patient is on a second drug , his dose should be maintained and the prednisolone could be decreased in larger increments (25-50%). Providing the PCV remains stable or continues increasing a dosage decrease of 25% of the current dose every 3 weeks is recommended. A typical duration of treatment is 3-6 months for prednisolone treatment and 4-8 months for all immunosuppressive treatment.

- The panel recommends assessing the PCV/Ht before any dose reduction to ensure continued response to treatment. (*LvdM – reliance on only clinical signs may result in a sever decrease in PCV before a relapse of the IMHA is noticed*).

5. Monitoring adverse effects from immunosuppressive treatment

- Commonly observed clinocopathological changes due to corticosteroid therapy are a stress leukogram, increased ALP activity (induced), polycythaemia, thrombocytosis, hyperlipidaemia and hyperglycaemia.
- Subclinical bacteriuria is common in dogs on long term glucocorticoid treatment.Guidelines for treatment of urinary tract infections suggest that antibiotics should be considered in these patients due to their immunosuppression and the risk of an ascending or systemic infection.
- Patients on azathioprine should have CBC and ALT activity monitored every 2 weeks during the first 2 months of therapy. Azathioprine can cause myelosuppression and hepatotoxicity in some dogs. Pancreatitis is also noted in some patients.
- Mycophenolate may cause severe ulcerative colitis and need to be discontinued in some patients. GIT signs at the higher dosage range may necessitate dose reductions.

6. Approach to relapse

- Retrospective studies show a relapse rate of about 11-15%.
- Confirm a relapse using the standardised criteria for diagnosing IMHA (Fig. 1) Anaemia could be caused by some of the medications being used due to GIT bleeding, bone marrow suppression etc.
- Assess the patient for any other trigger factors, such as infections.
- If the relapse occurs before any dose adjustments, add a second immunosuppressive drug.
- If the relapse occurs during drug tapering the panel suggests increasing the dose again, and tapering more slowly once remission has been re-established - double the time interval between dose reductions.
- If recurrent relapses occur, lifelong "lowest dose possible" treatment may be required. Alternatively there may be weak evidence for performing a splenectomy if infection with tickborne disease has been properly excluded. Two retrospective case series showed excellent survival post splenectomy (no control cases were included – which makes the studies statistically weaker). (*LvdM – It is vital that proper tick control measure are applied pre and post operatively*).

7. Emerging immunomodulatory treatments

Therapeutic plasmapheresis, liposomal clodronate, melatonin and hyperbaric oxygen therapy have been reported but need more investigation.

8. Antithrombotic treatment

- It is well recognised that dogs with IMHA are at an increased risk for thrombosis and that thrombotic disease is the leading cause of mortality and morbidity in dogs with IMHA. Immunosuppressive agents such as prednisolone also causean increased risk of thrombosis.
- Thromboprophylaxis is strongly recommended in all patients with IMHA except those where platelet count is low (<30 x109). Treatment should be initiated at time of diagnosis and continued until the patient is in remission and no longer on prednisolone. The greatest risk for thrombosis is within the first two weeks after initiation of treatment.
- Dogs with IMHA are predisposed to venous thromboembolism, and there is weak evidence suggesting that a regimen including anticoagulants as well as antiplatelet drugs may be prudent in the first two weeks.
- Drug Recommendations
  - Clopidogrel: 1.1- 4 mg/kg PO q24h. a single PO loading dose of double the maintenance dose up to a max of 10mg/kg may be useful to reach therapeutic plasma levels rapidly.
  - Aspirin has shown efficacy in dogs for arterial thrombi, butthere is insufficient evidence available for the prevention of venous thrombi. It should be administered at 1-2mg/kg PO q24h together with clopidogrel.
  - Injectible low molecular weight heparins are the most feasible anti-coagulants. Individual dosage adjustments using an anti-factor Xa assay is recommended , but not necessarily available. There is no normogram,s to adjust antiXa to aPTT activity in dogs . (*LvdM - The suggested doses often need escalation based on individual monitoring at referral facilities. So there is no real guidelines available for general practice – research properly if intending to use*).
  - Suggested starting doses are: Dalteparin(SC) 150 – 175 U/kg q8h; Enoxaparin (SC) 0.8 – 1.0 mg/kg q6-8h, Rivaroxaban (PO):1-2 mg/kg q24h.

9. Supportive care and antibiotic treatment

- Gastroprotectants should only be administered if there is evidence of GIT ulceration or there are

additional known risk factors for the development of GIT ulcers . If indicated a proton pump inhibitor is the drug of choice, given BID and is discontinued as soon as clinical signs abate.

- The risk of infection with vector borne pathogens should be assessed and treatment instituted whilst waiting for test results.

10. Monitoring dogs in remission

- Once remission is attained and treatment is stoped the panel recommends continued monitoring for four weeks and that no further testing is required beyond this.

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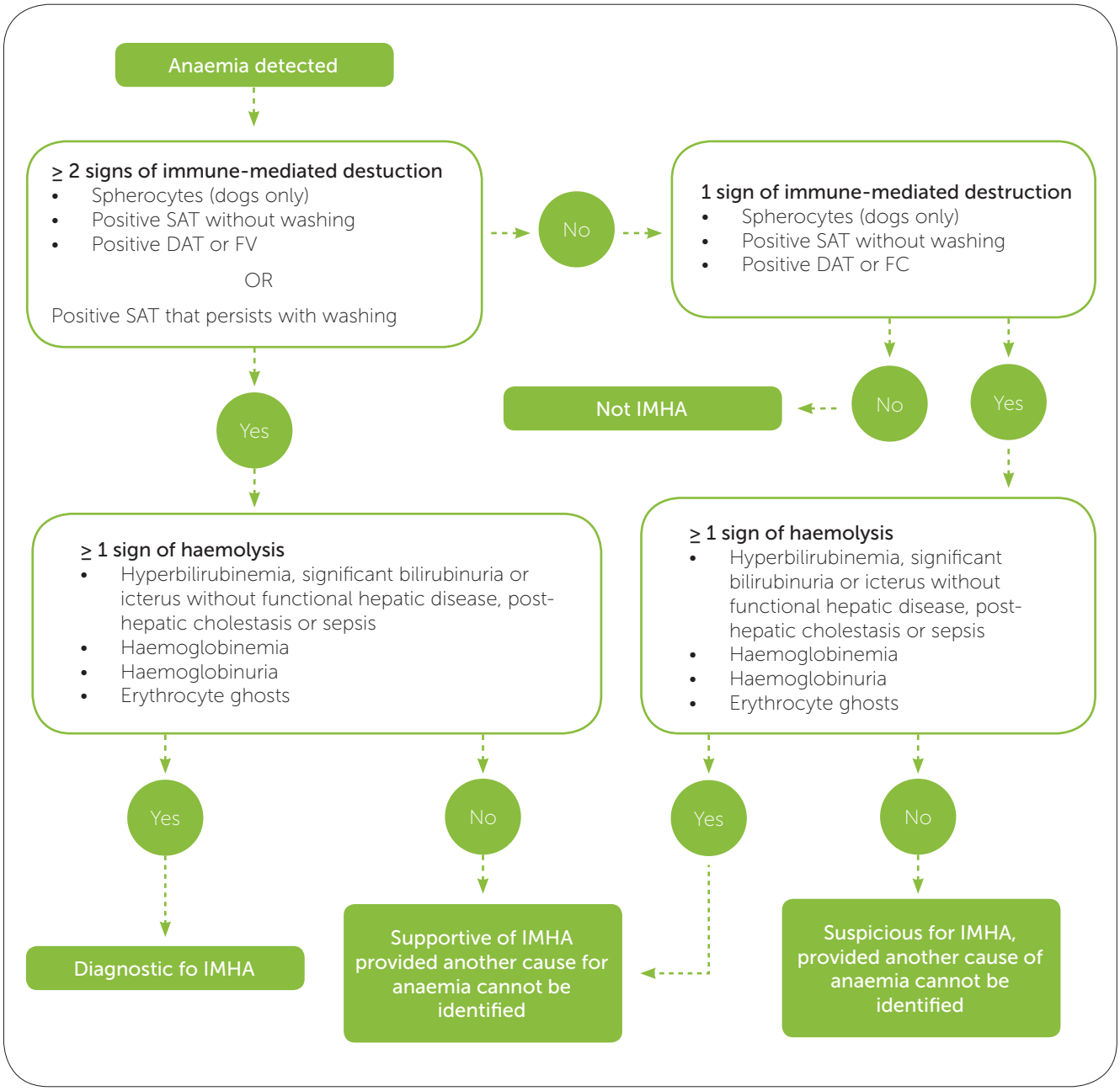

Fig. 1 - Diagnostic algorithm for immune-mediated haemolytic anaemia (IMHA). Having identified anaemia in a patient, biomarkers of immune-mediated destruction should next be assessed, including the saline agglutination test (SAT), direct antiglobulin test (DAT), and/or flow cytometry (FC); at least 2 should be present, or a positive SAT that persists with washing, to make a firm diagnosis of IMHA. Signs of haemolysis should then be assessed, at least 1 of which should be present for a firm diagnosis. Variations on this theme would yield a supportive or suspicious diagnosis, provided another cause of anaemia is not identified. Additional abbreviations: ≥, at least; dz, disease

Glossary and Definitions in Canine

Blood Transfusions - Separate Your Ps From Your Qs

Summarised from Review Article: Transfusion Medicine: An update on Antigens, Antobodies and Serologi testing in Dogs and Cats. Zarema, R, Brooks A, Thomovsky E. Topics in Companion Animal Medicine (2019) Vol 34: 36 -46.

Summarised by Dr L.L. van der Merwe BVSc MMedVet (Med) Small Animals

Antigen - any structure which induces an immune response in an individual

Epitope - the specific portion of an antigen which is bound by an antibody. A single antigen can have multiple different epitopes.

Polyclonal antibody - a collection of antibodies which all recognise the same antibody at different epitopes . Is made by exposing an animal to the antigen and harvesting serum - example snake antivenom.

Monoclonal antibody - multiple copies of a single antibody all binding to the same epitope is a monoclonal sample - these are usually engineered in a laboratory.

Alloantigens - "self" markers to which the individual does not usually react. These alloantibodies may cause an innume response in a different individual of the same species. Alloantigens are found on red blood cells .

Antigenicity - refers to how strongly an antigen can stimulate an immune response

Alloantibodies - can be present in an individuals' blood and cause no problems, but will react to blood from another individual.

Auto-antibodies - antibodies attacking antigens of the individuals own cells - e.g. IMHA

The presence or lack of alloantibodies, as well as the degree of immune response they induce, determines whether or not blood can be transfused without pre-transfusion testing.

Alloantibodies which produce a weak immune response, i.e. of low antigenicity, are considered to be of low clinical significance.

Research has shown that although alloantibodies against certain blood types(e.g. DEA 7) are present in transfusion naïve dogs - they are considered to be clinically insignificant. The first transfusion in dogs is thus generally considered to be "safe", the risk of an acute **haemolytic transfusion reaction** is very low. Exposure to transfused antigens recognised as foreign by the recipient will lead to alloantibody formation within 3 – 7 days of exposure. Subsequent transfusions are thus more likely to result in immune reactions. Humans and cats have naturally occurring alloantibodies to non-self blood from birth and pre-transfusion testing is critical.

Mechanisms of transfusion reactions.

Agglutination indicates that an antibody - antigen reaction is occurring on the red blood cell, sufficient to allow cross linking and clustering of the red blood cells. Antigen antibody reactions against the red blood cells can also be more subtle - not actually causing crosslinking - but still causing clinical disease (ISA negative IMHA). Dilution with saline is essential to exclude severe rouleaux formation.

Antigen antibody reactions can also be more severe and induce complement and cause red blood cell destruction - intravascular haemolysis. Extravascular haemolysis causes hepatosplenomegally and icterus. Intravascular haemolysis causes haemoglobinuria and can cause a systemic inflammatory response.

Blood types

Blood types are markers on the surface of the red blood cell. These molecules have other cellular functions (proteins, signalling molecules, ion channels) but have become recogniseable antigens for red blood cell identification in proportions of the population.

The original DEA system (Dog Erythrocyte Antigen) described blood types DEA 1.1, 1.2, 1.3, 3, 4, 5, 6, 7 and 8. Commercial blood typing is only currently available for DEA 1, 4, 5 and 7.. DEA 1 is the only commonly screened blood type which may cause an

acute haemolytic transfusion reaction. A haemolytic transfusion reaction is a type 2 hypersensitivity reaction and can occur within minutes to hours of a transfusion.

New research has demonstrated that DEA 1 is a single antigen, with an autosomal dominant inheritance pattern, with 4-5 alleles. Dogs are thus either negative or positive for DEA 1. If positive the degree of positivity (on the gel tests) can be 1+ to 4+ depending on the gene inherited. This degree has not been proven to correlate to the severity of the transfusion reaction. So it is best to consider dogs either DEA 1 positive or negative, and leave it at that.

Depending on the study population 30 – 60% of dogs are DEA 1 positive. There are distinct breed variability in the expression of this marker. Cross breeds are about 50% positive (JSAVA).

Delayed transfusion reactions occur when there is a lower level of antibodies or the response to the antibody is not haemolytic in nature. Shortened red cell lifespans have been described with DEA 3, 5 and 7. DEA 4 is present in 98% of the population. Dogs negative for DEA 1,3,5 and 7 and positive for 4 are considered universal donors.

There are many other antigens present on the red blood cell which we have not identified and may sensitize a patient to a second transfusion from the same or another donor. Two recently describe antigens are Dal and Kai1/Kai2. There is no commercial testing available for these.

Screening donor blood to prevent transfusion reactions

Blood typing can determine the presence of a known antigen. Cross matching will determine if a general antibody and / antigen reaction against red blood cells is occurring in the sample, but does not identify the specific antigen.

Blood type testing

Blood typing focuses on screening donor blood for the most antigenic blood types to minimise the risk of a transfusion reaction. Inhouse card testing (Rapivet®) and gel column (Avedia®) testing are available and have excellent sensitivity and specificity (> 90%) although the gel assay is marginally more accurate as there is less subjectivity in the interpretation of the result The result is limited to the antibody we are testing for. Generally we just test to confirm whether the patient/donor is DEA 1 negative or positive. (Author - DEA 1 negative donors are preferred as if sensitisation occurs in the recipient it will not cause a haemolytic transfusion with a subsequent transfusion).

Cross matching

The cross match is done in two parts: **a major and a minor cross match.**

1. **Major cross match: Donor RBC into recipient plasma - testing for host antibodies**
   My basic little story to understand: the host has a lot of plasma to provide antibodies for , and can produce more , for that small amount of RBC so the chances are there will be enough to induce an immune reaction.

2. **Minor cross match: Donor plasma into recipient RBC - testing for donor antibodies**
   My basic little story to understand: the donor plasma is a relative small and finite volume in relation to the recipient pool of RBC - so the chances that ere will be enough to induce an immune - reaction is less.

Even with a compatible crossmatch test red blood cell destruction may still be accelerated in the recipient as low titres may not cause enough agglutination for detection, yet the body will still mount an immune response – as the immune system is the "super sensitive gold standard" to detect antibodies.

Crossmatch testing is recommended in any dogs with a history of a previous transfusion or unknown transfusion history. Because dogs lack alloantibodies, only 15% of referral practices routinely crossmatched prior to blood transfusion (JEVCC).

The risk of acute haemolytic transfusion reactions was not increased by not performing a crossmatch, however, using crossmatching the blood did increase the duration of red blood cell survival of the recipient. (The author recommends crossmatching in disease conditions where red blood cell regeneration is compromised and repeated transfusions are anticipated. These patients are usually not emergency transfusions so there is time to organise the sample to the laboratory).

Where the transfusion is indicated due to haemorrhage or other regenerative causes of anaemia the use of DEA 1 negative donors should be sufficient to prevent transfusion reactions in previously transfused patients. Any delayed transfusion reaction will be offset by the patients' own red blood cell regeneration.

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CPD Questions

AC/0309/20

Managing Immune-mediated Haemolytic Anaemia

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 a33230

01. Which one of the following medications is the mainstay of treatment of IMHA in dogs?

a. Corticosteroids.
b. Azathioprine.
c. Cyclophosphamide.
d. Cyclosporine.
e. Mycophenolate mophetil.

02. Which one of the following statements regarding medications for IMHA is INCORRECT?

a. Cyclosporine suppresses T cells and inflammatory cytokines.
b. Cyclosporine has a rapid onset of action.
c. Azathioprine suppresses T-cell function.
d. Azathioprine has a rapid onset of action.
e. Bone marrow suppression occurs with azathioprine and leflunomaide.

03. Which one of the following characteristics of DEA 1 is most clinically relevant?

a. DEA1 is an allele.
b. DEA1 is present in 30 – 60% of dogs.
c. There is a breed variablility in the prevalence of DEA 1.
d. DEA 1 is a heamagglutinin.
e. DEA 1 is an alloantibody.

04. Which one of the following staements regarding transfusion therapy in dogs is INCORRECT?

a. Transfusion is indicated if a patient with anaemia has an elevated respiratory and heart rate.
b. Transfusion is indicated if a patient with anaemia has a packed cell volume (PCV) of 13% or lower.
c. If the dog has never had a transfusion before it can be done without cross-matching.
d. If the patient is four days or more out from a recent transfusion, you don't need to cross match.
e. Blood from multiple donors can safely be given in the first 3 days without expecting a haemolytic transfusion reaction.

05. Which of the following statement regarding complications of IMHA is INCORRECT?

a. Systemic inflammation is caused by the haemolysis.
b. Only infectious causes of haemolysis causes systemic inflammation.
c. Thromboembolisms is a very prevalent cause of mortality in IMHA.
d. Corticosteroids are procoagulable.
e. Inflammation causes hypercoagulation.

06. A cross match test will determine if a reaction between the recipient and donor will occur but will not identify the antigen. Which one of the following statements is CORRECT?

a. The cross match is done in as a major cross match looking for recipient antibodies.
b. A Major cross match is Donor RBC into recipient plasma.
c. A Major cross match is Recipient RBC into donor plasma.
d. A cross match is definitive for a safe transfusion.
e. Washing of the red blood cells is only necessary of the patient has IMHA.

07. Which one of the following statements regarding transfusion reactions is INCORRECT?

a. Delayed transfusion reactions occur when there is a lower level of alloantibodies.
b. Delayed transfusion reactions occur when the response to the antibody is not haemolytic in nature.
c. Blood typing can determine the presence of a known antigen.
d. Shortened red cell lifespans have been described with DEA 3, 5 and 7.
e. Only the DEA antigen group is responsible for transfusion reactions.

08. Which one of the following statements regarding blood transfusion and blood products for IMHA is CORRECT?

a. Consensus recommends that packed red blood cells (pRBC) not older than 7 – 10 days are transfused.
b. Consensus recommends that whole blood is transfused - not older than 7- 10 days.
c. Consensus recommends that fresh whole blood is transfused.
d. Consensus recommend that fresh frozen plasma is used to limit the development of DIC.
e. Consensus recommend that the number of transfusions given is related to the degree of mortality and should be used sparingly.

09. Which one of the following statements regarding the use of prednisolone treatment for IMHA is INCORRECT?

a. Prednisolone is used at 4 – 6 mg / kg divided BID for initiation of treatment.
b. Prednisolone should be administered at 50 - 60mg/m2 for dogs >25kg.
c. The prednisolone dose is relatively lower, in mg/kg ,for larger heavier dogs.
d. When the PCV has remained stable and >30% for 2 weeks after starting treatment then it is recommended to start decreasing the dose of prednisolone by 25%.
e. Continues increasing a dosage decrease of 25% of the current dose every 3 weeks is recommended.

10. Which one of the following statements regarding thromboprophylactic drugs is INCORRECT?

a. Thromboprophylaxis is strongly recommended in all patients with IMHA without exception.
b. Treatment should be initiated at time of diagnosis and continued until the patient is in remission and no longer on prednisolone.
c. The greatest risk for thrombosis is within the first 2 weeks after initiation of treatment.
d. Dogs with IMHA are predisposed to venous thromboembolism.
e. Anticoagulants as well as antiplatelet drugs are suggested in the first 2 weeks.

How to Create a Veterinary Practice Website That Wows

Robert Sanchez

A hospital website that is both visually and emotionally inviting will generate more clients. Here's how to make yours stand out.

The primary purpose of any veterinary practice website is to attract new clients, but what many people fail to recognize when creating their site is that the brain operates using 2 distinct neural systems that affect the viewing experience of potential clients. The strategy behind many practice sites speaks to the brain's cortex, with straightforward facts and logic about who you are, what do you do, and where and when do you do it. But the brain's cortex does not control emotions, social relationships, or decision making—that's the job of the limbic system. And the limbic system is asking a different question altogether: Can I trust you?

The best websites create a compelling and emotional user experience, speaking directly to the limbic brain. This article introduces the actors in this fascinating play, outlines how trust emerges, and offers some practical steps you can take to attract the right clients via your website.

Speak to the limbic system

The brain's cerebral cortex gives us the ability to reason, plan, and understand language. It is the defining feature of humanity and our brain's most recent addition—it likely evolved to help our ancestors become savvier masters of their terrain, solve more complex problems, and think ahead. The limbic system, on the other hand, controls our emotions and social behavior. It is full of practical vestiges of our early life as mammals, enabling us to quickly discern friend from foe, sew the seeds of cooperation, and bond with others.

When we meet someone new, our limbic system is always working out that question of trust: Should I put my faith in you? Are you safe to cooperate with? Or should I be wary? When trustworthiness is absent, you can feel it. You may experience this lack of trust when dealing with a seedy mechanic, when peering down a dark alley, or when engaging with a product or service on a website. The limbic system's evaluation of social relationships is always at work, sussing out who we can cooperate with and who we should steer clear of.

If your website is not designed strategically to speak to the limbic brain, most pet owners will feel uncomfortable proceeding. They may rationalize their trepidation—perhaps they would like to look at other options, or maybe they're just too busy to make a decision right now, among other reasons. But the real reason they choose not to check out your practice is because nothing on your website told them they could trust you.

Build trust

The subconscious brain acts fast, so communicating through emotion from the outset is the key to speaking to the limbic system. Within fractions of a second of viewing your site, the user starts discerning the safety and attractiveness of working with you. Canadian journalist Malcolm Gladwell calls this phenomenon "thin slicing," and behavioral economists call it the "representativeness heuristic." Basically, it means that we extrapolate early evidence to fill in the gaps that what we don't know about someone or, in this case, a business. For example, if your site is modern, users are more likely to think you have a more modern approach to veterinary medicine.

Providing that evidence for the potential client viewing your website is not difficult. Your site's fold—the area that appears on screen before scrolling down—should be contemporary and attractive, with messaging that accurately reflects your beliefs about veterinary care (more on this in a bit).

The best websites make you forget you are even on a website at all. Today's software makes it easy to layer in HTML5 video backgrounds instead of still images. You can find plenty of stock videos that create an emotional experience for users, but the gold standard of website introductions is an HTML5 video background created with custom footage from your practice that captures interactions and connections among your team, your patients, and your clients. This is where the magic happens.

Get the messaging right

An uncluttered website filled with pictures or videos that evoke genuine emotional experiences is only one side of the coin. The other, more neglected element is messaging. Most veterinary practice websites use the same tone and tenor of messaging—mainly because they are speaking to the cortex. So, how do we speak to the limbic part of the brain?

It's simple: Flip the script. Instead of writing primarily about your practice, write about how everything you do affects the pet owners who visit your practice. Here's an example courtesy of Countryside Veterinary Hospital in Toney, Alabama.

You will notice a few things here. First, this image effectively illustrates the care, attention, and expertise of people working in the practice. You'll also notice that the messaging highlights the hospital's special workplace culture and its positive impact on pet owners and their animal companions. This practice is doing a great job of showcasing it strengths.

In the following example from the same website, Countryside makes it clear that one of its primary differentiators is the quality and depth of its team. But instead of crowing about how great they are, the website drives it home by defining how its team actually makes a difference in pets' health. Again, the subject isn't the practice but rather the pet owner. Your website should highlight the main strengths of your practice. Intersperse pictures or videos that help tell the story, and drive the point home in a way that makes it all about the pet owner.

Next steps

Evaluate your practice website, page by page. Ask yourself about the emotional experience are you creating for visitors. Are you speaking to them in a meaningful way? Are you demonstrating empathy, building trust, and artfully positioning your skills and differentiators? The ultimate goal is to create a website that peels back the layers of the onion, gets to the heart of why you do what you do, and tells a story that makes the visitor go … "wow."

Robert Sanchez is the founder and CEO of Digital Empathy, an award-winning web design and marketing firm for veterinary practices. He frequently lectures at national conferences, leads a team of wonderful employees, sits on the board of VetPartners, and shares his home with two very spoiled dogs—Cole and Lula.

How to be a Veterinary Social Media Superstar

Where does your veterinary practice need to be online? How much work will this take? Is it worth it? We asked practice owners, social media and marketing team members and other experts for the answers.

There's no denying that social media is an invaluable marketing tool for veterinary practices. In fact, most would argue that it's vital for success today. But anyone with a Facebook page or a Twitter account knows that reaching peak popularity is not as simple as sharing a photo or adding a hashtag. So how can you run a successful practice while managing winning social media pages?

We talked to six veterinary social media mavens who've proven they have the know-how to navigate the intricacies of social media and create content that sparks engagement. Here's what they had to say.

Who's who

Laurie Hess, DVM, DABVP (avian practice)
Dr. Hess owns Veterinary Center for Birds and Exotics in Bedford Hills, New York, and is the author of Unlikely Companions: The Adventures of an Exotic Animal Doctor. She frequently appears as a lecturer, author and exotic pet expert in the media.

Laurie Hess Facebook; Twitter: @DrLaurieHess

Karin Kandur
Kandur is a marketing manager for Compassion-First Pet Hospitals and manages the social media accounts for multiple New Jersey veterinary hospitals, including Red Bank Veterinary Hospitals in Hillsborough, Mount Laurel and Tinton Falls.

Red Bank Veterinary Hospitals Facebook @RedBankVet; Instagram: @redbankvet; Twitter: @RBHVet

Kate Lawrence
Lawrence is marketing coordinator for Gulf Coast Veterinary Specialists in Houston, Texas.

Gulf Coast Veterinary Specialists Facebook: @gulfcoastveterinaryspecialists; Instagram: @gcvs; Twitter: @gcvspecialists

Bill Schroeder
Schroeder is vice president of InTouch Practice Communications, a full-service marketing agency dedicated to veterinarians.
Facebook: @InTouchVet; Twitter: @InTouchVet

Stephanie Serraino
Serraino is a marketing manager for Compassion-First Pet Hospitals and manages the social media accounts for multiple veterinary hospitals, including the two CARE Center locations in Cincinnati and Dayton in Ohio.

Facebook: @CAREcentervets;
Twitter: @CAREcentervets

Ernie Ward, DVM
Dr. Ward is a well-known veterinarian, speaker, author and founder of the Association for Pet Obesity Prevention. He also hosts the Veterinary Viewfinder podcast.

Facebook: @DrErnieWard; Instagram: @drernieward; Twitter: @DrErnieWard

Which social media platform should veterinary practices be using?

Ernie Ward, DVM: The four main social media platforms today are Facebook, Instagram, YouTube and Twitter-almost in that order as far as reaching pet owners at large. That's pretty much where you want to be.

Bill Schroeder: The most important platform is still Facebook because of the channel's reach and a veterinary practice's ability to target their audience. Close behind are Instagram and YouTube. (Editor's note: Bill shared with us big algorithm changes back in May. Learn about them at dvm360.com/FBchanges.)

How has social media had a positive impact on the veterinary hospitals you work for?

Karin Kandur: Social media lets us showcase the care we provide at Red Bank, highlight some of the patients who've benefited from our medicine, show behind-the-scenes aspects of our work, and educate pet owners so they can take better care of their pets. It also allows us to highlight the team and let others get to know them as both veterinary professionals and members of the community.

Kate Lawrence: Social media is one of the most important ways we communicate today. Businesses that ignore or underuse social media do a disservice to their hospitals and clients. Social media allows us to keep clients informed about our hospital, such as providing our new location when we were flooded during Hurricane Harvey last year. It also lets us engage with clients through stories of actual patients, grow clients' knowledge by sharing important health information and fun facts about pets and our hospital staff, and illustrate our state-of-the-art technology.

But what do I write?

Stephanie Serraino: Social media allows us to connect with our community by sharing stories of pet loss, love, survival and advanced veterinary care. We also get to share safety tips, patient updates, staff events and achievements as well as how we give back to our community. In turn, our community gets to share with us how these animals make their lives better.

What type of content drives the most social engagement?

Schroeder: Authentic content that demonstrates the practice's brand. Tell the stories of your clients and let pet owners feel the social climate of the practice. Use pictures, ask questions and respond quickly with well-thought-out responses.

Kandur: Several types of content yield positive feedback for us: practice and community service news (such as blood drives and in-house fundraisers for causes such as breast cancer awareness), meet-the-technician features, patient profiles showcasing success stories and interesting cases, personal stories from staff members, and staff photos that show our dedication to caring for pets, such as when they came in during a blizzard.

Laurie Hess, DVM: Definitely pictures-we take a lot of pictures. We have a photo release form that we ask clients to sign when they come in, and most people are pretty receptive to doing that. We then keep a library of our interesting photos so we can use them as needed.

Serraino: We find that success stories about critically ill patients do the best. A few years ago, CARE Center posted a story about a dog named Carmen that suffered severe smoke inhalation during a house fire. Her story, images and updates were shared globally and throughout the news media, including on CNN.

Are blogs a necessary part of social media marketing?

Dr. Ward: You need to own your content, and there's no better content to own than blog posts. [From them,] you can link to Facebook memes, Instagram photos and YouTube videos.

Schroeder: Absolutely. Not only are blogs a great way to share information, but they have tremendous search engine optimization value, because the keywords and phrases used demonstrate a practice's offerings and make the content very attractive to Google and other search engines.

Do you schedule content in advance?

Dr. Ward: The first thing to do is look out a month ahead to see whether there is a day or a celebration, such as National Veterinary Technician Week. Then decide what content, if any, you want to build around that event, whether it's a video or a blog or just a couple of memes. If we can plan in advance, we'll use Hootsuite, TweetDeck or another social media scheduler. I think every veterinary clinic should take advantage of that.

Dr. Hess: Seasons and holidays drive a lot of what we post. Around holidays and school vacations, for example, there's an increase in boarding, so we schedule posts about boarding during those times. Also, certain holidays drive particular kinds of posts. For example, people buy rabbits around Easter, so we have posts leading up to Easter about how you really need to think twice about before buying a rabbit.

How do you react to negative feedback online?

Dr. Ward: Being able to deal with negativity on social media is a must. The internet runs on hate, and you need to develop a mechanism to tune that out. If you're going to put yourself out there, be aware that you're going to encounter negativity-that I can guarantee. The best advice I can give somebody is that if you give in to the haters, they've won.

Kandur: I think the biggest downside to social media, including review sites such as Google and Yelp, is negative reviews. We probably make it more difficult for ourselves because we honour the confidentiality of the client-patient-veterinarian relationship and choose not to respond with case details for all to read. People don't understand the far-reaching effects of their words. Taking the professional road can be agonizing. It's frustrating to think how much our reputation can be affected when people hear only one side of the story.

Complaining ... or bullying?

Sometimes internet reviews turn into campaigns of abuse by not just an upset client but all the client's family, friends and social media contacts. We went deep into this topic in a recent dvm360 Leadership Challenge at dvm360.com/cyberbully.

Dr. Hess: Striking out or being negative in your response never helps. I always try to contact people who have written a negative post offline to discuss their concern so we can try to come to a satisfying conclusion. If they're happy, I ask them to remove the negative post. But there have been times when I have failed at that, and sometimes there's nothing you can do. If I feel like I've responded correctly and politely, but the person persists in wanting to leave the post or won't respond to me at all, then I create a public reply so that other people understand I've made my best efforts to work things out.

What's your No. 1 piece of social media advice?

Dr. Hess: It's important that posts have some regularity. We've created themes for certain days of the week where people expect certain things-Monday is Mammal Monday and Tuesday is Tank Tuesday [highlighting pets that call aquariums and terrariums home]. Related to these themes, we might offer a discount or a special gift because someone brought their ferret in on a Friday because they saw a Ferret Friday post. Once every week or two, we also post a "Did you know?" video where we mention a cool fact related to an exotic animal. Team members take turns creating these posts so people begin to recognize our staff while learning something interesting.

Schroeder: Bottom line: Create good, honest content that helps your audience, and interact naturally.

Diagnosis and Management of GI Motility Disorders

Albert E. Jergens, DVM, PhD, DACVIM

Gastrointestinal motility disorders are a diagnostic and therapeutic challenge for many practitioners. Here's how to narrow down the affected area so you can identify the cause and provide appropriate treatment.

Gastrointestinal (GI) motility disturbances are widely under recognised in dogs and cats, primarily because we do not have good tools to diagnose them. Delayed transit disorders are commonly suspected by clinicians and may involve the oesophagus (megaoesophagus and dysmotility), stomach (delayed gastric emptying), small intestine (postoperative ileus and pseudo-obstruction), or colon (constipation and megacolon). This two-part article discusses general causes of GI motility disorders affecting the oesophagus to the colon, and offers insight for appropriate diagnostic evaluation and treatment options for these disturbances. This article covers the GI tract as a whole; a second article will focus on oesophageal diseases that cause motility disturbances.

Gastric diseases and hypomotility

Vomiting is the predominant clinical sign in dogs and cats with gastric motility disorders. Gastric mucousal disorders may cause motility disturbances due to hypomotility or pyloric outflow obstruction. The most common causes of outflow obstruction are foreign bodies and/or intraluminal masses (e.g., mucosal and/or muscular proliferative diseases), whereas infiltrative diseases may cause generalised hypomotility. Gastric dilatation and volvulus may also cause outflow obstruction but is not further discussed here. Gastric hypomotility and defective propulsion may also be associated with diverse inflammatory conditions and metabolic/systemic diseases, such as chronic gastritis, inflammatory bowel disease (IBD), Helicobacter spp infection, ulceration/erosions, pancreatitis, hypokalaemia, hypocalcaemia and hypoadrenocorticism.

Gastric retention (i.e., retained gastric contents eight to 10 hours after meal ingestion) has two causes: gastric hypomotility and pyloric outflow obstruction. With pyloric outflow obstruction due to foreign bodies (an especially common cause in young animals; Fig. 1), an acute onset of vomiting typically occurs. These patients may be painful in the cranial abdomen on physical examination. Therefore, clinicians should take the time to rule in or out a foreign body with a diagnostic workup that includes abdominal imaging (survey radiographs for radiodense objects), with barium contrast if necessary to demonstrate a filling defect if the object cannot be visualised on survey radiographs (Fig. 2).

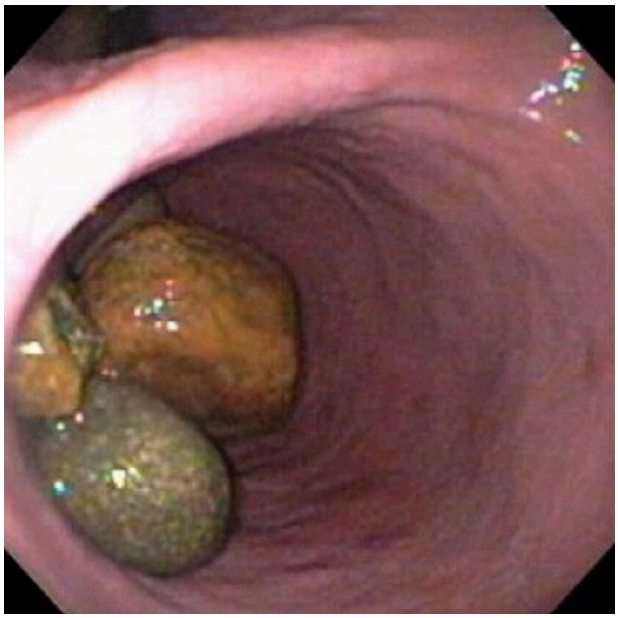

Fig. 1. Multiple rock foreign bodies causing pyloric outflow obstruction in a 1-year-old Siberian husky. The rocks were removed endoscopically.

Contrast radiography can be beneficial if ultrasound is unavailable or unrewarding. Gastroscopy serves to confirm the cause of obstruction, and can help with removing the obstructing object and/or sampling mass lesions via exfoliative cytology or forceps biopsy (Fig. 3). With infiltrative disease (e.g., nonspecific chronic gastritis, IBD), intermittent episodes of vomiting food and bile, haematemesis, melaena, abdominal discomfort and weight loss may occur. In patients with chronic gastritis, retained gastric fluids and ingesta noted on gastroscopy may be a clue that a concurrent motility disturbance is present (Fig. 4).

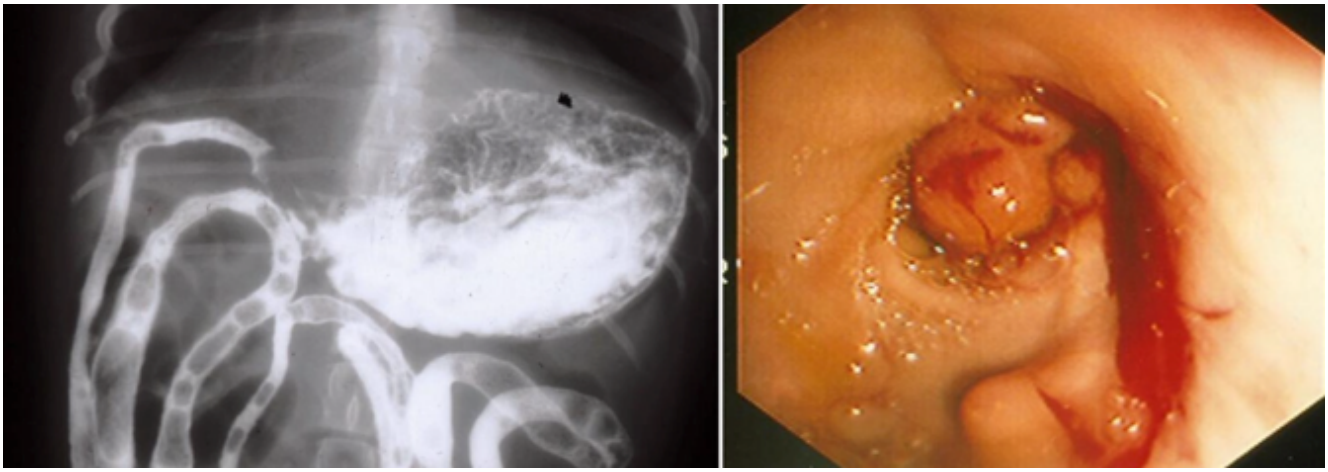

Fig. 2 - Gastric outflow obstruction caused by a pyloric mass in a 12-year-old poodle. Left: Ventro-dorsal contrast radiograph showing the filling defect at the pylorus. Right: Endoscopic appearance of a large antral polyp occluding the pylorus. A smaller polyp can be seen at the 6 o'clock position relative to the large polyp. Exfoliative cytology of the antral mass was consistent with benign adenomatous hyperplasia, and the mass was removed surgically.

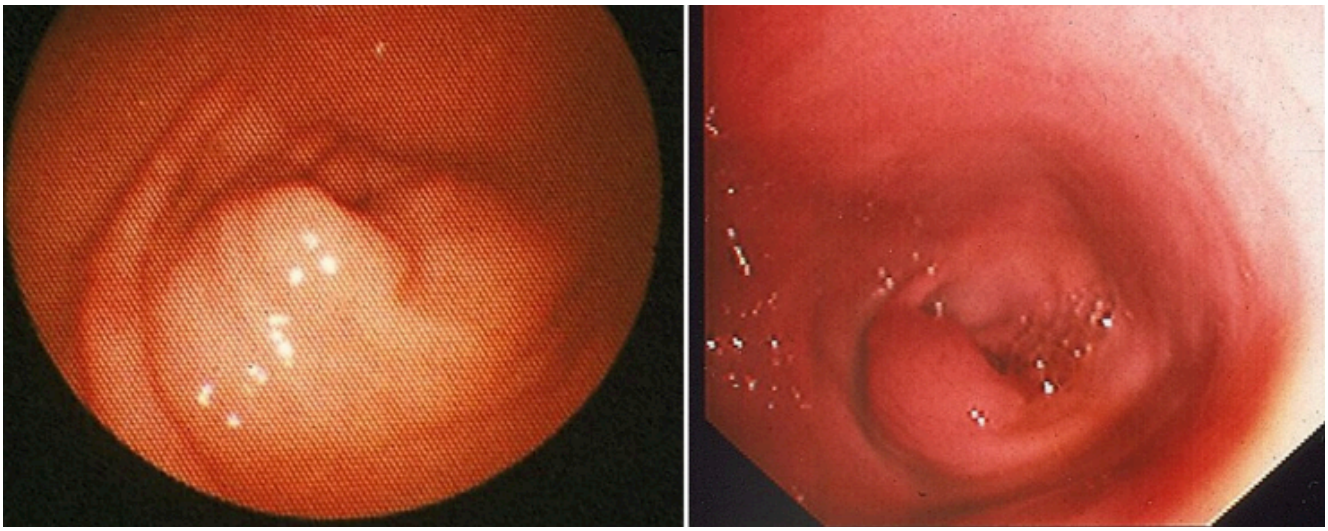

Fig. 3 - Pyloric masses having a similar appearance in separate dogs. Left: Redundant mucosal mass at the pylorus in a 1-year-old Bichon frise with chronic postprandial vomiting. Right: Pyloric mass in a 12-year old terrier X diagnosed with gastric retention. Endoscopic biopsy revealed mucosal hypertrophy in the Bichon but gastric adenocarcinoma in the terrier. Pyloric masses always warrant endoscopic/surgical biopsy for a definitive diagnosis.

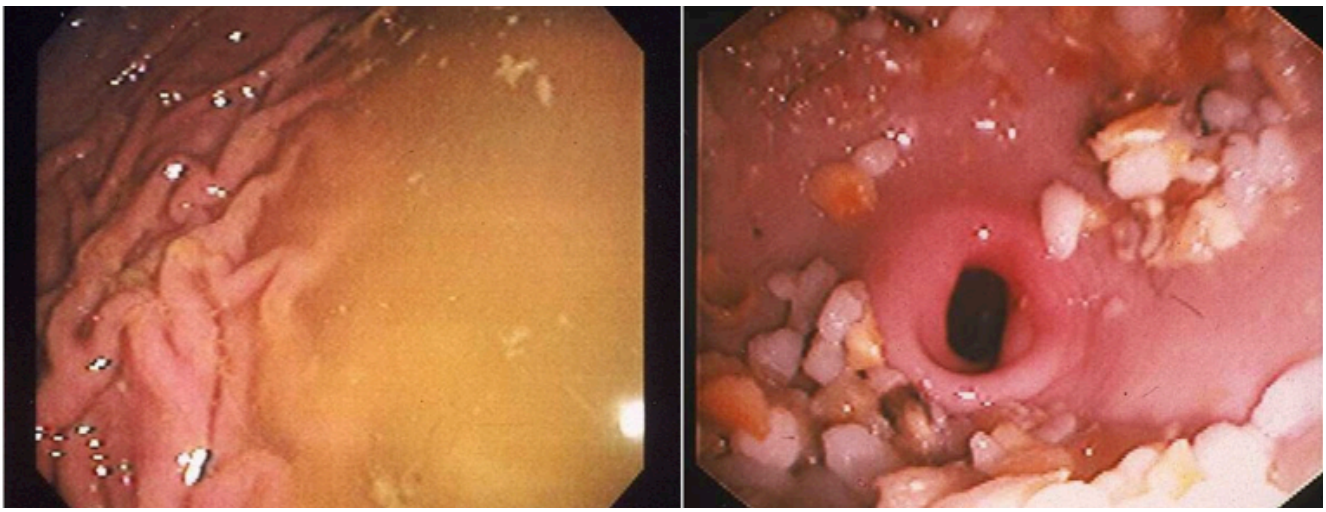

Fig. 4 - Endoscopic clues to gastric retention include the presence of large volumes of gastric fluid and/or ingesta (left) or retained ingesta in patients whose food has been withheld for over 14 hours (right)

Keep in mind that gastric motility disturbances other than mechanical obstruction (e.g., delayed gastric emptying) have diverse causes, including mucosal inflammation (e.g., chronic gastritis, IBD), infection (gastric Helicobacter spp), and dysmotility of unknown cause. Delayed gastric emptying has also been associated with with numerous secondary conditions, including hypokalaemia, metabolic disorders (e.g., Addison's disease, uraemia, diabetes mellitus), use of concurrent drugs (e.g., anticholinergics), and acute abdominal inflammation.

Treatment of gastric motility disorders is as follows:

1. **Mechanical obstruction:** gastric polyp (polypectomy), tumour (biopsy/gastrectomy +/- chemotherapy), foreign body (endoscopic/ surgical removal)
2. **Gastric emptying disorders:** inflammation/IBD (hydrolyzed diet +/- prednisolone), infection (antibiotics), idiopathic (hydrolyzed/ultra-low-fat diet + prokinetic drugs [e.g., cisapride, ranitidine, erythromycin])

Small and large intestinal motility disturbances

As noted earlier, intestinal motility disturbances can be difficult to document due to inadequate diagnostic tools that yield variable results. For example, methods to measure intestinal transit may include barium contrast studies (with or without food), ultrasonography (very operator dependent), and wireless motility capsule. If a structural disorder (e.g., mechanical obstruction) is suspected, it is advisable to start with survey abdominal radiography followed by contrast radiography to rule out specific intestinal diseases.

Ultimately, identifying and treating the underlying disease process is key to clinical remission. Enteroscopy may be useful for removal of proximal duodenal foreign bodies (Fig. 5). Clinical signs suggestive of small intestinal diarrhoea include large stool volume, watery diarrhoea, foetid smell, presence of melaena and systemic signs (hyporexia, ravenous appetite and/or weight loss); whereas normal stool volume, haematochezia, mucoid stools and/or frequent attempts to defaecate are most characteristic of large intestinal diarrhoea.

Intestinal obstruction

Obstruction of the small intestine is a common reason for dysmotility, especially with luminal foreign bodies. While these objects can lodge anywhere along the small or large intestine, many are diagnosed as lodged jejunal foreign bodies. Linear foreign bodies cause obstruction because of their ability to gather/pleat the intestine to cause partial or complete obstruction, and potential perforation, if not resolved. Intussusception in younger dogs and cats (< 1 year of age) can also cause bowel obstruction (Fig. 6). Patients with complete intestinal obstruction often present with acute signs

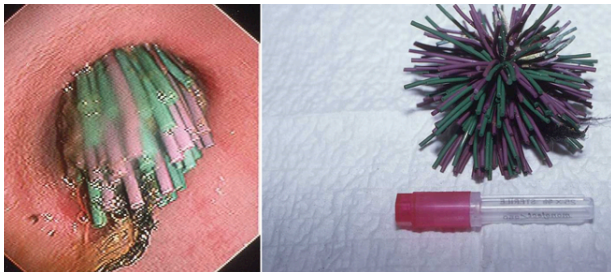

Fig. 5 - Duodenal foreign body in a 3-year-old cat with acute severe vomiting. Left: Endoscopic appearance of a duodenal foreign body occluding the proximal duodenal lumen. Right: The foreign body (play toy) extracted via duodenoscopy.

of vomiting, whereas chronic clinical signs (vomiting, diarrhoea hyporexia, and/or weight loss) are most consistent with chronic obstructive disorders. Other causes of intestinal dysmotility include dysautonomia, postoperative ileus, viral enteritis, opioid-induced dysmotility, and the rarely occurring idiopathic pseudo-obstruction (i.e., intestinal dilation with no evidence of mechanical obstruction).

Parvovirus

Canine parvovirus can cause hypomotility (functional ileus) of the small intestine as a consequence of severe intestinal inflammation/necrosis secondary to viral infection. Because parvovirus is viral in origin, the mainstay of therapy is supportive care. By supplementing nutrition, correcting hydration and providing other supportive measures (e.g., anti-emetic therapy), motility disturbances and clinical remission can be addressed adequately. With infiltrative disease, such as IBD, alimentary lymphoma, and GI histoplasmosis, obtaining mucuosal biopsies will generally yield a definitive diagnosis and the need for specific therapy.

Constipation and megacolon

Colonic motility disturbances are characterised predominantly by constipation and megacolon. Constipation denotes infrequent or difficult defaecation with the passage of dry faeces. The causes of constipation are numerous and include intra- or extraluminal mechanical obstruction (Fig. 7) (bones, hair, neoplasia, pelvic fracture), neuromuscular dysfunction (dysautonomia, lumbosacral disease), metabolic/endocrine disease (hypokalaemia, hypothyroidism), and medications (opioids, anticholinergics). Megacolon is characterised by excessive colonic dilation thought to be associated with functional disurbances in colonic smooth muscle.

Treatment strategies for colonic dysmotility are aimed at removal of the underlying cause; adding dietary fibre; using enemas, stools softeners, or manual extraction of hard faeces; and administering promotility drugs (cisapride, erythromycin, misoprostil). When mass lesions present in the colon are causing partial obstruction, the main differentials

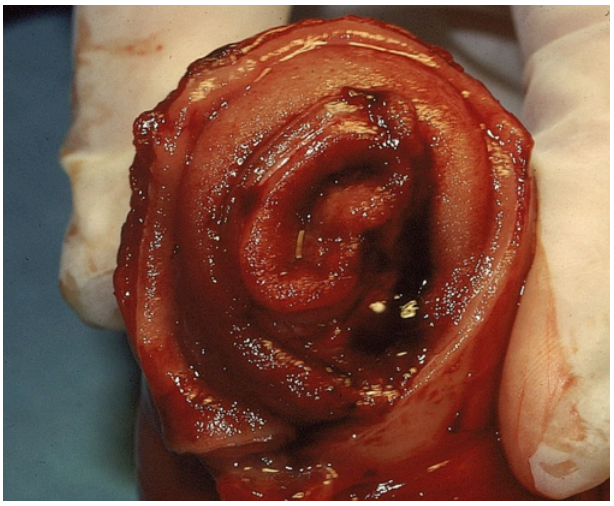

Fig. 6 - Surgical image of intussusception (unknown cause) in a 9-month old mixed breed dog with a history of intermittent diarrhea, melena and abdominal pain. Corrective surgery was curative.

are adenocarcinoma and inflammatory polyp, both of which usually can be removed (surgically or via endoscopy) without further complications.

Medical therapy for GI motility disturbances

The primary drug choices for GI motility disorders include agents that stimulate intestinal smooth muscle. These include metoclopramide, cisapride, erythromycin, ranitidine, and misoprostol, each of which has a different mechanism of action.

Gastro-oesophageal reflux

For gastro-oesophageal reflux and gastric hypomotility, metoclopramide (a dopamine antagonist) and cisapride (a serotonin 5-HT [hydroxytryptamine] agonist) are the primary therapeutic choices because they promote gastric emptying and increase lower oesophageal sphincter tone to prevent reflux. Metoclopramide will have limited effect distal to the pylorus but will likely facilitate gastric contractions and movement of fluid/ingesta through the pylorus. Because cisapride affects the smooth muscle of the entire GI tract, it may be beneficial in treating different causes of GI hypomotility involving the stomach and intestines.

Small intestinal dysmotility

For small intestinal dysmotility, cisapride, ranitidine and erythromycin are good treatment options. Ranitidine is a histamine 2 antagonist but has prokinetic activity, especially when treating small intestinal hypomotility. Erythromycin works via motilin-like activity and has prokinetic activity at sub-antimicrobial doses; it is also useful for stimulating smooth muscle of the small intestine. Occasionally, dogs with small intestinal IBD that are experiencing continued diarrhoea despite diet and immunosuppressive therapy will respond to a promotility drug, suggesting that infiltrative diseases

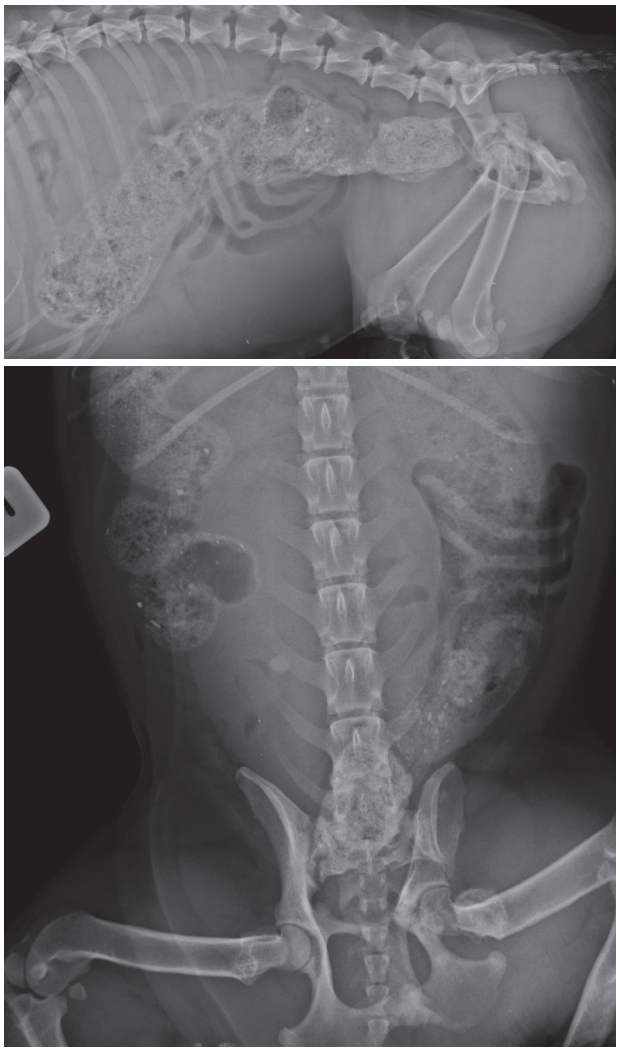

Fig. 7 - Lateral and VD radiographs showing a faecolith obstruction and subsequesnt constipation due to ingestion of bones and concrete dust compounded by a previous pelvic fracture which has narrowed the pelvic inlet. The faecolith was eventually crushed using a forceps after 2 weeks of lactulose and Kleen -Prep treatment both in practice and at home.

are associated with dysmotility. A suggested trial period of four to six weeks is recommended in these cases.

Colonic dysmotility / constipation

For colonic dysmotility/constipation, cisapride is recommended as a first choice agent. Unfortunately, there are few effective promotility medication options for stimulating colonic motility. Pilot study data suggest that misoprostil following meal ingestion may stimulate colonic motility in dogs.[1]

Dr. Jergens is an internist and holds the Donn E. and Beth M. Bacon Professorship in Small Animal Medicine and Surgery, and serves as associate chair for research and graduate studies in the Department of Veterinary Clinical Sciences at Iowa State University College of Veterinary Medicine.

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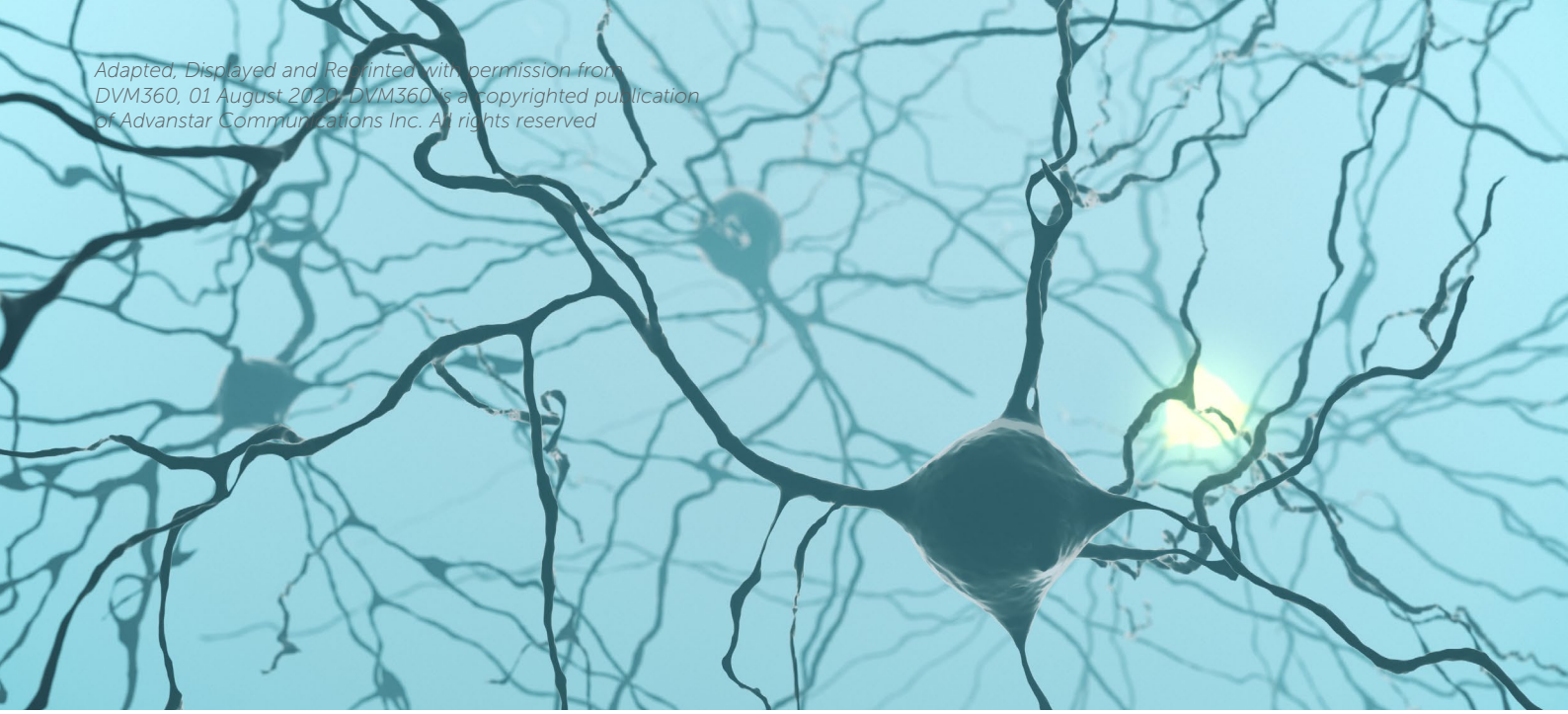

From Periphery to Perception: The Pathway to Pain

Sarah Mouton Dowdy

In explaining how a noxious stimulus in the periphery becomes an electrical impulse in the cerebral cortex, veterinary anaesthesiologist Dr. Mike Barletta ultimately makes a case for multimodal pain management.

In explaining how a noxious stimulus in the periphery becomes an electrical impulse in the cerebral cortex, veterinary anaesthesiologist Dr. Mike Barletta ultimately makes a case for multimodal pain management.

Physiologic pain, or acute adaptive pain—the kind that prompts you to pull your hand away when you touch a hot stove, can prevent injury and even promote healing after injury.

Pathologic pain refers to chronic, maladaptive pain, which Dr. Barletta described as "an expression of the pathologic operation of the nervous system that can follow acute pain if not prevented or treated."

Pain versus nociception

"Nociception is everything that happens before a noxious stimulus reaches the cerebral cortex," explained Dr. Barletta. "It includes the periphery, nerves, and spinal cord—everything up to the cortex. Once that stimulus is processed by the cortex and the organism becomes aware of it, we start calling it pain." Transduction, transmission, modulation, and projection are thus part of nociception.

Pain occurs at the point of perception.

For Dr. Barletta, treating pain starts with an understanding of its anatomy and physiology, beginning with the difference between nociception and pain followed by the five steps in the pain pathway: transduction, transmission, modulation, projection, and perception. With this foundation, the drugs to use for each step and why become more clear.

1 Transduction

The first phase in nociception, called transduction, occurs in the periphery and involves special receptors called nociceptors. "These nociceptors are the peripheral end-terminals of Aδ and C fibress," explained Dr. Barletta. "Mechanical, thermal, and chemical stimuli open different potassium and sodium channels located at the end-terminal of these nerve fibres to activate transduction, in which energy is converted from one form (which can cause tissue damage) to another (an electrical impulse)." If transduction isn't prevented or treated, he continued, tissue damage will cause the release of inflammatory mediators (histamine, bradykinin, tumour

necrosis factor-alpha, interleukin-beta, interleukin-6, nerve growth factor, adenosine, hydrogen ions, adenosine triphosphate, and prostaglandin E2) that can bind to peripheral receptors and recruit more inflammatory cells, resulting in peripheral sensitisation. Dr. Barletta referred to it as "inflammatory soup."

These inflammatory mediators can activate nociceptors that are usually silent to cause hyperalgesia (an overreaction to a painful stimulus) and allodynia (a painful reaction to a nonpainful stimulus, like touch). They also decrease the threshold for some receptors. "TRPV1 receptors are a really big deal in pain and usually have a temperature threshold of 45°C (113°F), which means you're going to start feeling that a stimulus is really hot and painful when it's at 42°C," he explained. "Now that threshold decreases to 35°C (95°F) in hyperalgesia. If you've ever had a bad sunburn, for example, bathing in lukewarm water can feel super hot—like it's burning your skin."

Transduction can be prevented with local anaesthetics, opioids, non-steroidal anti-inflammatory drugs (NSAIDs), and corticosteroids. "Local anaesthetics block sodium channels, which is one of the reasons why we use local blocks when performing surgery," Dr. Barletta noted. "As for opioids, most research findings concur that we have mu receptors in the periphery, and NSAIDs and corticosteroids decrease the inflammatory response."

Dr. Barletta said he often gets asked whether animals should always be pre-treated before a stimulus is applied. His response: "Ideally, yes. We definitely provide a local anaesthetic and administer opioids before that." Whether to give NSAIDs before surgery is a common conundrum, continued Dr. Barletta. "If I want to prevent pain, I should probably give as much as I can before the stimulus, but at that point, I don't know how the surgery and anaesthesia event are going to go down," he elaborated. "What if the patient is going to be hypotensive for most of the surgery and now maybe its kidneys aren't well perfused, and then I'm giving an NSAID on top of that? Those kidneys won't be very happy."

He explained that there are two options to choose from here: "Some say you should always wait to give NSAIDs until after surgery because you never know what's going to happen. However, I've seen someone spay a healthy cat in 2 minutes. The animal doesn't even have time to develop hypotension in 2 minutes, so in this situation it's probably OK to administer NSAIDs before surgery." However, he continued, if you're performing surgery on an animal that's pretty sick, such as a dog with haemoabdomen, it's highly likely you'll have to deal with hypotension. So, here's Dr. Barletta's rule of thumb on the matter:

"If the animal is young and healthy and it's a routine procedure, I don't have any problem administering an NSAID before surgery. If it's a critical patient, I'm probably going to wait until postoperative, as long as everything goes smoothly and blood pressures are fine."

Nerve fibres explained

(i) Aδ fibres
The myelinated Aδ fibres are thick (1-5 µm in diameter) and conduct impulses at a rapid rate (5 to 30 m/sec). "They have a small receptive field and are responsible for sharp, fast, transient, localised pain," Dr. Barletta explained. "They can be either unimodal, in which they transmit only one type of signal (either mechanical or heat), or polymodal, meaning they transmit more than one type of signal (both mechanical and heat)." Aδ fibres are predominant in the skin, which is why you can localise pain pretty well when, for example, you cut your own finger.

(ii) C fibres
The unmyelinated C fibres are thinner (0.25-1.5 µm in diameter) and conduct impulses more slowly (0.5 to 2 m/sec) than Aδ fibres. "Their receptive field is larger, and they're responsible for the slow, diffuse, burning, gnawing sensation of second pain, which persists after a noxious stimulus is terminated," Dr. Barletta said. "All C fibres are polymodal (mechanical and heat; mechanical and cold; and mechanical, cold, and heat)." C fibres are predominant in the viscera, which is why the pain from a stomach ache can be difficult to pinpoint.

(iii) Aβ fibres
"In normal circumstances," Dr. Barletta explained, "the myelinated Aβ fibres, which have a fast conduction velocity and a diameter ranging from 5 to 15 µm, are responsible for transmitting touch and pressure. But during chronic or pathologic pain, wide dynamic range neurons become activated and cause these fibres to start conveying signals as nociception instead of proprioception." This changeup is caused by peripheral sensitisation and central sensitisation (also known as windup).

2. Transmission

"After the nociceptors are stimulated in the periphery during transduction, the electrical signal travels to the dorsal horns of the spinal cord through the nerve fibres—Aδ, C, and (in chronic pain) Aβ fibres—via sodium, potassium, and calcium ion channels in a process called transmission," Dr. Barletta said. "Aδ fibres synapse in lamina I-V of the spinal cord and C fibres in lamina II, which is also referred to as the substantia gelatinosa." To block nociception during transmission, Dr. Barletta recommends local anaesthetics, which primarily block sodium channels.

"Interestingly," he added, "alpha-2s can potentiate the local anaesthetic effect, and some studies have shown

that when you add dexmedetomidine to your local anaesthetic it increases the duration of your local bock. Buprenorphine does the same thing." When asked if he has a preference between dexmedetomidine and buprenorphine, Dr. Barletta explained that he prefers the former for economic reasons: "Buprenorphine is going to be more expensive. Dexmedetomidine is expensive, too, but the volume you use is very minimal, so you're not actually spending that much money." He does, however, make an exception if the patient is sick enough that he doesn't think it will handle systemically administered dexmedetomidine.

3.Modulation

Modulation occurs in the periphery, spinal cord, and supraspinal structures. Focusing on spinal modulation, Dr. Barletta explained that the response to peripheral nociceptive stimuli can be modified in the spinal cord by the release of local endogenous modulators and the activation of descending pathways.

Opioids, serotonin, and norepinephrine, for example, can decrease the response to nociception, while glutamate, substance P, and prostaglandins can increase it. And when the response to nociception is enhanced, central sensitisation can occur. "This can be the result of brief but intense nociceptor activity, such as a surgeon cutting through skin, or of inflammation and nerve injury," he continued.

"Over time, repeated impulse activity of C fibres, release of a variety of neurotransmitters by the Aδ and C fibres, and activation of N-methyl-D-aspartate (NMDA) receptors can cause central sensitisation resulting in enhanced responses to impulses (e.g., hyperalgesia, allodynia)."

The presence of glutamate together with the depolarisation of the cell membranes (which displaces Mg++ from the channel itself) in the dorsal horns activates normally dormant NMDA receptors. "This should ring a bell here," Dr. Barletta paused to explain, "because there's a drug we use every day in anaesthesia that's an NMDA antagonist: ketamine. Does it help prevent chronic pain? Unfortunately, that's a hard study to set up. How can I really show that? Using it seems to make sense, but it unfortunately doesn't work that well when used to treat chronic pain. It's relief is very temporary."

Another unfortunate fact is that once these NMDA receptors are open and activated, it's really hard to shut them down. This is one of the many theories that try to explain phantom limb pain, Dr. Barletta added. "You can remove the affected limb, but in the spinal cord all of this is still active. Neurons keep going into the dorsal horn where they release neurotransmitters that activate these NMDA receptors. The signal goes to the brain, and the cortex still thinks it hurts. That's how nasty chronic pain is."

Ultimately, pain is a multimodal problem requiring a multimodal plan for prevention and treatment

Dr. Barletta noted that the downregulation of GABAergic and glycine receptor activity and the activation of microglia also occur during central sensitisation. "All of this mimics what happens in the periphery for peripheral sensitisation," he said. "The microglia become activated and do exactly the same thing as white blood cells in the periphery—they start releasing interleukins, tumour necrosis factor, etc.—and all of this will recruit more microglia and it will just keep going on and on and you end up with this windup, or central sensitisation."

NMDA antagonists, local anaesthetics, opioids, alpha-2 agonists, and NSAIDs all work in the dorsal horns. He also noted that, while not often used clinically, antidepressants and anticonvulsants are used for the purpose in research settings.

4. Projection and perception

Projectioninvolves the transport of the electrical signal from the spinal cord to the supraspinal structures.Three main large tracts are involved: the spinothalamic tract (from lamina I-V to the thalamus), the spinoreticular tract (to the reticular formation and thalamus), and the spinomesencephalic tract (to the midbrain).

Once the electrical impulse reaches the cerebral cortex, it's perceived as pain.

Some of the drugs Dr. Barletta points out for blocking pain perception may seem strange, such as isoflurane and sevoflurane. "They're not analgesic drugs," he explained. "They act at the cortex, minimizing or stopping the perception of pain, but they're not doing anything with nociception. That's why if you're anaesthetised just with inhalants, I can perform surgery without you feeling anything, but nociception is still happening. So as soon as I turn the gas off and you start waking up, you're going to be miserable." Other drugs that work in the cerebral cortex include opioids and alpha-2 agonists.

Make it multimodal

Ultimately, pain is a multimodal problem requiring a multimodal plan for prevention and treatment. Dr. Barletta put it this way: "Multimodal analgesia is the idea. We're trying to block, or even better prevent, these pathways at different locations and with different drugs to decrease the chance of creating chronic pain."

The ABCs of Veterinary Dentistry

'W' is for Waiting to Treat

When it comes to dental care, sometimes the best course of action is no action at all.

I can vividly remember back in 1974 when Dr. Wiggins, one of my professors at Auburn University College of Veterinary Medicine, advised us students to wait until a deciduous "double" tooth falls out by itself. Fast forward many years, and I now know that postponing the extraction of persistent primary teeth often causes harm to my patients.

Waiting to perform optimum dental care is sometimes the treatment of choice and at other times is a bad idea. This article covers some of those situations where waiting is the best option. A second article will outline conditions for which waiting is just wishful thinking.

Plaque and tartar without inflammation

Periodontal disease arises from gingivitis causedby plaque irritating the gingiva. When left to accumulate, plaque calcifies into rough calculus (tartar), which allows more plaque to collect on the crown. In predisposed dogs and cats, plaque inflames the gingiva (gingivitis) and eventually progresses to loss of support (periodontal disease).

Periodontal disease cannot occur without gingivitis, but gingivitis can occur without progressing to periodontal disease. Regardless, once calculus contacts the gingiva and inflammation is noted, it is time for an anaesthetized professional oral hygiene visit that includes dental scaling, irrigation, and polishing; tooth-by-tooth probing and full-mouth intraoral imaging; and treatment for any underlying disease discovered. Waiting will only increase patient discomfort and the extent of disease (Fig. 1).

If calculus is clinically present but inflammation is not, the professional oral hygiene visit can wait either until inflammation becomes apparent or a year has passed from the time when the last professional oral hygiene visit, to remove the accumulated plaque and calculus.

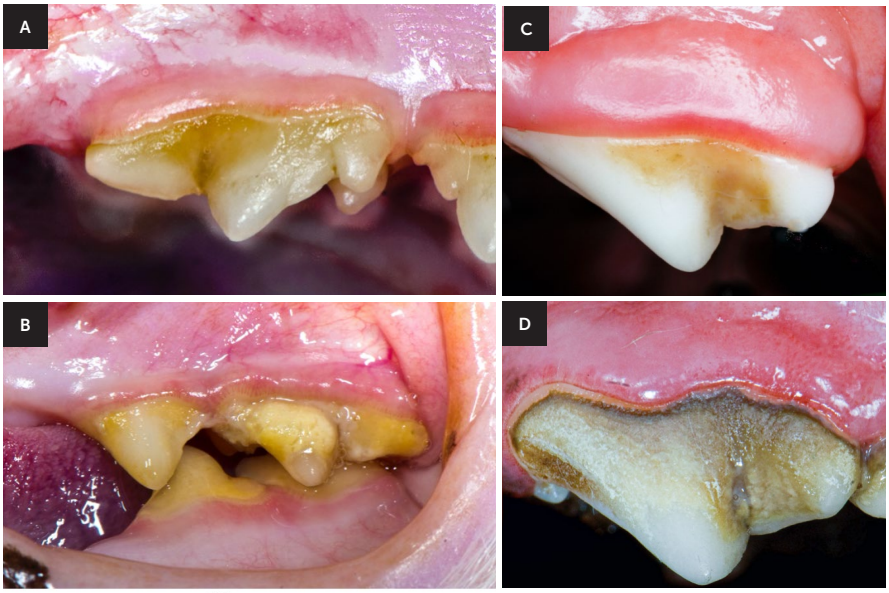

Fig. 1 - (A) Moderate accumulation of plaque and calculus without inflammation on a cat's right maxillary fourth premolar does not require immediate treatment. (B) Gingival swelling and inflammation in the presence of plaque and calculus on a cat's third and fourth premolars does. (C) Minimal calculus and inflammation on a dog's left maxillary fourth premolar can wait for scaling, while (D) moderate calculus and inflammation on a dog's left maxillary fourth premolar calls for a professional oral hygiene visit.

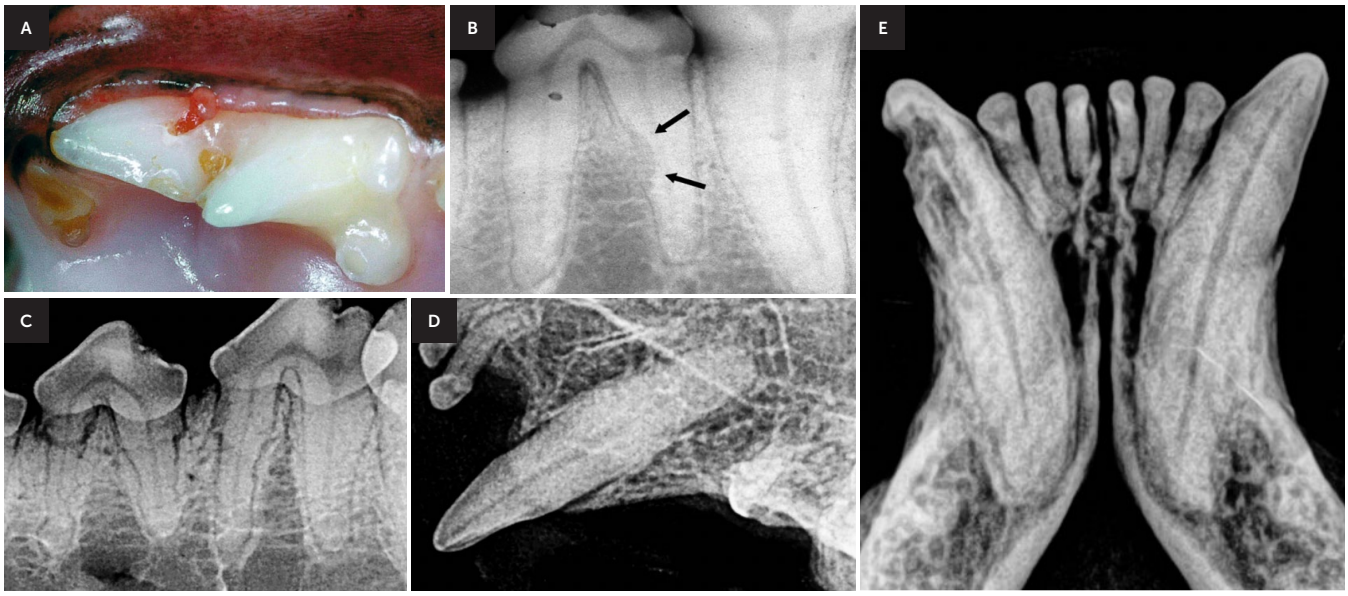

Fig. 2 - (A) Clinical appearance of external tooth resorption in a cat's right maxillary fourth premolar that has extended into the oral cavity. Waiting is not an option here; extraction is indicated. (B) Radiograph showing resorption (arrows) of a dog's right mandibular fourth premolar that is confined subgingivally. A wait-and-see approach can be taken with clinical rechecks and radiographs at 6- to 9-month intervals. (C) Radiograph showing resorption of a dog's left mandibular third and fourth premolars extending to the oral cavity. Extraction is indicated. (D) Radiograph of a cat's left maxillary canine displaying stage 2 tooth resorption that has not progressed to oral cavity exposure; extraction is not indicated at this time. (E) Radiographic evidence of resorption of a cat's right mandibular canine tooth extending to the oral cavity. Immediate extraction is indicated. (Note the left canine appears to be similarly affected but to lesser extent; extraction is still indicated).

Stage 2 external root resorption confined to the subgingiva

External root resorption can occur anywhere on a cat's or dog's tooth but starts in the periodontal ligament space. Many root resorptions are discovered via intraoral radiographs obtained during the anaesthetized professional oral hygiene visit. Even though external root resorption is considered progressive, immediate extraction is not necessary when the resorption is shown clinically and radiographically to be located far below the gingival margin in the apical third of the tooth. Once external root resorption is exposed to the oral cavity, bacterial contamination of dentin and pulp results in painful inflammation. In those cases, extraction is the treatment of choice (Fig. 2).

Internal root resorption is a different story in that by its nature the pulp is infected and considered painful. For internal root resorption, waiting is not an option. Root canal therapy or extraction are the treatments of choice.

Supernumerary and malpositioned teeth

Extra teeth in large-breed dogs often can be accommodated without causing harm or requiring extraction. Small-breed dogs and most cats do not have this luxury. Supernumerary teeth in small- and medium-sized breeds can cause tooth crowding, leading to gingivitis and periodontal disease resulting from the accumulation of food and oral debris between the teeth irritating the gingiva. Immediate extraction of the tooth (teeth) causing crowding is indicated in these cases (Fig. 3).

Not all teeth erupt in normal positions. Malpositioned teeth may be secondary to birthing trauma, inherited defects, or trauma after birth. The decision to treat or wait to move or extract abnormally positioned teeth should be made after an examination that includes probing and intraoral radiographs. If the malpositioned tooth is functional and not causing discomfort, waiting to extract is the best option (Fig. 4). Generally, whether to extract abnormally located teeth can present a conundrum. If there is no obvious mucousal penetration causing discomfort, a wait-and-see approach is appropriate.

Abrasion and attrition

Chronic abrasion (i.e., tooth wear caused by contact with a non-dental object, such as from self-grooming or chewing on tennis balls) and attrition (caused by contact of a tooth with another tooth from misaligned opposing teeth) may result in excessive wear and direct or indirect trauma to the pulp. Repeated low-grade trauma stimulates odontoblasts to produce tertiary (reparative) dentin for repair and protection. Tertiary dentin often appears as a reddish-brown shiny spot in the centrer of the worn surface.

As long as the rate of wear is gradual, reparative dentin production will keep up with loss of tooth structure without causing pulpal exposure. When the rate of wear is faster than the rate of tertiary dentin

Fig. 3 - Supernumerary maxillary incisor (A) and left maxillary first premolar (B) in a dog. Crowding is not seen clinically, so extraction is unnecessary. (C) Supernumerary third premolar in a cat causing obvious crowding; extraction is indicated. (D) Supernumerary maxillary third incisors and crowding caused by the maxillary right first incisor calls for surgical removal of the supernumerary and crowded teeth (E).

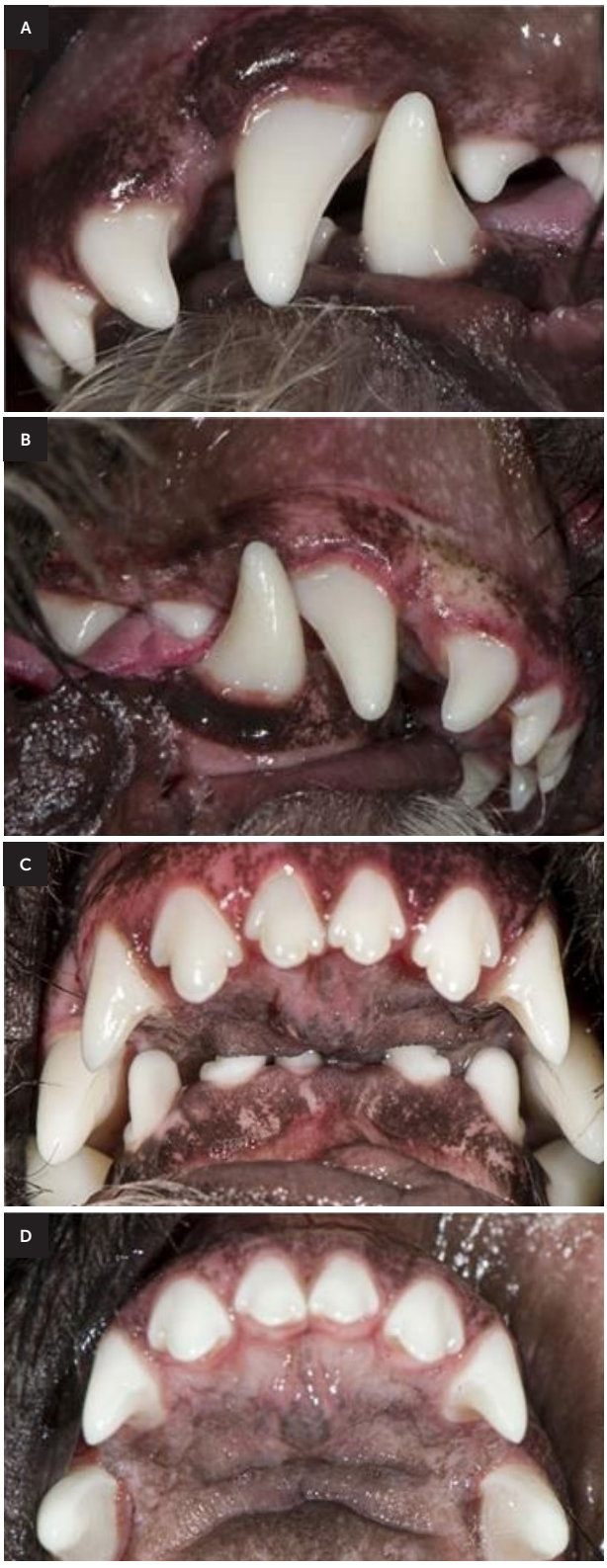

Fig. 4 - Left (A) and right (B) mandibular canines malpositioned caudal to the maxillary canines in functional positions; waiting is appropriate in these cases. (C) In this patient with a marked overbite (mandibular distoclusion), the mandibular incisors are impinging on but not penetrating the hard palate mucosa (D), so extraction is not necessary.

to bacterial invasion pulpitis and eventually pulp necrosis. Probing the worn area with an explorer and radiographic examination will help evaluate endodontic and periodontal involvement of worn teeth to see whether therapy is indicated. In cases of pulp exposure, treatment (root canal therapy or extraction) is indicated because pain and infection usually result (Fig. 5).

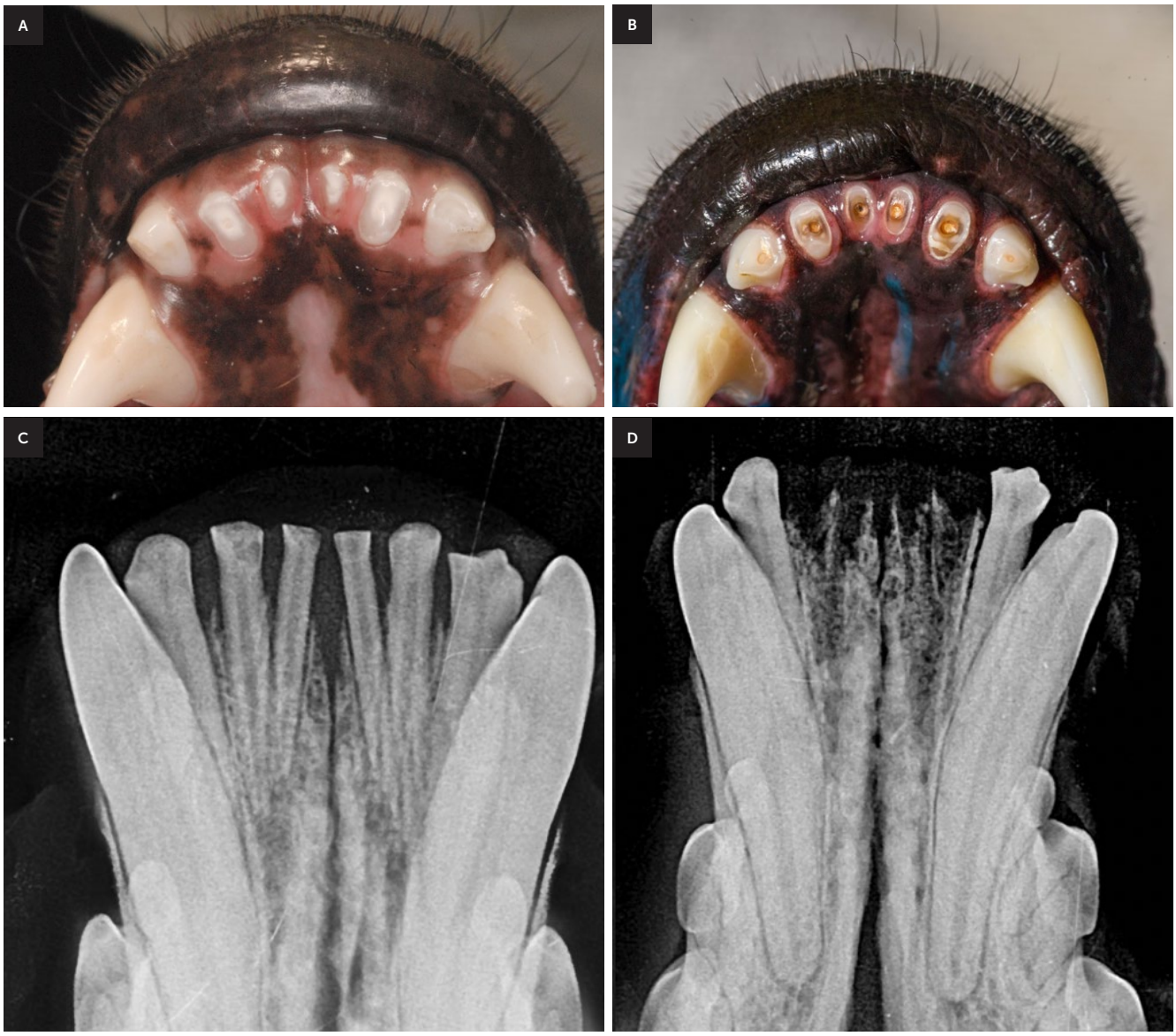

Fig. 5 - (A and B) Worn mandibular incisors without clinical or radiographic evidence of pulp exposure. (C) Radiograph demonstrating enlarged right and left first and second incisor pulp chambers consistent with pulpal exposure and necrosis. (D) Radiograph following extractions

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