Pain Management E-Book

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Pain is not always a bad thing, and all pain is not the same. Acute (protective) pain differs from chronic (maladaptive) pain in terms of function and treatment. This article describes the types of pain, the reasons why chronic pain can be difficult to treat, and the use of gabapentin and amantadine for treatment of chronic pain.

**ACUTE PAIN**

Acute pain in response to tissue damage is often called protective pain because it causes the patient to withdraw tissue that is being damaged to protect it from further injury (e.g., a dog withdrawing a paw after it steps on something sharp) or to become less active to protect tissue that is already damaged but healing (e.g., a cat sleeping frequently after abdominal surgery). A commonly used definition of acute pain reflects its normal role in tissue protection and healing: “an unpleasant sensory and emotional experience associated with actual or potential tissue damage.” Untreated or undertreated pain can cause myriad adverse effects, including but not limited to insomnia, anorexia, immunosuppression, cachexia, delayed wound healing, increased pain sensation, hypertension, and behavior changes that can lead to changes in the human–animal bond. Hence, we administer analgesic drugs to patients with acute pain, not to eliminate the protective portion but to control the pain beyond that needed for protection (i.e., the pain that negatively affects normal physiologic processes and healing). This latter type of pain decreases quality of life without providing any adaptive protective mechanisms and is thus called maladaptive pain. It serves no protective purpose but can cause the pain-mediated adverse effects previously mentioned.

**CHRONIC PAIN**

Chronic pain falls largely into the maladaptive pain category. This pain is often not protective because chronic pain is generally not caused by conditions that require rest for tissue healing, even if an acute injury that might have healed actually started the pain process. A common definition of chronic pain reflects
its lack of a role in tissue protection and healing: “pain that has persisted beyond the normal tissue healing time … pain without apparent biological value.”1 As with acute pain, chronic pain can cause the pain-mediated adverse effects previously mentioned and thus requires analgesic treatment.

Components of Chronic Pain
The most common cause of chronic maladaptive pain in mammals is osteoarthritis, or degenerative joint disease, and the second most common cause is cancer. The overall sensation of pain from any cause is multifactorial, and pain from either osteoarthritis or cancer generally has both inflammatory (which can be protective if not excessive) and neuropathic (which is always maladaptive) components.3

Inflammatory Pain
Inflammatory pain is a very common component of most pain syndromes, making the inclusion of anti-inflammatory drugs (e.g., nonsteroidal anti-inflammatory drugs [NSAIDs] or piroxepa [grapiprant]) an integral component of chronic pain therapy for all patients, unless use of these drugs is contraindicated (e.g., gastrointestinal ulceration, renal dysfunction, or hepatic dysfunction). However, anti-inflammatory drugs alone may not completely alleviate pain that is moderate to severe and/or longstanding. It isn’t that anti-inflammatories don’t work; rather, it is that they can’t control pain from other sources, like neuropathic pain. For adequate control of moderate to severe chronic pain, multimodal analgesia (i.e., use of more than 1 mode of treatment) is almost always necessary.

Neuropathic Pain
Neuropathic pain is commonly defined as “pain caused by a disease or lesion which leads to damage or dysfunction of the somatosensory (pain) system.”4 Neuropathic pain is maladaptive and can be fairly intense; pain impulses are often described by people as “lightning bolts” or “stabbing pain.” Components of neuropathic pain include peripheral sensitization (increased pain stimuli from peripheral tissues) and central sensitization (increased activity of the pain pathway at the dorsal horn neurons of the spinal cord, often referred to as windup). Neuropathic pain also often includes ectopic firing of nociceptors, which normally do not fire unless stimulated, and down-regulation of opioid receptors, making drugs in this class largely ineffective for treating this pain syndrome. Pain is naturally controlled to some extent by the descending inhibitory limb of the pain pathway; however, this limb is often dysfunctional in patients with neuropathic pain.

These pathologies in the pain pathway can lead to abnormal pain sensations such as hyperalgesia (exaggerated pain sensation from a mildly painful stimulus) and allodynia (pain sensation from a nonpainful stimulus). Examples of neuropathic pain commonly encountered in veterinary medicine include nerve entrapment, nerve damage after surgery or trauma, amputations, tumors associated with or impinging on nerves, lumbosacral disc disease/degeneration, discospondylitis, feline lower urinary tract disease, chronic changes associated with osteoarthritis (degenerative joint disease) (FIGURE 1), and many others.3,5,6 It is intuitive that neurologic injury or damage can lead to neuropathic pain but

FIGURE 1. Degenerative joint disease in the cubital joint of a cat (A) and the coxofemoral joints of a dog (B).
perhaps not as clear how diseases like osteoarthritis can lead to neuropathic pain; this lack of clarity has contributed to the undertreatment of this condition. Although osteoarthritis and other chronic pain conditions may not involve direct nerve damage, chronic pain (especially if untreated or undertreated) can lead to pain pathway changes that are inherent to neuropathic pain, including ectopic activity in afferent nerves, peripheral sensitization, central sensitization, impaired inhibitory modulation, and pathologic activation of cells that are normally not active in the pain process. Adding to the potential for undertreatment of chronic pain is the fact that these changes make chronic pain difficult to treat. The pain often no longer results from the inciting cause but rather results from these changes in the pain pathway.

Treatment of Chronic Pain
Unfortunately, the changes that lead to chronic pain are neither predictable nor consistent, making pain a truly individual sensation that often requires individual therapy. However, we have a variety of drugs that may effectively treat chronic pain in general and some that treat neuropathic pain in particular. This article focuses on 2 attainable and affordable options, gabapentin and amantadine, which are not only fairly specific for neuropathic pain, but are also nonopioid drugs. The opioid shortage has not threatened the supply of these drugs and diversion for human abuse is probably nonexistent. Neither drug is controlled by the Drug Enforcement Administration (DEA).

Gabapentin and amantadine each has a greater potential than tramadol for effectively treating chronic pain because of their mechanisms of action. Tramadol, a class IV DEA-controlled drug, is commonly used in veterinary medicine yet is unlikely to be effective when used alone for treatment of either acute or chronic pain; it was recently shown to be ineffective for treatment of osteoarthritis pain in dogs. This finding is not surprising because the opioid effects of tramadol in dogs are minimal. However, tramadol may provide some mild analgesia that would be useful in a multimodal protocol because of tramadol’s role as a serotonin and norepinephrine reuptake inhibitor, which may provide analgesia through modulation of the descending inhibitory limb of the pain pathway. Tramadol effectively treats osteoarthritis pain in cats, but those who have tried administering this drug to cats know that it can be difficult because its taste seems to be especially noxious to cats.

GABAPENTIN

What Is Gabapentin?
Gabapentin is a drug in the anticonvulsant class that is approved by the Food and Drug Administration (FDA) for treatment of seizures and certain neuropathic pain conditions in humans. It is commonly used off-label to treat seizures (not described in this article) and neuropathic pain in animals. The drug is structurally similar to the inhibitory neurotransmitter gamma-aminobutyric acid (GABA), which explains its name but not its mechanism of action. Although not completely defined, the primary mechanism of action is presynaptic inhibition of calcium channels (specifically the alpha-2-delta-1 subunit) and subsequent calcium influx, which leads to decreased release of excitatory neurotransmitters (FIGURE 2). This is the mechanism for both seizure control and pain relief.

Why Use Gabapentin?
Gabapentin is useful because chronic pain commonly has a neuropathic component that...
is not controlled by anti-inflammatory drugs, opioids, or other drugs/drug classes.

Which Patients Should Receive Gabapentin?
Gabapentin should be administered to patients with a known neuropathic lesion (e.g., extruded disc, nerve injury), suspected neuropathic lesion (e.g., painful back, neck), or chronic pain that is not controlled by anti-inflammatory drugs or for which these drugs are contraindicated.

What Is the Dosage for Gabapentin?
Gabapentin is often overlooked because it has a reputation for a prolonged onset of action and/or ineffectiveness. Both of these characteristics can be true, but both may also result from inadequate dosing or dosing regimens, and it is my experience that inadequate dosing is very common. Most of us learned to prescribe gabapentin at 5 mg/kg PO q12h; however, although this regimen may be effective for some patients, neither the dose nor administration frequency is adequate for many patients. Dose and administration frequency can vary among individuals, meaning that we may have to work a little bit to find each patient’s optimal dose (BOX 1). But finding the optimal dose is not all that hard to do; we just need to understand the pharmacokinetics of the drug—and enlist the pet client’s help.

Why is the dose so variable? To answer that, let’s first look at data from human medicine, for which the pharmacokinetics are better defined. After oral administration, gabapentin is absorbed from the gastrointestinal tract via an L-amino acid transport system, which is saturable, meaning that as the dose of gabapentin increases, the serum concentration does not increase linearly. In humans, as the dose increases from 900 to 3600 mg/day, the bioavailability of gabapentin drops from 60% to 33%. We know that this same pharmacokinetic phenomenon also occurs in dogs, and we presume that it occurs in cats, although this phenomenon has yet to be studied in cats. What we do know about cats is that the bioavailability of gabapentin is highly variable, at least in nonfasted cats. This finding means that dosages that provide serum concentrations of gabapentin adequate for analgesia may be higher and more variable among patients than we once thought. The dose range has long been reported as 3 to 20 mg/kg, but use of doses as high as 50 mg/kg has been anecdotally reported. Although your initial dose may not be 50 mg/kg, you may eventually reach that dose as you challenge the effective dose for any individual patient.

In addition to escalating doses, more frequent administration may be necessary. The pharmacokinetics for dogs and cats indicate that gabapentin administration every 6 to 8 hours, rather than every 12 hours as commonly used, may be needed to provide serum concentrations of gabapentin that are adequate for analgesia. Thus, for each patient, both the dose and the administration frequency may need to be explored, for which we need to engage the client/caregiver. Mild sedation, which can be easily recognized by clients, can be used to guide the need for dosage changes. Working with the client, choose a reasonable endpoint for determining effective pain relief (e.g., taking longer walks, climbing stairs) and discuss the signs of sedation. Then choose a starting dose and, by placing frequent phone calls to the client to check on progress, guide the client on titrating the dose of gabapentin until either endpoint—sedation or decreased pain—is reached. Clients should also be advised that ataxia might occur at higher dosages, especially in large breed dogs and/or dogs with decreased muscle mass. If ataxia occurs, the same guidelines as described for the occurrence of sedation (e.g., decreasing the dose) should be followed.

What Efficacy Data Are Available for Gabapentin?
No controlled research studies on the use of gabapentin for the treatment of chronic pain in dogs and cats have been performed. Unfortunately, lack of research is a common problem for most methods of chronic pain treatment in veterinary medicine. Several case reports note analgesia when gabapentin was used for treatment of chronic pain. And in a clinical study on
BOX 1 Recommended Gabapentin Treatment Guidelines

**START**

Start at 5 mg/kg PO q12h for mild pain and 10 mg/kg q12h or 5 mg/kg q8h for moderate to severe pain. The interval of q8h is preferred.

- **Does the patient have renal or hepatic disease?**
  - **YES**
    - The starting dose may be as low as 3 mg/kg q12h.
  - **NO**
    - Did pain relief occur in 3 to 5 days?
      - **YES**
        - Stay at that dose.
      - **NO**
        - Is the patient comfortable and not sedate?
          - **YES**
            - Return to the previous (nonsedating/non-ataxic) dose and maintain that dose for 7 days.
          - **NO**
            - Try increasing again. Gradually increasing the dose over time often decreases the occurrence of sedation or ataxia.

- **Did pain relief occur in 3 to 5 days?**
  - **YES**
    - Increase the dose by roughly 25% per dose.
  - **NO**
    - If the administration is q12h, use the same dose q8h.
      - **YES**
        - The starting dose may be as low as 3 mg/kg q12h.
      - **NO**
        - Increase the dose by roughly 25% per dose.

- **Is sedation [or ataxia] reached before pain relief?**
  - **NO**
    - Is the patient comfortable?
      - **YES**
        - Stay at that dose.
      - **NO**
        - Return to the previous (nonsedating/non-ataxic) dose and maintain that dose for 7 days.

- **Is the patient comfortable and not sedate?**
  - **YES**
    - Is the patient comfortable and not sedate?
      - **NO**
        - If the administration is q12h, use the same dose q8h.
          - **YES**
            - The starting dose may be as low as 3 mg/kg q12h.
          - **NO**
            - Increase the dose by roughly 25% per dose.

**END**

If gabapentin therapy is to be discontinued (e.g., the pain is completely resolved or the gabapentin is not working), to prevent potential rebound pain, withdraw the drug gradually over 1 to 3 weeks (depending on the duration of therapy). Have the client continue to monitor the patient. Drug effectiveness is sometimes easier to identify while the drug is being withdrawn.
postoperative pain in dogs undergoing mastectomy, although pain scores did not differ, dogs receiving NSAIDs plus gabapentin required fewer opioid rescue doses than dogs receiving NSAIDs alone; thus, the gabapentin did seem to have an effect.16 When looking at chronic conditions, among dogs with Chiari-like malformation and syringomyelia, pain scores did not differ among those receiving gabapentin and those receiving topiramate (an anticonvulsant and treatment for migraines in humans), but quality of life scores were better for the dogs receiving gabapentin.17 However, dogs receiving gabapentin immediately after surgery for herniated disc extrusion, which would be a mix of chronic pain from the disc and acute pain from the surgery, demonstrated no significant pain relief with gabapentin compared with placebo.18 The fact that the results between these studies are mixed is probably explained by 2 factors: 1) chronic pain has many facets, and pain from sources other than neuropathic were probably present; and 2) doses of gabapentin were low (5 to 10 mg/kg q12h) and perhaps ineffective, which actually makes the fact that any improvement was seen quite encouraging. More appropriate dosing, as defined by patient-specific dose and administration-interval adjustments, could potentially provide analgesia.

In a thermal research model, gabapentin seems to provide minimal benefit for control of acute pain caused by heat.19 However, because acute pain is not often neuropathic pain, the lack of evidence does not mean that gabapentin is not effective. Even in chronic pain studies, results for gabapentin may be mixed in terms of provision of analgesia because chronic pain is complicated and no one drug will work for all types of chronic pain in all patients. However, gabapentin is a common addition to analgesic protocols and, based on its mechanism of action, effectiveness can be anticipated if the dose and diagnosis (that the source of pain is actually neuropathic pain) are correct.

What Are the Adverse Effects of Gabapentin?
The primary side effect (not necessarily an adverse effect) of gabapentin therapy in dogs and cats is sedation. This side effect is generally dose-related and usually alleviated by decreasing the dose. If sedation occurs and the dose is reduced, sedation can often be avoided if the dose is slowly titrated back up to its original high/therapeutic level over several weeks. Sedation is more common in patients receiving other sedating drugs (e.g., tramadol). Dose-dependent ataxia and weakness can occur, especially in older patients with decreased muscle strength. In rats and humans, gabapentin is primarily cleared by the kidneys; in dogs, it is also metabolized by the liver.20 In cats, the route of clearance is unknown but is presumed to be primarily renal. Thus, gabapentin may undergo more rapid accumulation, and thus more rapid onset of adverse effects, in cats and dogs with renal disease and dogs with hepatic disease. For these patients, a lower starting dose and slower dose escalation is recommended.

How Is Gabapentin Supplied and What Does It Cost?
Gabapentin is available as a generic drug in tablets and capsule of various strengths. It is also available as a liquid, but some liquid formulations contain a low concentration of xylitol. The liquid can be compounded without xylitol. Gabapentin is not a DEA-controlled drug and is not expensive.

Are There Any Other Drugs in This Class (Anticonvulsants) That Are Used for Pain Relief?
Pregabalin is FDA-approved for the treatment of numerous neuropathic pain syndromes in humans. Dosing at 1 to 2 mg/kg PO q12h is supported by pharmacokinetic studies in dogs21 and cats.22 Pregabalin is DEA class V and fairly expensive.

NOTE: Gabapentin is also commonly used for behavior modification, especially in cats, before stressful events such as trips to a veterinary clinic.23 The dosage for this indication is 50 to 150 mg/cat PO at least 2 hours before the scheduled stressor will occur. In very anxious or fractious cats, the same dose is often administered the night before the stressor will occur.

AMANTADINE

What Is Amantadine?
Amantadine is a very interesting drug with multiple uses stemming from varied mechanisms of action. Amantadine is FDA-approved for the treatment of influenza virus A infection and Parkinson’s disease in humans. The mechanisms of action by which amantadine treats those 2 conditions differ, and the mechanism by which it contributes to analgesia differs yet again. In its role in pain management, amantadine antagonizes N-methyl-D-aspartate (NMDA) receptors
in the central nervous system (FIGURE 2), a mechanism akin to that of ketamine, which reverses or prevents central sensitization. A key component of central sensitization is the opening of NMDA receptors with subsequent transmission of excitatory electrical signals.

Why Use Amantadine?
Amantadine plays a role in pain control because central sensitization, often called central plasticity,3 can be a stand-alone component of moderate to severe and/or undertreated pain and is a major component in the development of neuropathic pain.

What Efficacy Data Are Available for Amantadine?
Less information is available about the use of amantadine than use of gabapentin for the treatment of chronic pain in veterinary patients, but 1 controlled research study of dogs with chronic refractory hindlimb osteoarthritis has been reported.26 In that study, dogs receiving NSAIDs plus amantadine (3 to 5 mg/kg PO q24h for 21 days) were more active and scored lower on a lameness scale than dogs receiving NSAIDs alone. Researchers of a study of heat-induced pain in cats receiving oxymorphone concluded that “amantadine might decrease the antinociceptive (analgesic) dose of oxymorphone in some but not all cats.”27 These results are not surprising because the mechanism of action of amantadine is more likely to be effective in animals with central sensitization, which was not caused in this research model. As stated in the discussion of gabapentin, there is a scarcity of studies on treating chronic pain with any drug or modality in veterinary patients.

What Are the Adverse Effects of Amantadine?
No adverse effects or drug interactions in dogs or cats receiving amantadine have been reported.7

How Is Amantadine Supplied and What Does It Cost?
Amantadine is moderately expensive. Amantadine is supplied as tablets and capsules of various strengths and can be compounded as a liquid without xylitol.

Are There Any Other Drugs in This Class (NMDA-Receptor Antagonists) That Are Used for Pain Relief?
Ketamine, which is used as an infusion to provide analgesia, is used for acute pain relief, and reports from human medicine show that it can be used to treat some forms of chronic pain.28 Methadone, a potent opioid, also has some NMDA-receptor antagonistic effects. Other oral drugs in this class include memantine, dextromethorphan, and a few others,29 none of which have been studied for relief of neuropathic pain in veterinary patients.

Your first choice for known nerve damage should be gabapentin, and your first choice for pain that is suddenly worse than expected with no signs of worsening disease should be amantadine.

Which Patients Should Receive Amantadine?
Amantadine should be considered as part of the initial multimodal therapy for any patient that has moderate to severe chronic pain and as an add-on drug for patients that have worsening chronic pain despite presumably adequate pain control and no worsening of the inciting disease.

What Is the Dosage for Amantadine?
Because amantadine’s contribution to pain relief is not really analgesia (it is technically called antihyperalgesia), the drug must be used as part of a multimodal protocol with true analgesic drugs like NSAIDs, opioids, and gabapentin. The dosage for dogs and cats is 3 to 5 mg/kg PO q24h to q12h, with q12h preferred. Data from recent pharmacokinetic studies indicate that twice daily dosing is probably more effective than the traditional once daily dosing in dogs33 and cats.25 To decrease the central sensitization component of chronic pain, treatment duration probably needs to be long; thus, the current minimum duration recommended is 21 days.26 Longer durations may be necessary, and many patients may need amantadine for life.
CONCLUSION

Although research evidence is currently lacking, the scientific mechanisms of gabapentin and amantadine support their use as part of analgesic protocols for chronic pain relief in dogs and cats. Each can effectively treat chronic pain when the dose, administration frequency, and duration of treatment are correct. When to choose one drug over the other? That choice is somewhat personal preference but also based on disease progression. Either or both can be part of initial therapy for chronic osteoarthritis pain. I often use both (usually with no dosage adjustments) if pain is severe. Your first choice for known nerve damage should be gabapentin, and your first choice for pain that is suddenly worse than expected with no signs of worsening disease should be amantadine. But you really cannot go wrong; grab one of them—or both of them—and try! Gabapentin and amantadine are best used as part of a multimodal protocol, especially when pain is moderate to severe. TVP

References

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Gabapentin and Amantadine for Chronic Pain: Is Your Dose Right?

LEARNING OBJECTIVES
Readers will be able to compare protective pain and maladaptive pain, discuss the reasons that chronic pain can be difficult to treat and the need for multimodal analgesia, describe the role of gabapentin and amantadine in the treatment of chronic pain, and prepare treatment protocols.

TOpic Overview
This article describes the complexity of chronic pain, the need for multimodal therapy, and the use gabapentin and amantadine at correct dosages and administration frequencies in treatment protocols.

1. What is another name for acute pain in healing tissue?
   a. Maladaptive
   b. Neuropathic
   c. Protective
   d. Osteoarthritic

2. Define maladaptive pain.
   a. Pain lasting more than 30 days
   b. Pain without biological value (not protective)
   c. Joint pain that develops from mild osteoarthritis
   d. Pain that prevents further damage to injured tissue

3. How does chronic pain with no physical neurologic damage become neuropathic pain?
   a. Damage occurred at some point in the pain syndrome, which led to neuropathic pain.
   b. Sensory pathway changes in response to chronic moderate/severe stimulus are essentially pathology of the nervous system.
   c. Neuropathic and chronic pain are equal.
   d. Cancer pain can become neuropathic if the tumor impinges on nerves.

4. Why is it important to adequately treat pain?
   a. Pain causes adverse effects that can negatively affect the patient’s overall health.
   b. Untreated pain can get worse as changes occur in the pain pathway.
   c. Pain can decrease the patient’s quality of life.
   d. All of the above.

5. Assuming no contraindication, which one should generally be the first choice in treatment and why?
   a. Gabapentin; chronic pain is always neuropathic pain, even if it is only mild.
   b. Amantadine; the NMDA receptors are always activated in chronic pain, making chronic pain very difficult to control.
   c. Opioids; only opioids control excruciating pain.
   d. Anti-inflammatory drugs; inflammation pain is a common source of chronic pain.

6. In which of these patients would you have the client increase the dose of gabapentin right now?
   a. Experiencing mild sedation at the current dose.
   b. Renal disease patient still showing some signs of pain despite a dose increase in the past 24 hours.
   c. Experiencing no sedation and still exhibiting pain despite a dose increase 3 days previously.
   d. Exhibiting no signs of pain despite being on a low dose of gabapentin administered q12h.

7. What role does amantadine play in pain relief?
   a. Antihyperalgesia
   b. Anti-inflammation
   c. Calcium channel blockade
   d. NMDA-receptor agonist

8. Current pharmacokinetic data for gabapentin and amantadine support a dosing interval of:
   a. At least q8h for gabapentin and q12h for amantadine
   b. Q12h for gabapentin and q24h for amantadine
   c. Q24h for gabapentin and q12h for amantadine
   d. At least q8h for gabapentin and once every 21 days for amantadine

9. Amantadine and gabapentin can be administered concurrently.
   a. True
   b. False

10. Which patient would be least likely to benefit from gabapentin or amantadine? A patient:
   a. With chronic untreated otitis that snaps if touched, even gently.
   b. With worsened elbow arthritis despite no evidence of worsening.
   c. Hit by a car and in extreme pain.
   d. After a forelimb amputation secondary to a painful nonunion of a radius-ulna fracture.
Grapiprant for Control of Osteoarthritis Pain in Dogs

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For centuries, nonsteroidal anti-inflammatory drugs (NSAIDs) have been used for pain control. However, their use has been associated with potentially life-threatening adverse drug events. Grapiprant is a novel drug for treatment of osteoarthritic pain in dogs. Approved in 2016, it is a non-cyclooxygenase (COX)-inhibiting NSAID. Grapiprant is a highly selective, potent inhibitor of the prostaglandin (PG) E₂ EP₄ receptor, which is primarily responsible for the pain and inflammation associated with osteoarthritis. Grapiprant works downstream of traditional NSAIDs in the arachidonic acid cascade. A review of the arachidonic acid cascade and its role in inflammation can be found in a recent review.

In the United States, grapiprant has been approved for treatment of osteoarthritis in dogs. However, because it has only been 3 years since drug approval, nonbiased controlled clinical trials have not yet compared its safety or efficacy with that of other NSAIDs. Although grapiprant has not been on the market long enough for clinical trials and adverse events to be effectively assessed, this report reviews the existing literature about its safety or efficacy, especially information about its use in dogs with osteoarthritis. Grapiprant is not approved for use in cats.

**PHARMACODYNAMICS**
Grapiprant belongs to the piprant class of drugs, which are prostaglandin receptor antagonists; grapiprant selectively targets EP₄ receptors and physiologic responses to prostaglandin mediated by EP₄, grapiprant has the potential to influence multiple actions of PGE₂. For example, PGE₂/EP₄ homeostatic interactions include gastrointestinal, hemostatic, renal, and immune regulation. Other functions include wound healing, bone metabolism, and skin immunity. Although its primary ligand is PGE₂, EP₄ also binds to PGF₂α and PGE₁, but it is not clear whether physiologic responses to these ligands are blocked by grapiprant.

Because grapiprant’s antagonism of the EP₄ receptor seems to be selective, its effects at other sites are minimized. The magnitude of effect increases with plasma concentration. Binding is competitive, suggesting that efficacy is time-dependent, further suggesting that the dosing interval be based on elimination. According to in vitro studies of receptors in dogs and rodent and human pain models, the minimum plasma drug concentration for efficacious analgesia in dogs is 114 to 164 ng/mL. The dose needed to achieve this concentration, based on pharmacokinetic studies in dogs, is 2 mg/kg. However,
according to Freedom of Information laws in Europe, the dose justification has not been fully supported and more than one dose a day may be necessary.9

PHARMACOKINETICS
Several studies have described the disposition of grapiprant in dogs.8,10,11 Key pharmacokinetic parameters covered here are oral bioavailability, tissue distribution, and elimination.

Oral Bioavailability
Oral disposition presents some factors that influence bioavailability. In female Labrador retrievers, grapiprant given as a pure powder in a capsule was approximately 100% orally bioavailable when given to fasted animals, but only 60% bioavailable when given to fed animals.10 Furthermore, the time to peak plasma concentration was prolonged from 1 (fasted) to 3 hours, and the peak plasma concentration was reduced by approximately 40% when animals were fed. Study findings that plasma concentrations remained above the presumed minimum effective concentration (164 ng/mL) for approximately 6 hours regardless of feeding suggest that the drug can be given with food;10 however, efficacy might be affected and clients should be consistent as to how they administer the drug. Oral bioavailability is also affected by formulation, being 40% to 60% higher when given as the approved tablet rather than as a suspension.11 As such, manipulation of the approved formulation, including use of compounded grapiprant, should be avoided. If the tablet is scored (e.g., 20 and 60 mg), the dose can be calculated in half-tablet increments. If not scored (e.g., 100 mg), equal distribution of drug cannot be assumed and equal dosing may not occur with each drug half. No evidence could be found to address efficacy of crushed tablets.

In fasted beagles, oral bioavailability increased disproportionally more than dose, suggesting saturation of drug-metabolizing enzymes or transporters.8 Another study demonstrated an increase of 20- to 25-fold in maximum serum concentration and area under the curve in 4 beagles receiving 6 or 50 mg/kg.11 As such, care should be taken to avoid overdosing, and extra caution may be indicated for patients when hepatic function is of concern.

Tissue Distribution
As with other NSAIDs, grapiprant is highly (about 95%) bound to plasma proteins,3 and its displacement can increase unbound, pharmacologically active drug. However, clearance is likely to increase for the unbound drug. Nonetheless, caution may be indicated when using grapiprant with other highly protein-bound drugs (e.g., cefovecin, doxycycline), particularly if hepatic function is impaired. High binding to proteins at the site of inflammation may prolong efficacy beyond that predicted by its half-life.

Elimination
Grapiprant is cleared primarily via cytochrome-mediated (CYP450) hepatic metabolism, although the specific enzymes have not been identified. Differences in CYP450 metabolism are increasingly being identified as a possible cause of adverse drug events among dogs of different breeds.12 Furthermore, for other NSAIDs, polymorphisms resulting in different rates of hepatic metabolism (poor to ultra-efficient metabolizers) have been identified in dogs.13 Grapiprant metabolites are excreted in bile, urine, and feces, but neither the extent of metabolism nor enterohepatic circulation have been reported. An elimination half-life of approximately 5 hours indicates that the drug will not significantly accumulate in healthy dogs after 24-hour dosing.11 After grapiprant administration is discontinued, most of the drug will be eliminated within 24 hours. Indeed, during a 24-hour dosing interval, approximately 90% of the drug will be eliminated within 15 hours, leaving a substantive drug-free period for organs of elimination, which may facilitate safety. Efficacy may be affected, although binding of NSAIDs to inflammatory proteins at the site of action has been demonstrated to contribute to a longer than expected duration of effect, based on elimination half-life.14,15

Safety of grapiprant in dogs that weigh less than 3.6 kg or are younger than 9 months has not been established.

An elimination half-life of approximately 5 hours indicates that the drug will not significantly accumulate in healthy dogs after 24-hour dosing.11
As with all NSAIDs, the lowest minimum effective dose should be established for each patient. A duration of administration has not been stipulated.

Efficacy
Evidence of grapiprant’s efficacy is largely limited to field trials implemented during the approval process. All clinical trials were based on a placebo rather than a positive control (NSAID). In 1 trial, 262 dogs (131 per treatment group) with osteoarthritis, ranging from 2 to 17 years of age and weighing 4 to 60 kg, received either placebo or grapiprant at the labeled dose of 2 mg/kg PO q24h for 28 days. Evidence of efficacy was based on client-completed Canine Brief Pain Inventory responses and was considered successful for 48% of dogs receiving grapiprant and only 31% receiving placebo. However, the implication of this narrow efficacy difference (17%) warrants further consideration. According to the U.S. Food and Drug Administration (FDA) adverse event reporting site (fda.gov/animal-veterinary/safety-health), the fourth most common adverse event recorded for grapiprant was lack of efficacy, yet this effect was noted in only 1.4% of dogs receiving the comparison drug, deracoxib (TABLE 1). These data indicate a need for head-to-head randomized controlled clinical trials comparing grapiprant efficacy with that of other NSAIDs.

Safety/Adverse Events
The potential safety of grapiprant is supported by 3 sources: animal safety studies, field trials implemented during the approval process (as reported in the Freedom of Information Act and the product package insert), and postmarket surveillance.

Animal Safety Studies
Among 8 beagles receiving 1 or 50 mg/kg

<table>
<thead>
<tr>
<th>ADVERSE EVENT</th>
<th>GRAPIPRANT NO. (%)</th>
<th>DERACOXIB NO. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total†</td>
<td>2,272</td>
<td>10,070</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>703 (30.94)</td>
<td>1,378 (13.7)</td>
</tr>
<tr>
<td>Vomiting/emesis</td>
<td>516 (22.71)</td>
<td>3,384 (33.6)</td>
</tr>
<tr>
<td>Anorexia§</td>
<td>450 (19.81)</td>
<td>2,330 (23.1)</td>
</tr>
<tr>
<td>Ineffective as an anti-inflammatory¶</td>
<td>402 (17.69)</td>
<td>140 (1.4)</td>
</tr>
<tr>
<td>Lethargy</td>
<td>291 (12.81)</td>
<td>413 (4.1)</td>
</tr>
<tr>
<td>Euthanasia</td>
<td>134 (5.90)</td>
<td>1,146 (11.4)</td>
</tr>
<tr>
<td>Elevated ALT</td>
<td>130 (5.72)</td>
<td>1,241 (12.3)</td>
</tr>
<tr>
<td>Increased creatinine</td>
<td>119 (5.24)</td>
<td>1,330 (13.2)</td>
</tr>
<tr>
<td>Weight loss</td>
<td>112 (4.93)</td>
<td>573 (5.7)</td>
</tr>
<tr>
<td>Elevated SAP#</td>
<td>106 (4.67)</td>
<td>1,556 (15.5)</td>
</tr>
<tr>
<td>Increased BUN</td>
<td>99 (4.36)</td>
<td>1,567 (15.6)</td>
</tr>
<tr>
<td>Death</td>
<td>43 (1.89)</td>
<td>995 (9.9)</td>
</tr>
<tr>
<td>Melena/blood in vomitus</td>
<td>39 (1.72)</td>
<td>848 (8.4)</td>
</tr>
<tr>
<td>Renal failure</td>
<td>16 (0.70)</td>
<td>152 (1.5)</td>
</tr>
<tr>
<td>Intestinal perforation</td>
<td>0</td>
<td>542 (12.7)</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>0</td>
<td>575 (5.7)</td>
</tr>
<tr>
<td>GI ulceration**</td>
<td>0</td>
<td>372 (3.7)</td>
</tr>
</tbody>
</table>

*Reporting site is fda.gov/animal-veterinary/safety-health.
ALT=alanine aminotransferase; BUN=blood urea nitrogen; GI=gastrointestinal; SAP=serum amyloid protein.
†Data not corrected for number of units sold.
¶Including bloody diarrhea (n = 160) and loose stools (n = 95).
§Also reported as decreased appetite, not eating, or inappetence.
††Including lack of anti-inflammatory efficacy (122).
#Including increased ALT (n = 130) as well as SAP (n = 106).
**Gastric and intestinal.
grapiprant PO q24h for 9 months (4 dogs/group), no serious clinical-pathologic adverse drug events were noted. Although total serum protein and albumin concentrations decreased significantly compared with those of controls, all remained within normal limits. Indicators of hemostasis did not change. A second study, also among beagles, demonstrated that grapiprant at 6 or 50 mg/kg/day for 15 days was well tolerated. In contrast, studies of deracoxib cited in the product package insert revealed abnormalities consistent with altered renal function in dogs receiving 5 times the recommended dose for 6 months, as well as vomiting, melena, and gastrointestinal lesions in dogs receiving 5 to 50 times the recommended dose for 14 days.

Field Trials
Data from a grapiprant clinical trial involving client-owned dogs with osteoarthritis receiving either placebo (n=131) or grapiprant (n=131) at 2 mg/kg daily for 28 days indicated a higher incidence of adverse drug events in the grapiprant group. However, these events were not serious; the most common were vomiting (24% with grapiprant versus 9% with placebo), diarrhea or soft stool (17% versus 13%), anorexia (9% versus 7%), and lethargy (6% versus 2%).

Similarly, according to the product package insert, among 366 client-owned dogs given 2 or 5 mg/kg q24h or 4 mg/kg q12h, the most common adverse events were intermittent diarrhea, vomiting, and anorexia. Serum alkaline phosphatase and alanine aminotransferase increased and total protein decreased with dose, but the dogs remained clinically normal.

Postmarket Surveillance
A third source of adverse drug event data is the FDA adverse event reporting site. The proportion of the most common adverse events (the number of times the event was reported divided by the total number of reports for that drug) was determined for grapiprant and then compared with the same events reported for deracoxib (TABLE 1). Of the approximately 2300 reports for grapiprant, the most common adverse events were diarrhea (31%) and vomiting (22%). The incidence of serious adverse events was consistently (numerically) lower than that for deracoxib. However, these data do not account for differences in the number of dogs in the United States who received either drug.

Grapiprant is a sulfa drug, as are many NSAIDs approved for use in dogs, but unlike sulfonamide antimicrobials, grapiprant does not contain an arylamine.

Grapiprant is a sulfa drug, as are many NSAIDs approved for use in dogs, but unlike sulfonamide antimicrobials, grapiprant does not contain an arylamine. As such, although allergies typical of sulfonamide antimicrobials are unlikely, adverse events associated with sulfa components may occur. Pork liver is added as a flavoring agent, which could potentially contribute to food allergies.

It is important to remember that even if grapiprant targets only EP4 receptors, these receptors play roles at several other body sites. Thus, grapiprant use may lead to other adverse events.

**DRUG INTERACTIONS**
Two common pathways in drug, diet, or supplement interactions involve efflux transport proteins such as P-glycoprotein (MDR-1, ABCB-1) and CYP450 drug-metabolizing enzymes. According to the product package insert, grapiprant is a substrate for P-glycoprotein, which is present at portals of entry and tissue sanctuaries. As such, extra attention might be given to possible adverse drug events in breeds deficient in P-glycoprotein or if grapiprant is given with other medications known or suspected to interact with P-glycoprotein (e.g., cyclosporine, rifampin, imidazole antifungal drugs [ketoconazole, itraconazole, fluconazole]).

The specific CYP450 enzymes responsible for metabolizing grapiprant have not been reported. However, the effects of other drugs that are broad inducers (e.g., phenobarbital, rifampin) or inhibitors (e.g., cyclosporine, chloramphenicol, imidazole antifungal drugs) of CYP450 enzymes mandate extra attention to potential adverse drug events if used with grapiprant. Grapiprant does not seem to alter the metabolism of other drugs, including itself; the product
package insert indicates that in dogs, grapiprant is not an inhibitor of CYP1A2, CYP2C9, CYP2C19, CYP2D6, or CYP3A4. Furthermore, in beagles receiving either 6 or 50 mg/kg, grapiprant did not accumulate in plasma despite a 28-day treatment period. As such, grapiprant probably can be safely combined with a variety of other drugs; however, more studies are needed.

**SUMMARY**

Overall, grapiprant may be useful for dogs with osteoarthritis. However, its relative efficacy compared with other NSAIDs has not yet been demonstrated.

- **Pharmacodynamics:** Grapiprant antagonizes EP₄ (highly selectively).
- **Pharmacokinetics:** Oral bioavailability is negatively affected by food and potentially by manipulation of the formulation; clearance decreases in a dose-dependent fashion.
- **Efficacy:** Research findings may suggest that the efficacy of grapiprant is less than that of other NSAIDs.

**Safety:** Safety of grapiprant may be superior to NSAIDs but direct comparison studies are needed.

**Adverse events:** Although grapiprant can lead to adverse events typical of NSAIDs, more serious adverse events are not common.

**Drug interactions:** Drug interactions seem to be minimal, suggesting that grapiprant can be combined safely with most classes of non-NSAID analgesic drugs. Nonetheless, because the drug has been approved for only a short time, given that the EP₄ receptor plays a major role in the normal physiology of multiple organs (including the gastrointestinal tract and kidneys) and hemostasis, caution is recommended with its use, particularly in the presence of comorbidities.

More studies are needed to demonstrate the breadth of grapiprant’s analgesic efficacy and to compare its safety with that of COX-2–targeting NSAIDs. To facilitate the generation of safety evidence, report adverse events (including therapeutic failure) to the manufacturer or FDA ([fda.gov/animal-veterinary/safety-health](http://fda.gov/animal-veterinary/safety-health)).

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**Dawn Merton Boothe**

Dr. Boothe received her DVM degree from Texas A&M in 1980, completed a small animal internship at Auburn University in 1980, received an MS degree from Texas A&M in 1985, completed a residency in small animal medicine in 1985, completed a PhrMA Fellowship in 1989, and received a PhD degree in 1989. In 1990, she joined Texas A&M in the Department of Veterinary Physiology & Pharmacology and became a professor. In 2003, she joined the Departments of Anatomy, Physiology and Pharmacology and Clinical Sciences at Auburn in 2003, where she directs the Clinical Pharmacology Laboratory. Dr. Boothe has authored or coauthored approximately 125 peer-reviewed scientific publications, 60 book chapters, and 2 textbooks. She received the Texas A&M University Achievement Award in Teaching, the Jack Mara Scientific Achievement Award for contributions in clinical pharmacology of the critical care patient, Auburn University Outstanding Graduate Student Mentor Award, the Zoetis Award for Excellence in Research, and an Alumni Professorship.
References


Use of Acupuncture for Pain Management

Ronald Koh, DVM, MS, CVA, CVCH, CVFT, CCRP
Louisiana State University School of Veterinary Medicine

Successful pain management encompasses pharmacologic and nonpharmacologic interventions. This is especially true for chronic, neuropathic, or persistent pain. While pharmacologic options remain the mainstays, nonpharmacologic interventions are an important part of a comprehensive pain management plan.

Acupuncture is a safe, nonpharmacologic intervention with minimal adverse effects that most animals tolerate well. It has become more accepted for pain relief in veterinary medicine. In fact, the pain management guidelines published by the American Animal Hospital Association, American Association of Feline Practitioners, and World Small Animal Veterinary Association endorse acupuncture as a safe adjunct treatment for pain management in dogs and cats that should be strongly considered as a part of a multimodal pain management regimen.

Acupuncture can be used independently or integrated into conventional analgesia protocols. It has significant analgesic effects on inflammatory, neuropathic, cancer, and visceral pain states. It can help ease acute pain from neuromusculoskeletal injuries and surgery, as well as chronic spinal and osteoarthritic pain. Veterinary clinical trials also provide evidence for its effectiveness.

**HOW DOES ACUPUNCTURE WORK?**
Acupuncture is the stimulation of certain points on the body that correspond to neurovascular bundles, blood plexuses, sites of nerve branching, and motor endplate zones (Table 1). Recent evidence suggests that the effects of acupuncture are likely mediated by the nervous system at peripheral, spinal, and supraspinal levels. Neurophysiologic effects of analgesia in response to acupoint stimulation include release of endogenous opioids and neurotransmitters (e.g., endorphin/endomorphin, enkephalin, 5-hydroxytryptamine), activation of the descending pain inhibitory pathway, and inhibition of inflammatory mediators (e.g., cyclooxygenase-2, interleukin-1β, interleukin-6). Acupuncture also causes micro-trauma and vasodilation to improve local circulation and catalyze healing. Recent evidence suggests inhibition of microglial activation by acupuncture may play a key role in neuropathic pain diseases.

**CLINICAL Efficacy**
In 1 noncontrolled study, acupuncture alone or combined with analgesics reduced chronic pain and improved quality of life in dogs with neurologic and musculoskeletal diseases. Results were similar for acupuncture plus manual therapy in dogs with musculoskeletal pain; the authors found immediate...
short-term improvement in comfort level and mobility compared with before treatment. 9

In 2 controlled studies in dogs with hip dysplasia, a gold bead implanted at acupoints significantly reduced osteoarthritic pain. 10,11 A 2-year follow-up study revealed that gold-bead acupuncture provided long-term pain relief, an effect not observed in dogs receiving placebo. 12

In another controlled study, neither acupuncture nor carprofen significantly differed from placebo on gait analysis of dogs with hip dysplasia, but only acupuncture was associated with a decrease in validated chronic pain scores. 13 A controlled, blinded study in dogs undergoing hemilaminectomy found significantly lower postoperative pain scores in the acupuncture than the control group. 14

Two studies showed that among cats undergoing ovariohysterectomy, the need for rescue analgesia after surgery was lower in the acupuncture than the control group. 15,16 Similar results were found in dogs undergoing mastectomy. 17

In horses, 2 controlled studies found acupuncture was effective in treating back pain. 18,19 A recent study showed horses with chronic laminitis were improved by acupuncture after receiving 2 acupuncture treatments 1 week apart. 20

<table>
<thead>
<tr>
<th>AFFECTED AREA OR CONDITION</th>
<th>COMMON ACUPUNCTURES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>General pain</strong></td>
<td>LI-4, LIV-3, ST-36, BL-60</td>
</tr>
<tr>
<td><strong>Inflammation</strong></td>
<td>LI-4, LI-11, ST-36, GV-14, Er-jian</td>
</tr>
<tr>
<td><strong>Calming effect</strong></td>
<td>GV-20, GV-21, An-shen, Bai-hui</td>
</tr>
<tr>
<td><strong>Bone and arthritic pain</strong></td>
<td>BL-11, BL-23, KID-3 (combined with local acupoints)</td>
</tr>
<tr>
<td><strong>Dental pain</strong></td>
<td>ST-6, ST-7, LI-4, LIV-3, ST-36, EA: LI-4 bilateral, ST-36 bilateral</td>
</tr>
<tr>
<td><strong>Otitis and ear pain</strong></td>
<td>TH-21, SI-19, GB-2, ST-36, An-shen, EA: ST-36 bilateral, SI-19 to An-Shen</td>
</tr>
<tr>
<td><strong>Abdominal or visceral pain</strong></td>
<td>ST-36, LIV-8, BL-24, ST-25, LI-10, EA: ST-36 bilateral, LI-10 bilateral</td>
</tr>
<tr>
<td><strong>Neck</strong></td>
<td>GB-20, GB-21, GV-14, SI-3, LU-7, BL-60, Jing-jia-ji, EA: GB-20 to GB-21, Jing-jia-ji bilateral</td>
</tr>
<tr>
<td><strong>Shoulder</strong></td>
<td>LI-15, TH-14, SI-9, BL-11, EA: LI-15 to SI-9, BL-11 bilateral</td>
</tr>
<tr>
<td><strong>Elbow</strong></td>
<td>LI-10, LI-11, LU-5, HT-3, TH-10, SI-9, EA: LI-10 to SI-9, LI-11 to HT-3</td>
</tr>
<tr>
<td><strong>Carpus</strong></td>
<td>LI-4, TH-5, HT-7, SI-9, EA: LI-4 to SI-9, TH-5 to HT-7</td>
</tr>
<tr>
<td><strong>Coxofemoral</strong></td>
<td>GB-29, GB-30, BL-54, BL-40, ST-41, EA: BL-54 bilateral, GB-29 to GB-30, ST-41 bilateral</td>
</tr>
<tr>
<td><strong>Stifle</strong></td>
<td>ST-35a, ST-35b, ST-36, GB-34, BL-40, BL-54, EA: ST-36 bilateral, ST-35a/b to BL-40, BL-54 bilateral</td>
</tr>
<tr>
<td><strong>Tarsus</strong></td>
<td>BL-60, KID-3, LIV-3, ST-41, BL-54, EA: ST-41 to KID-3, BL-54 bilateral</td>
</tr>
<tr>
<td><strong>Vertebral column</strong> (intervertebral disk disease)</td>
<td>GV-14, LI-4, ST-36, LIV-3, Bai-hui, Hua-tuo-jia-ji, Liu-feng (front or hind limbs), PC-8, KID-1, EA: GV-14 to Bai-hui, Hua-tuo-jia-ji bilateral, ST-36 bilateral, KID-1 or PC-8 bilateral</td>
</tr>
<tr>
<td><strong>Treatment settings</strong></td>
<td>1. DN: 20 to 30 min</td>
</tr>
<tr>
<td>2. EA: 2 to 20 Hz (dense-disperse waves) for 20 to 30 min</td>
<td></td>
</tr>
</tbody>
</table>
METHODS OF STIMULATION

Acupoints can be stimulated by dry needle (DN), electroacupuncture (EA), aqua-acupuncture (AQ), laser acupuncture (LA), moxibustion, and material implantation (FIGURE 1). Each method traditionally serves a different purpose. DN involves the insertion of fine, sterile needles into acupoints. These needles vary in size (28- to 36-gauge) and length (0.25 to 1.5 inches). The needles are typically left in place for approximately 10 to 30 minutes.

In EA, acupoints are stimulated by applying electricity through needles for 10 to 30 minutes. EA has more profound and prolonged analgesic effects than other techniques. Low-frequency EA (2 to 10 Hz) produces longer-lasting alleviation of inflammatory pain and inhibits nerve injury–related allodynia/hyperalgesia more potently than do higher frequencies (100 Hz).

With AA, 0.1 to 0.5 mL of sterile fluid (e.g., saline, vitamin B₁₂) is injected into acupoints. It is commonly used after DN or EA to prolong the effect of acupoint stimulation.

LA, the modern practice of stimulating acupoints using low-level energy of wavelengths (630 to 960 nm), may provide anti-inflammatory and antinociceptive effects.

HOW IS ACUPUNCTURE INTEGRATED?

Veterinarians who have received formal training can incorporate acupuncture into conventional practice settings. Basic or advanced veterinary acupuncture courses are available at the Chi Institute of Traditional Chinese Veterinary Medicine, or through the International Veterinary Acupuncture Society.

Before acupuncture, underlying pain or medical conditions are always diagnosed as part of conventional care. Once standard treatment measures are underway, acupuncture can be used as an integrative modality to reduce acute or chronic pain. For outpatients, it can be offered at the clinic, once or twice a week. For inpatients, it can be performed in the hospital, once a day before discharge. Practices that do not offer acupuncture can refer patients to veterinarians with CVA (certified veterinary acupuncturist) credentials.

Veterinarians who perform acupuncture must obtain informed consent beforehand. The discussion of acupuncture in the context of conventional medicine must focus not only on the efficacy of acupuncture but also expectations and potential adverse effects. A multimodal approach with acupuncture may allow for a reduction in dose of conventional analgesics and therefore a decrease in their adverse effects. For patients that are resistant to pain medications or cannot tolerate their side effects, acupuncture can be a reasonable alternative treatment.

As with any therapy, not every patient responds to acupuncture; therefore, realistic expectations need to be set for clients. The author often requires clients to commit to sessions once or twice a week for at least 4 to 6 treatments, especially for chronic conditions. Although many patients may not need even 4 treatments to experience benefits, shorter durations and lower intensities of treatment may result in suboptimal outcomes. Acupuncture has both immediate and cumulative analgesic effects following repeated treatments.

SAFETY AND CONTRAINDICATIONS

Acupuncture is safe when performed correctly by licensed veterinarians certified in veterinary acupuncture. Common minor adverse effects after acupuncture include tiredness, increased water intake, soreness, muscle spasm, and minor bleeding, which typically resolve quickly. Other rare complications include infection, dermatitis, and broken needle fragments. Acupuncture needles should not be placed on infected or inflamed skin, open wounds, or sites of tumor and fractures; around the abdomen of a
pregnant animal; or in specific points that may contribute to premature parturition (i.e., ST-36, SP-6, BL-40, BL-60, and BL-67). Deep needle insertion into acupoints around the lung fields (e.g., SP-21, LIV-13, LIV-14, GB-24, BL-12 to BL-19) is contraindicated. Acupuncture should be used cautiously or avoided in patients with clotting abnormalities. Do not apply EA across the thorax area (heart position) in animals with heart disease or pacemakers. Be cautious when using acupoints around the eyes.

CASE EXAMPLES

Case 1: Chronic Pain Associated With Polyarticular Osteoarthritis
A 13-year-old female spayed Weimaraner had osteoarthritis in multiple joints (elbows, carpi, hips, and stifles) and back pain. Despite the combination of firocoxib, gabapentin, tramadol, and glucosamine-chondroitin, her pain was worsening and her mobility was deteriorating. She developed urinary incontinence and was awoken more often during the night.

An internist suggested acupuncture as a last resort before euthanasia. The dog received acupuncture twice weekly for 4 weeks initially, then every 2 to 4 weeks. After 3 treatments, the dog could rise up and walk without assistance and sleep normally. Her urination incontinence was resolved after 6 treatments. She continued to receive acupuncture monthly for pain management. She died at home at age 16.

Her acupuncture treatment consisted of the following:

1. **DN**: GV-20, TH-5, SI-9, GB-34, BL-40, LIV-3, Bai-hui
2. **EA**: LI-4 to LI-11, ST-36 to ST-41, BL-11 bilaterally, BL-23 to *Shen-shu* (crossing), BL-54 bilaterally; 2 to 20 Hz for 20 minutes
3. **AA**: TH-4, LU-5, LI-10, SI-9, BL-23, BL-54, ST-36, BL-39, KID-3; 0.1 to 0.2 mL per acupoint

Case 2: Pain and Neurologic Deficits Associated With Meningoencephalitis of Unknown Cause
A 4-year-old male neutered Yorkshire terrier was diagnosed with meningoencephalitis of unknown cause at the cervical region. He had nonambulatory tetraparesis with severe cervical pain and was hospitalized. He received IV fluids, immunosuppressive doses of dexamethasone, fentanyl constant rate infusion, cytosine arabinoside, and gabapentin. On day 4 of hospitalization, he was referred for acupuncture treatment. Despite medications, his neck was still severely painful on manipulation and he continued to have nonambulatory tetraparesis.

Shortly after his first acupuncture treatment, he could stand on his own unassisted and his cervical pain was markedly improved—he could move his neck without yelping. His fentanyl was discontinued the next day because of an improved pain level. The next day, after his second acupuncture, he became ambulatory on 4 limbs with minimal assistance. He continued to make significant progress over the next 3 days with daily acupuncture. On day 7, he had full range of motion of his neck and was ambulatory with mild ataxia.

His acupuncture treatment consisted of the following:

1. **DN**: GV-20, LU-7, SI-3, LI-4, LIV-3, ST-36, *Jing-jia-ji*
2. **EA**: GV-16 to Bai-hui, GB-20 to GB-21 (crossing), BL-23 bilaterally, PC-8 bilaterally, KID-1 bilaterally; 2 to 20 Hz for 20 minutes
3. **AA**: *Jing-jia-ji*, GV-14, Liu-feng; 0.1-0.2cc per acupoint

**SUMMARY**
Given the low risk for adverse effects and observed benefits for acute and chronic pain, acupuncture can
For patients that are resistant to pain medications or cannot tolerate their side effects, acupuncture can be a reasonable alternative treatment.

Ronald Koh
An assistant professor and section chief of the Integrative Medicine and Rehabilitation Service at the Louisiana State University School of Veterinary Medicine, Ronald Koh received his veterinary degree in Taiwan and completed a specialty internship and master's program in acupuncture at University of Florida College of Veterinary Medicine in 2010 and 2012, respectively. He will be finishing his residency in Veterinary Sports Medicine and Rehabilitation in 2019. His clinical interests include using acupuncture, integrative therapies, rehabilitation, nutrition, and supplements for pain management, neurological disorders, geriatric conditions, and palliative and hospice care.

References
For most canine and feline patients, dental cleanings and thorough evaluation of the oral cavity is recommended at least annually. For these patients, general anesthesia is required for an accurate assessment of the health of the oral cavity and for a thorough performance of dental cleaning. According to the 2013 American Animal Hospital Association Dental Care Guidelines for Dogs and Cats, general anesthesia with a secured airway is necessary for proper assessment and treatment of canine and feline patients.

However, general anesthesia in veterinary patients is not to be taken lightly. Clients have significant concerns and anxiety when thinking about their pets being placed under general anesthesia.

As practitioners, to help reduce the incidence of anesthetic-related complications, we should perform an accurate presurgical/preanesthetic assessment of each patient. A thorough physical examination, baseline screenings, and appropriate diagnostic testing to identify any underlying conditions will help us optimize the condition of the patient before the procedure.

In addition, we should consider adding regional anesthesia to the anesthetic protocol. Regional anesthesia decreases the patient’s dependence on general anesthesia, which benefits both the patient and the practitioner. This article emphasizes the benefits and describes the drugs and techniques involved in proper administration of regional anesthesia.

ASSESSING PAIN
As veterinarians, we do not have the luxury of asking our patients if they are experiencing pain. However, the International Association for the Study of Pain states that the inability to communicate pain does not necessarily negate the possibility that pain is being experienced. Dogs and cats with dental disease experience
When a regional anesthetic drug is injected into canine and feline patients, it is imperative that the administrator first aspirate the syringe to avoid inadvertent intravenous injection.

discomfort that the client and general practitioner may not appreciate. According to the 2015 American Animal Hospital Association/American Association of Feline Practitioner Pain Management Guidelines for Dogs and Cats, an animal’s inability to self-report pain and discomfort leaves the assessment and recognition of pain with the veterinary professional.4 According to a 2014 survey of veterinary surgeons in the United Kingdom, among the top 3 perceived common causes of chronic pain in their patients was dental disease.5 This pain may not be appreciated by the practitioner or client until after the disease has been treated and the patient has resumed its prepain normal behavioral activities.4

**WHAT IS PAIN?**

As described by the International Association for the Study of Pain, pain is a sensory or emotional stimulus associated with actual or apparent tissue trauma that is perceived as unpleasant.6 The purpose of pain is to elicit a reaction from the body to prevent additional damage to the affected area. Response to a painful stimulus can range from hyperalgesia, an exaggerated response to a stimulus normally perceived as painful,7 to allodynia, a painful response to a stimulus that is not normally perceived as painful.7 When pain is incurred for an extended duration, central sensitization (windup) can occur. This response occurs when the associated neurons repeatedly fire at a lower than normal threshold8 and is commonly seen in patients suffering from chronic oral pain (e.g., severe periodontal disease).9

When tissue is injured, the painful response is recognized by the central nervous system through a process called **nociception**. Recognition and processing of the painful stimulus occur in 4 steps: transduction, transmission, modulation and perception.10

1. **Transduction** is the transformation of energy from a painful stimulus into nerve impulses by pain receptors.10 Transduction can be inhibited by multimodal pain-relieving methods (e.g., use of local anesthetics, nonsteroidal anti-inflammatory drugs, and opioids).11

2. **Transmission** is the movement of nerve impulses to the spinal cord and then to the brain.10 This process can be inhibited by use of local anesthetics, opioids, and alpha-2 agonists.

3. **Modulation** is the transmission of the painful stimulus at the spinal cord to be transmitted to the brain as pain or to inhibit further transmission to the brain.10

4. **Perception** is the method by which impulses are recognized as pain.10 Perception can be inhibited by administering opioids, alpha-2 agonists, anesthetics, and inhalants.11

Ideally, multimodal pain management techniques should be used to try to lessen the amount of general anesthesia that is needed for a given procedure. For dentistry and oral surgery, one of the most effective ways to help block a painful stimulus is to use regional anesthesia.

**REGIONAL ANESTHESIA**

For the dentistry and oral surgery patient, regional anesthesia can eliminate transduction and transmission, thereby decreasing pain perception and central sensitization.11 A study conducted in 2013 found that

<table>
<thead>
<tr>
<th>DRUG</th>
<th>CONCENTRATIONS</th>
<th>ONSET</th>
<th>DURATION OF ACTION</th>
<th>PREFERRED DOSE, DOGS</th>
<th>PREFERRED DOSE, CATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine</td>
<td>2%</td>
<td>Within 5 minutes</td>
<td>60-120 minutes</td>
<td>2 mg/kg</td>
<td>1 mg/kg</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>0.25%, 0.5%, 0.75%</td>
<td>8-30 minutes</td>
<td>4-10 hours</td>
<td>2 mg/kg</td>
<td>2 mg (4 quadrant block at 0.5 mg/quadrant)</td>
</tr>
</tbody>
</table>
use of regional anesthesia reduced the minimum alveolar concentration of isoflurane needed without causing any adverse effects on the hemodynamic state of the patients. Keeping patients in a lighter plane of anesthesia increases client satisfaction because the patients recover quickly from the effects of anesthesia and are therefore less likely to be discharged with a drug “hangover.” Regional anesthesia helps create a painless transition from general anesthesia to consciousness and continues to work after the procedure to reduce patient discomfort and allow oral pain medication to begin working.

**COMMONLY USED MEDICATIONS**

**Lidocaine**
A short-acting medication that is commonly used for regional anesthesia is lidocaine. This medication is usually supplied as a 2% solution (20 mg/mL). It has a rapid onset of action (within 5 minutes) and a relatively short duration of action (60 to 120 minutes). If you want intraoperative pain relief only and would like to have the local anesthetic metabolized by the time the patient is awake, this drug would be the most appropriate choice for regional anesthesia. Cats are significantly more sensitive than dogs to this medication. Reported doses are 6 mg/kg for dogs and 3 mg/kg for cats. However, we prefer to use a maximum dose of 2 mg/kg in the dog and 1 mg/kg in the cat (TABLE 1). Note that the total dosage of this medication is additive. If lidocaine is being used as part of an induction agent or maintenance pain medication (e.g., constant rate infusion), the total dosage must not exceed 5 mg/kg in dogs. Our preference is to keep the dosage below 2 mg/kg in both canine and feline patients. A potential side effect of lidocaine administration is central nervous system excitation, which could result in convulsions.

**Bupivacaine**
A longer-acting medication used for regional anesthesia is bupivacaine. This medication comes in different concentrations: 0.25% (2.5 mg/mL), 0.5% (5 mg/mL), and 0.75% (7.5 mg/mL). The onset of effect has been reported to be as short as 8 minutes and as long as 30 minutes, and the effect has been reported to last from 4 to 10 hours, depending on where it is placed. For cats, the toxic dose of this medication is greater than 2 mg/kg total dose. As with lidocaine, cats are also extremely sensitive to bupivacaine. The maximum dosage of this medication should never be exceeded. For dogs and cats, the recommended total dosage of bupivacaine is less than 2 mg/kg (TABLE 1). Unlike lidocaine, bupivacaine is not used in constant rate infusions because it is highly cardiotoxic. However, in dogs and cats, the total dosage for local blocks is additive. Complications include neurotoxic and cardiotoxic complications (e.g., tremors, seizures, coma, respiratory depression, profound cardiac depression, ventricular fibrillation, and asystole).

When a regional anesthetic drug is injected into canine and feline patients, it is imperative that the administrator first aspirate the syringe to avoid inadvertent intravenous injection. We have never experienced any adverse side effects or complications when careful technique is used and recommended dosages are followed.

**ADDITION OF AN OPIOID**
An adjunctive medication that can be added to bupivacaine to extend the action of the regional block is buprenorphine. We recommend that buprenorphine be added to the regional block at a dose of 15 mcg per patient. Studies have shown that when bupivacaine was combined with an opioid such as buprenorphine, it increased the duration of action of the local anesthetic agent by threefold. In a study evaluating the effect of adding buprenorphine to bupivacaine in an infraorbital nerve block and its effects on the minimum alveolar concentration of isoflurane, the anesthetic requirement for patients that received the combination was less than that for patients that received bupivacaine alone. The same study also provided support for prolonged regional anesthetic effects. The authors speculated that the addition of buprenorphine to a regional anesthetic block may extend the duration of the block to 48 to 96 hours after administration instead of 4 to 10 hours without buprenorphine.

**REGIONAL ANESTHESIA TECHNIQUE**
Regional anesthesia is safe for the patient as long as the administrator has a grasp of the anatomy surrounding the region to be injected and uses safe technique. Because regional anesthesia is administered when the patient is under general anesthesia, it can be invasive if careful technique is not used. Another consideration is whether regional anesthesia should be used when biopsy samples are being collected or a resection is
being performed in an area that may contain neoplastic cells. Placement of regional anesthesia in an area containing neoplastic cells could push those cells away from the original tumor margins; therefore, caution should be exercised when performing regional blocks in an area that may be neoplastic.

Anatomy Considerations
Anatomic landmarks for the most commonly used regional anesthesia techniques are the infraorbital foramen, mandibular foramen, angular process, and the facial vascular notch (FIGURE 1). Innervation to the maxilla, and hence the nerve(s) that innervate the dental structures and surrounding soft tissues, is supplied by the infraorbital nerve, which is a branch of
the maxillary nerve and its associated branches. Innervation to the mandible is supplied by the inferior alveolar branch of the mandibular nerve.

Cranial Infraorbital Nerve Block
The cranial infraorbital nerve block inhibits stimulation to the following nerves: infraorbital, incisivomaxillary, rostral superior alveolar dental, external nasal, internal nasal, and superior labial. This block desensitizes the maxillary first, second, and third premolars, canine, and incisor teeth on the same side on which the block is administered. It also desensitizes the associated soft tissues, skin of the muzzle, and the upper lip on the ipsilateral side of block administration.

To perform the cranial infraorbital block in the dog, palpate the infraorbital foramen as a depression in the alveolar mucosa apical (dorsal) to the distal root of the maxillary third premolar or the mesial root of the maxillary fourth premolar. In the cat, the infraorbital foramen is located at the mesial aspect of the third premolar. The needle should be inserted just into the canal, parallel with the canal or directed slightly ventral to it. If you insert the needle apically (dorsally), it could penetrate the retrobulbar space or the globe of the eye. If you insert the needle too far ventrally, it could contact the floor of the infraorbital canal, preventing insertion deeper into the canal. The chosen drug, after appropriate dose calculation, should then be slowly injected into the canal. The drug should be infused into the canal rather than infiltrated directly into the nerve. To prevent intravascular infusion, after inserting the needle into the canal, rotate the syringe 360 degrees, aspirating at each quarter turn. After injection into the canal, remove the syringe and apply digital pressure to the opening of the infraorbital canal for 1 minute.

Caudal Infraorbital (Maxillary) Nerve Block
The caudal infraorbital nerve block inhibits stimulation to the following nerves: maxillary; infraorbital; caudal, middle, and superior alveolar dental; incisivomaxillary; and rostral superior alveolar dental. This block desensitizes the maxillary 1st and 2nd molars and all premolars, canine, and incisors of the ipsilateral quadrant. Also blocked are the bone and soft tissues of the maxilla on the ipsilateral side of block administration, along with the skin of the nose, cheek, and upper lip on the ipsilateral side.

The technique for the caudal infraorbital nerve block is identical to that for the cranial infraorbital nerve block. The needle should be inserted into the canal and directed approximately half the length of the zygomatic arch. The direction of the needle should be parallel with the canal. If the needle is inserted apically (dorsally) or ventrally, the problems described above can occur. The same injection procedure described for the cranial infraorbital block should be followed,
ending with digital pressure to the rostral opening of the canal for 1 minute.

Other approaches to the caudal infraorbital block include the subzygomatic approach and a technique using the maxillary tuberosity. However, because of variations in skull type and breed, we prefer the approach described above.

Caudal Inferior Alveolar Nerve Block
The caudal inferior alveolar nerve block inhibits innervation to the inferior alveolar branch of the mandibular nerve before it dives into the mandible; to the caudal, middle, and rostral mental nerves; and to the incisive nerve.14 Anesthesia to this region desensitizes all teeth (incisors, canine, premolars, molars), associated labial tissues, the rostral lower lip, and the rostral intermandibular tissues on the side in which it is placed.14 There are 2 approaches to the caudal inferior nerve block: intraoral and extraoral.

Intraoral Approach: To perform this block, palpate the angular process of the mandible on the external surface of the patient. This is a palpable prominence located at

The technique for the caudal infraorbital nerve block is identical to that for the cranial infraorbital nerve block.

![Images of caudal infraorbital nerve block](A) Canine skull. (B) Canine cadaver. (C) Feline skull. (D) Feline skull, dorsal view. (E) Feline cadaver.

FIGURE 3. Location of the infraorbital canal and appropriate depth of penetration into the canal for the caudal infraorbital (caudal maxillary) nerve block. (A) Canine skull. (B) Canine cadaver. (C) Feline skull. (D) Feline skull, dorsal view. (E) Feline cadaver.
the caudal-most aspect of the mandibular body (FIGURE 1, A, B, E, F). Insert the needle intraorally through the gingiva at the location of the distal aspect of the mandibular third molar in the dog or the mandibular first molar in the cat (FIGURE 4). Then insert the needle on the lingual aspect of the mandible, as opposed to the buccal surface, directed toward the angular process, attempting to palpate and deposit the block at the opening of the mandibular foramen (FIGURE 5). The opening of the mandibular foramen is located half the distance between the alveolar crest distal to the last molar and the angular process of the mandible. After the needle is inserted into the region of the mandibular foramen, the syringe should be rotated 360 degrees, aspirating at each quarter turn. Because this foramen and nerve may be difficult to palpate, you can place the local anesthetic along periosteum of the body of the mandible in the location of the mandibular foramen, which should cause the local anesthetic to spread over a large surface area. After withdrawing the syringe, apply digital pressure to the area of the foramen for 1 minute to allow the block to diffuse within the tissues.

**Extraoral Approach:** To perform this block, palpate the facial vascular notch (FIGURE 1, A, C, D). This structure is located on the ventral aspect of the caudal mandible. The needle should be inserted directly through the skin in the middle of this structure.
(FIGURE 6), directed parallel with the lingual aspect of the mandible and continued dorsally to half the width of the mandible (FIGURE 7). This is the location of the mandibular foramen, similar to the intraoral approach. As described for the intraoral approach because this foramen and nerve may be difficult to palpate, you can place the local anesthetic alongside periosteum of the body of the mandible in the region of the mandibular foramen, which should cause the local anesthetic to spread over a large surface area. Injection technique is the same as above.

DISCUSSION
Multimodal pain-relieving efforts should be pursued for canine and feline patients. Just because our patients cannot communicate their pain to us does not negate the fact that they are experiencing pain. Regional anesthesia, especially when combined with an opioid, can provide a more comfortable procedure for our patients and a more satisfying experience for our clients. When safe technique is used, regional anesthesia not only helps the dentistry and oral surgery patient but also increases client satisfaction. Regional anesthesia reduces the general anesthetic requirement, provides intraoperative and postoperative pain relief, and contributes to a smoother postoperative recovery. These benefits increase client satisfaction by making the patient’s postoperative recovery more comfortable and allowing the patient to be discharged with less drug hangover. Clients expect the same services for their pets as they do for themselves. We can reduce their concerns about use of general anesthesia for dental cleaning patients if at the time of discharge, our patients are awake, pain-free, and able to ambulate as well as when they came in that morning. TVP

References

FIGURE 6. Location of the facial vascular notch on an anesthetized dog. For the caudal mandibular nerve block, the needle is inserted through the skin and directed on the lingual aspect of the mandible for half the width of the mandible in the location of the mandibular foramen.

FIGURE 7. (A, B) Canine skull showing the location of the mandibular foramen identified by using the facial vascular notch.


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Regional Anesthesia for the Dentistry and Oral Surgery Patient

LEARNING OBJECTIVES
Upon completion of this article, the reader will be able to describe the processing of pain in the canine and feline patient. After reading the article, the reader will be able to recognize common regional anesthesia techniques commonly used in dentistry and oral surgery procedures.

TOPIC OVERVIEW
This article describes regional anesthesia techniques for dentistry and oral surgery procedures in the canine and feline patient. It includes information on medications and dosages that can be used, regional anatomy to be aware of when performing the local blocks and written and photographic diagrams explaining techniques.

1. Which of the following statements regarding pain is incorrect?
   a. Pain is a sensory or emotional experience associated with actual or perceived tissue trauma that is perceived as unpleasant.
   b. The purpose of pain is to allow a reaction from the body to prevent additional trauma or damage to an affected area.
   c. The incapability to communicate pain negates the possibility that pain is being experienced by the patient.
   d. Painful perception can range from an over exaggeration of pain to a painful response from something that is not normally painful.

2. Which statement best describes the definition of allodynia?
   a. An exaggerated response to a painful stimulus
   b. A response to something not normally perceived as painful
   c. Associated neurons are repeatedly firing at a lower threshold
   d. The insensibility to pain without loss of consciousness

3. When tissue is injured, the painful response is processed by the central nervous system through:
   a. Perception
   b. Modulation
   c. Transmission
   d. Nociception

4. Which of the following statements regarding regional anesthesia is incorrect?
   a. Lidocaine is a regional anesthetic medication with a short onset and a short duration of action (60-120 minutes).
   b. Bupivacaine has long duration of action (4-10 hours), and its time of onset is longer than lidocaine.
   c. Dogs are significantly more sensitive to the side effects of lidocaine when compared to cats.
   d. The maximum dose of bupivacaine that should be administered to a cat as a regional anesthetic agent is 2 mg/kg.

5. Which of the following statements regarding the addition of buprenorphine to bupivacaine when performing local and regional anesthesia is correct?
   a. When buprenorphine is added to bupivacaine, a 3-fold increase in the duration of action of the local anesthetic was seen with patients.
   b. The minimum alveolar concentration of isoflurane required for patients was increased when the combination of bupivacaine and buprenorphine was used.
   c. Studies have shown that when buprenorphine is added to bupivacaine, the combination block will last less than 4 hours post administration.
   d. The recommended amount of buprenorphine to be added to the bupivacaine for a regional anesthetic block is 15 mg/kg.

6. True/False. When considering placement of a local block in an area that may contain neoplastic cells (biopsy or surgical resection), the administrator should not be concerned about transporting abnormal cells beyond the margins of the original tumor.
   a. True
   b. False
7. Which of the following statements concerning local blocks is incorrect?
   a. The cranial infraorbital nerve block desensitizes the maxillary 1st, 2nd, and 3rd premolars, canine teeth and incisor teeth on the ipsilateral side of administration.
   b. The caudal infraorbital nerve block desensitizes all maxillary premolars, incisors, and canine teeth as well as the maxillary molars on the ipsilateral side.
   c. The caudal inferior alveolar nerve block (intraoral) desensitizes the incisors, canine, premolars and molars on the ipsilateral side of administration.
   d. The rostral inferior alveolar nerve block (extraoral) desensitizes the incisors, canine, premolars and the 1st and 2nd mandibular molars on the ipsilateral side.

8. You are presented with a 13-year-old, spayed female West Highland White Terrier that you diagnose with stage 4 periodontal disease. You decide that the right maxillary 4th premolar (108), right maxillary 1st and 2nd molars (109, 110) and the left mandibular canine tooth (304) need to be extracted. Which of the following regional anesthetic blocks should be placed to provide intraoperative and post-operative pain relief?
   a. Right caudal inferior alveolar and left caudal infraorbital nerve block
   b. Right cranial infraorbital and left rostral inferior alveolar nerve block
   c. Right caudal infraorbital and left rostral inferior alveolar nerve block
   d. Right caudal infraorbital and left caudal inferior alveolar nerve block

9. True/False: The intraoral approach to the caudal inferior alveolar nerve block deposits the regional anesthetic medication at the same location as the extraoral approach, which is at the middle mental foramen.
   a. True
   b. False

10. Which of the following statements is correct regarding placement of the cranial infraorbital nerve block?
    a. The infraorbital foramen in the dog is located at the mesial aspect of the maxillary 1st premolar tooth.
    b. The infraorbital foramen in the cat is located at the mesial aspect of the maxillary 2nd premolar tooth.
    c. The infraorbital foramen in the dog is located at the distal aspect of the maxillary 3rd premolar tooth.
    d. The infraorbital foramen in the cat is located at the distal aspect of the maxillary 4th premolar tooth.

NOTE Questions online may differ from those here; answers are available once CE test is taken at vetfolio.com/journal-ce. Tests are valid for 2 years from date of approval.
Photobiomodulation (PBMT), or laser therapy, is a rapidly growing treatment modality used for a variety of medical conditions in companion animals. PBMT is painless, noninvasive, and easily administered in a primary care setting.\(^1\) Therapeutic laser devices are estimated to be used by 20% of all companion animal practices in North America.\(^1\)

PBMT accelerates healing in a number of tissues, provides analgesia, and decreases inflammation through modulation of immune and inflammatory responses.\(^2\) PBMT has been used in human and veterinary medicine to improve wound healing, treat snake bites, decrease pain and inflammation resulting from musculoskeletal conditions, improve neurologic function after trauma or injury, treat stomatitis and other oral inflammatory conditions, treat intraoperative and postoperative inflammation, and enhance healing of sport-related injuries.\(^1\) The focus of this article, however, is on treatment of joint conditions in companion animals.

### TABLE 1 PBMT Glossary*

<table>
<thead>
<tr>
<th>TERM</th>
<th>DEFINITION</th>
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<tbody>
<tr>
<td>Coherent</td>
<td>Photons travel in the same phase in time and space</td>
</tr>
<tr>
<td>Collimated</td>
<td>Light divergence is minimized over a distance</td>
</tr>
<tr>
<td>Duty cycle</td>
<td>Percentage of total emission time to total treatment time in a pulsed laser</td>
</tr>
<tr>
<td>Fluence, J/cm(^2)</td>
<td>Energy absorbed per area treated</td>
</tr>
<tr>
<td>Frequency, Hz</td>
<td>Number of waveforms in a defined time interval</td>
</tr>
<tr>
<td>Irradiance, W/cm(^2)</td>
<td>Power intensity</td>
</tr>
<tr>
<td>Joule</td>
<td>Energy unit used to measure dose or rate of energy delivery</td>
</tr>
<tr>
<td>Monochromatic</td>
<td>Light of 1 wavelength</td>
</tr>
<tr>
<td>Spot size</td>
<td>Radius of the laser beam</td>
</tr>
<tr>
<td>Watt</td>
<td>Unit of power measured as 1 J/second</td>
</tr>
<tr>
<td>Wavelength, nm</td>
<td>Distance between crests of electromagnetic waves</td>
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*Hz= hertz; J=joule; nm=nanometer; PBMT=photobiomodulation therapy; W=watt.*

Adapted from a table published in “Photobiomodulation Therapy in Veterinary Medicine: A Review” in Topics in Companion Animal Medicine.\(^7\)
WHAT IS PHOTOBIOMODULATION THERAPY?

Since its development, PBMT has been referred to by many names; terms such as cold laser, low-level laser therapy, phototherapy, and low-level light therapy appear in the literature and have caused confusion. Participants at a nomenclature consensus meeting recommended that the term photobiomodulation be adopted to mean "a form of light therapy that utilizes nonionizing forms of light sources, including lasers, light-emitting diodes (LEDs), and broadband light, in the visible and infrared spectrum." PBMT is defined as a "therapeutic use of light, absorbed by the chromophores found in the body, to stimulate nonharmful and nonthermal reactions within the cell that result in a beneficial therapeutic outcome." Although PBMT describes the effects of the therapeutic modality, the term LASER (commonly lowercased) is an acronym for light amplification by stimulated emission of radiation. Veterinary lasers can be used for either therapeutic or surgical applications, depending on the laser.

HOW DOES PBMT WORK?

Studies have shown that PBMT alters the inflammatory response and affects cell signaling. The main factors underlying the laser’s therapeutic effects are increased reactive oxygen species (ROS), adenosine triphosphate (ATP), and nitric oxide (NO). Increased ROS activates the endogenous anti-oxidant enzyme systems; increased ATP supplies cells with energy for reparation; increased NO promotes angiogenesis, modulates the inflammatory and immune responses, and mediates vasodilation. The fundamental step that eventually results in the production of increased ATP is photostimulation of the enzyme cytochrome c in the mitochondrial respiratory chain. Cytochrome c absorbs light in the spectrum of 500 to 1000 nm (therapeutic window) and breaks the bond with NO, which allows bonding with oxygen and production of cytochrome c oxidase at an optimal rate. Cytochrome c oxidase is responsible for the formation of ATP. Additional electrons are accepted by oxygen to produce ROS.

PBMT reduces the pain and inflammation of osteoarthritis and joint disease through several mechanisms of action. PBMT has been shown to reduce cyclooxygenase 2 and bradykinin production (bradykinins induce pain by stimulating afferent nociceptors). Cytokines and growth factors that have anti-inflammatory, anti-oxidative, and anti-apoptotic properties are increased. PBMT reduces the production of inflammatory markers such as interleukin 1 beta, tumor necrosis factor alpha, and prostaglandin E2. PBMT decreases neutrophils in joint fluid, relieves pain, and increases joint mobility and function. PBMT decreases inflammation in tendons and ligaments while increasing tensile strength, collagen fibril size, and fibroblast production. Research has shown that after cruciate transection and subsequent tibial plateau-leveling osteotomies, PBMT reduces cartilage degeneration and synovial inflammation and improves peak vertical force. It has also been shown to accelerate bone healing and promote recovery of atrophied muscles. All these PBMT effects can be amplified when combined with multimodal therapy for the treatment of joint disease.

<table>
<thead>
<tr>
<th>CLASS</th>
<th>DESCRIPTION</th>
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| 1     | Not hazardous to the eyes and requires no eye protection.  
      | Examples: laser printers and CD players. |
| 1M    | Not hazardous to the eyes unless using optical instruments such as binoculars or microscopes. |
| 2/2M  | Limited to 1 mW of power.  
      | No protective eyewear is needed, but extended viewing is not recommended.  
      | This class includes point-of-sale scanners. |
| 3R    | Have output of up to 5 mW.  
      | Are not a fire hazard.  
      | Only an optical hazard if focused or viewed for an extended period of time. This class includes laser pointers. |
| 3B    | Have output of up to 500 mW and wavelengths from 300 nm up to the far infrared.  
      | Requires protective eyewear.  
      | This class includes therapy lasers from 5 to 500 mW. |
| 4     | Have output greater than 500 mW.  
      | Can burn skin or cause permanent eye damage.  
      | Protective eyewear must be worn when operating these devices. |

*Adapted from a table published in “Fundamental information” in Veterinary Medicine: Photobiomodulation."
**Laser Fundamentals and Classifications**

Fundamental PBMT terms and definitions are summarized in Table 1. All lasers are classified according to potential to cause optical damage by wavelength, power, and exposure duration. Classes 3B and 4 can be used safely; however, classes 1, 1M, 2/2M, and 3R are not appropriate for any use in veterinary rehabilitation. Table 2 describes laser classifications.

### Tissue Penetration

One of the most critical elements of laser therapy is depth of penetration. Laser light is monochromatic, collimated, and coherent, enabling it to penetrate through tissues to a cellular level. When light interacts with biological tissue, it is either absorbed, scattered, or reflected.

### Wavelength

A therapy laser will emit light in the 620- to 1200-nm range, often called the therapeutic window. Wavelengths that minimize scattering and reflection as well as absorption by unwanted chromophores will provide optimal penetration into the tissue and ensure a better therapeutic result. Melanin, hemoglobin, and oxyhemoglobin chromophores absorb shorter wavelengths (600 to 800 nm), making these wavelengths better for superficial areas. Wavelengths above 1000 nm are primarily absorbed by water, making tissue penetration difficult. Surgical lasers, such as the CO₂ laser, produce wavelengths around 10,600 nm, which are strongly absorbed by water and therefore can be used for surgical applications. Wavelengths of 800 to 1000 nm can achieve appropriate depth of penetration to treat most musculoskeletal conditions.

### Power and Duration

Penetration depends on wavelength and tissue type, not laser power (watts [W]) or laser intensity (irradiance) at the tissue surface (W/cm²). Using a higher-powered laser delivers more photons to the penetration depth and also determines the time needed to deliver the energy. Lower-powered lasers must be used for a longer time to achieve the same dose. Very low-powered lasers will have no measurable results even when used for long periods of exposure.

### Dosage

**Manual**

Another consideration with regard to PBMT is dosage applied to the tissue. Dosage is expressed as the amount of energy (joules [J]) delivered to a certain surface area (cm²). When calculating the correct dose, the therapist must consider the size of the patient, body type, coat length and color, skin color, and depth of the condition to be treated. When joint conditions are being treated, the dose can be influenced by the size of the patient, whether the fur is clipped, and the joint involved. In general, the larger the patient, the larger the dose required for a therapeutic effect. For most joints, 8 to 12 J/cm² will work well; however, for some joints (e.g., the elbow), a higher dose may be required. Table 3 lists commonly used doses for joints and Box 1 summarizes the benefits of PBMT for joint disease.

### Box 1 Benefits of Laser Therapy for Joint Disease

- Pain reduction
- Reduced markers of inflammation in the joint
- Reduced swelling and edema
- Increased joint mobility and function
- Stimulation of collagen synthesis
- Decreased dosage or frequency of pharmaceutical use for patients with chronic disease

Adapted from “Musculoskeletal Disorders and Osteoarthritis” in Laser Therapy in Veterinary Medicine: Photobiomodulation.

### Box 2 Benefits of Laser Therapy for Joint Disease

- Pain reduction
- Reduced markers of inflammation in the joint
- Reduced swelling and edema
- Increased joint mobility and function
- Stimulation of collagen synthesis
- Decreased dosage or frequency of pharmaceutical use for patients with chronic disease

Adapted from “Musculoskeletal Disorders and Osteoarthritis” in Laser Therapy in Veterinary Medicine: Photobiomodulation.

### Box 3 Benefits of Laser Therapy for Joint Disease

- Pain reduction
- Reduced markers of inflammation in the joint
- Reduced swelling and edema
- Increased joint mobility and function
- Stimulation of collagen synthesis
- Decreased dosage or frequency of pharmaceutical use for patients with chronic disease

Adapted from “Musculoskeletal Disorders and Osteoarthritis” in Laser Therapy in Veterinary Medicine: Photobiomodulation.

### Preset

Many of the newer laser units have preset protocols for treating various conditions. The operator inputs parameters such as size, coat length and color, and area and condition treated, and the machine uses this input to calculate the fluence required. Settings can be manually changed if the therapist wishes to adapt or change the dose. Protocols vary with the manufacturer, and it is in the best interest of the patient for the practitioner to understand laser dosimetry. However, the presets on newer machines have increased safety features and enable veterinarians to confidently delegate delivery of the therapy to well-trained persons.
TREATMENT TECHNIQUES

Before beginning treatment, ensure that the patient is wearing protective eyewear and is comfortable and appropriately positioned, providing good access to the area being treated. If that area is a joint, ensure access to all sides of the joint. Passive range of motion therapy before and after PBMT is a good idea to ensure improved function.

Treatment techniques will vary according to the condition treated, the joint treated, and the type of laser used. In general, clipping the area will allow the best penetration of light to the underlying tissues; however, if clipping is not possible then the dosage needs to be adjusted. Be cautious not to overheat the coat or skin if using lasers with higher wattage or wavelengths less than 900 nm.

When treating joints, treat a broad area. For example, treat the specific joint and surrounding muscles and tendons as well as satellite areas of pain. Treating a comprehensive area will ensure a more consistent outcome.

Treatment technique will vary with the laser used. Lower-powered lasers (less than 1 W) can use a point-to-point method in which a dose is delivered for up to 30 seconds in 1 location before the probe is moved. This method can be more time-consuming, depending on which joint is being treated and whether multiple joints are involved. Higher-powered lasers use a scanning method that delivers the dose over a large area, ensuring that the handpiece is moving during treatment. The therapy can be delivered with a contact or off-contact method, depending on the unit. The contact method allows for tissue compression and can cause deeper penetration. The off-contact method is frequently used over bony prominences or excessively painful areas.

For acute or chronic painful joint conditions, it is useful to begin with an induction phase of treatment, followed by more frequent treatment sessions until a significant effect is noticed. For patients with acute joint injury or a flare-up of chronic arthritis, daily treatment is recommended. After clinical signs have improved significantly, then treatments can be reduced to twice weekly for 2 to 3 weeks and then further reduced to maintenance according to the patient’s response. It is not unusual for patients with osteoarthritis to receive treatment every 3 to 6 weeks, depending on response. In general, 4 to 6 treatments are needed to see improvement, although 8 to 10 sessions may be needed for patients with multiple joint involvement or severe disease. Be sure that clients are aware that each patient responds differently to PBMT.

USE OF PBMT WITH METAL SUTURES AND IMPLANTS

Smooth metal implants and staples will primarily reflect diffuse near-infrared light; thus, heating of the implants is not a concern. However, with small patients (e.g., cats and small dogs) the implant will be covered.

<table>
<thead>
<tr>
<th>JOINT</th>
<th>DOSE</th>
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<tbody>
<tr>
<td>Carpus</td>
<td>1-4 J/cm²</td>
</tr>
<tr>
<td>Elbow</td>
<td>4-8 J/cm² but may be up to 20 J/cm² for chronic conditions</td>
</tr>
<tr>
<td>Shoulder</td>
<td>8-10 J/cm²</td>
</tr>
<tr>
<td>Hip</td>
<td>10-12 J/cm² or higher for large breed dogs</td>
</tr>
<tr>
<td>Stifle</td>
<td>4-8 J/cm² for small dogs; 10-12 J/cm² for large breed dogs</td>
</tr>
<tr>
<td>Tarsus</td>
<td>1-4 J/cm²</td>
</tr>
</tbody>
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by superficial tissue only. Because of the light reflection, these areas will need an increased dose; therefore, to ensure patient comfort, adjustments need to be made to decrease the power or time of treatment. Because the light does not penetrate the hardware, apply the laser 360 degrees around the limb. Do not hover the laser over sutures; instead, apply the laser to both sides of the suture line.6

PRECAUTIONS FOR PBMT USE
Keep in mind the following safety precautions when using PBMT:1
1. Use protective eyewear (for patient and therapist), specific for the laser being used.
2. Do not treat over a pregnant uterus or open fontanelles.
3. Do not treat over malignancies.
4. Remove all metal from the patient (e.g., jewelry, leashes, collars).
5. Use caution with dark skin (melanin increases absorption by chromophores). Use your hand to monitor skin temperature while PBMT is being applied.

SUMMARY
PBMT is a valuable modality that can be used to treat a variety of joint conditions in dogs and cats. For PBMT to be effective, the dose must be appropriate for the particular condition, joint, and patient. Additional veterinary clinical studies are required to document further benefits and determine optimal parameters for all applications. Tvp

References

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Manipulative Therapies for Hip and Back Hypomobility in Dogs

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Does the following scenario sound familiar? A client brings in their young to middle-aged canine athlete and claims that the dog moves stiffly, has a roached topline, has changed its gait, or is not as fast as it used to be. An examination shows no signs of lameness, but does reveal a little pain on palpation of the thoracolumbar junction or on hip extension. Radiographs show no significant abnormalities, so the dog goes home with a prescription of rest and nonsteroidal anti-inflammatory drugs (NSAIDs).

At follow-up, the client reports that the NSAIDs helped a little, but they really don’t want to keep the dog on them forever, especially without a diagnosis. Additional diagnostic imaging modalities provide no definitive diagnosis, leaving the client with two options: keep the dog on NSAIDs or retire it. Hitting this dead end is frustrating for veterinarians, but even more so for the client.

Later, the client hears anecdotes from friends about dogs that underwent chiropractic/spinal manipulation, which greatly improved the dogs’ mobility and enabled them to jump again, resolved the intermittent lameness, or returned them to competition. The client seeks out this therapy for their dog and is happy with the results. And they wonder: Why didn’t my veterinarian recommend this?

Many veterinarians are uncomfortable with referring patients for chiropractic/spinal manipulation. The perception is that there is very little evidence-based medicine on manipulative therapies, and the risk of herniating discs or hurting the dog is too high. However, with educated referrals to trustworthy practitioners (Box 1), primary care veterinarians can not only expand their ability to help their patients feel and perform better, but also help maintain positive, trusting relationships with clients.

HISTORY OF CHIROPRACTIC/SPINAL MANIPULATION THERAPY

The term chiropractic comes from the Greek words cheir (“hand”) and praktos (“done”), thus translating to “done by hand.” It was first used in 1895 when D.D. Palmer performed the first chiropractic adjustment on a janitor, “curing his deafness.”1 Before this, however, various forms of spinal manipulation had been used for hundreds or even thousands of years. Hippocrates (460–357 BC) stated, “Get knowledge of the spine, for this is the requisite for many diseases.” Claudius Galen (130–200 AD), the “prince of physicians,” cured a Roman scholar (Eudemus) of a paralyzed hand by “adjusting” his neck. Traditional Chinese Medicine uses a form of spinal manipulation and manual therapy called Tui-na.
In 1905, D.D. Palmer was arrested for practicing medicine without a license, as were many other chiropractors over the following decades. In 1963, the American Medical Association (AMA) developed a “committee of quackery” designed to “contain and eliminate” the chiropractic profession, and until 1980 they considered it unethical for physicians to associate with “unscientific practitioners.” In 1987, the AMA was found to have unlawfully conspired to “contain and eliminate the chiropractic profession.” Since then, spinal manipulation has been gaining recognition and an effort has been made to provide more evidence-based medicine in manipulative therapies. However, the evidence is still lacking and the number of certified animal chiropractors remains limited, so many clients who desire manipulative therapies turn to practitioners, who may not have had appropriate training, for help.

**PRINCIPLES OF THERAPY**

Chiropractic/spinal manipulation is based on the theory that joint restrictions cause biomechanical and/or neurologic alterations. In chiropractic, the impediment of neurologic input due to a restriction in normal joint motion is referred to as the “subluxation” or “vertebral subluxation complex,” based on the original theory that physical displacement of a bone was causing direct pressure on a spinal nerve. However, current research has shown that simple bone displacement is not always detected, and that inflammation and edema, as well as a decreased range of motion in a joint, may also cause a change in nerve conduction. This limitation in motion decreases stimulation of mechanoreceptors, thus affecting afferent communication of the joint with the spinal cord; furthermore, increasing stimulation of joint mechanoreceptors by joint mobilization may inhibit the transmission of nociceptors. Another theory suggests that rotation of cervical vertebrae may cause a twist in the dura mater, resulting in stretching of the dentate ligaments and thereby pulling on the spinal cord. Resolving inflammation and edema, relieving tension or pressure on nerves and fascia, and decreasing nociceptor stimulation by improving joint range of motion are the principles of how chiropractic/spinal manipulation is believed to help with pain and function.

**CURRENT RESEARCH**

There is clearly a lack of high-quality prospective, randomized, placebo-controlled clinical trials on manipulative therapies in veterinary and human research. However, several recent reviews and meta-analyses on the various uses of spinal manipulation in people show moderate evidence for the indications of its use. Primary literature in the human field has shown that a single cervical manipulation is capable of producing immediate and short-term benefits for mechanical neck pain; that high-velocity, low-amplitude (HVLA) manipulative therapy significantly decreases neck pain, arm pain, and neck disability index scores; and that applying a mechanically assisted instrument to the level of C5 improved shoulder strength for internal rotation.

In veterinary research, there is minimal data on the use of chiropractic/spinal manipulation, with most of the research focusing on equine than canine patients. Equine studies have shown that spinal manipulative therapy increases amplitudes of dorsoventral displacement of the thoracolumbar vertebral column, which increases passive spinal flexibility in actively ridden horses, increases spinal mechanical nociceptive

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**BOX 1 Professional Chiropractic Associations and Training Programs**

The American Veterinary Chiropractic Association (AVCA) and the International Veterinary Chiropractic Association (IVCA) are professional organizations that require members to pass a certification examination and perform continuing education. Veterinarians do not have to belong to one of these organizations to practice animal chiropractic or spinal manipulation; however, most states require that chiropractors practicing animal chiropractic belong to one of these organizations. Depending on the state, they may need to practice under the direct supervision of a veterinarian.

There are currently 6 training programs in animal chiropractic or veterinary spinal manipulation in the United States:

- Animal Chiropractic Education Source (ACES; Meridian, TX)
- Healing Oasis (Sturtevant, WI)
- Health Pioneers Institute (Naperville, IL)
- Integrative Veterinary Medicine Institute–Veterinary Medical Manipulation (IVMI-VMM; Reddick, FL)
- Options for Animals (Wellsville, KS)
- Parker University (Dallas, TX)
Several certification programs exist for veterinarians and chiropractors to become trained in animal chiropractic or veterinary spinal manipulation.

thresholds, and produces a less extended back and better symmetry in pelvic motion.\textsuperscript{18-21}

to the author’s knowledge, only 2 recent publications on canine manipulative therapies exist in primary literature.\textsuperscript{22,23} One study evaluated “manual therapy” and not necessarily manipulative therapy in combination with acupuncture for the treatment of musculoskeletal pain; however, this study did not define what type of manual therapy was used: spinal manipulation, joint mobilization, or massage/soft tissue mobilization.\textsuperscript{21} The other paper retrospectively evaluated chiropractic abnormalities in dogs with urinary incontinence/retention, and found that most of the chiropractic lesions associated with urinary incontinence and retention, identified by this single observer, were between L3 and L5.\textsuperscript{22} Anecdotal reviews of chiropractic therapy for veterinary patients similar to this paper have also been published.\textsuperscript{24,25} These reviews have been met with criticism based on the minimal amount of evidence from primary literature; however, such articles should drive interest in conducting further high-quality research.

**LIMITATIONS AND CONTRAINDICATIONS**

It should be stressed that pursuit of a diagnosis through advanced imaging and other warranted testing should be strongly recommended before chiropractic/spinal manipulation is performed. Every effort should be made to rule out diseases such as neoplasia and trauma, including fractures, luxations, and ligament tears. Severe neurologic dysfunction is not likely to be improved by chiropractic/spinal manipulation, as parenchymal changes to the spinal cord cannot be alleviated by correcting alignment/hypomobility issues.
It may take several treatments to correct alignment/hypomobility of joints, and alignment issues may not be resolvable in patients with conformational abnormalities. Similarly, hypomobility is not expected to resolve in cases of osteoarthritis and spondylosis; however, these patients may gain some relief by diminishing the activity of nociceptors. In patients with conformational challenges or patients that perform chronic repetitive activities, hypomobility and alignment issues are likely to recur. Recurrence may also develop if associated soft tissues are not addressed through stretching, relieving trigger points, and performing strengthening activities.

CHIROPRACTIC/SPINAL MANIPULATION FOR HIP AND BACK HYPOMOBILITY: BASIC MOBILIZATIONS

When performed by a qualified practitioner, the specific adjustments for restricted thoracolumbar facet joints and sacroiliac joints described below can provide pain relief as well as improve spinal mobility in dogs. Improving “stiffness” or hypomobility of the spine (or any restricted joint) can theoretically prevent hypermobility of a nearby joint, which can result in injury (e.g., cranial cruciate ligament rupture, medial shoulder instability, intervertebral disc disease, and chronic ventral longitudinal ligament damage leading to spondylosis).

Thoracolumbar Anatomy

From T1 to T10, the dorsal spinous processes (DSPs) face caudally; at T11, the DSP is straight vertical (anticlinal); and from T12 to L7, the DSP angle slightly cranially. The facet joints from T1 to T10 are in the dorso-ventral plane; from T10 to L7, they are in the sagittal plane (FIGURE 1).

Adjustment

Adjustments are made to the facet joints by performing an HVLA thrust on the restricted segment, at a direction and angle dictated by the location and number of restricted joints.

Joints from T1 to T10: If the vertebra is rotated or one side is restricted but not the other, the HVLA thrust is straight lateral to medial toward the restriction (FIGURE 2). If both facet joints are restricted, it is dorsal to ventral at about a 45° angle caudal to cranial (or along the plane of the DSP) toward the restriction.
Joints from T11 to L7: If both facet joints are restricted, the HVLA thrust is straight dorsal to ventral (FIGURE 3). If the vertebra is rotated, or one facet joint is stuck while the other is not, the thrust is at a 10° lateral to medial angle toward the restriction (FIGURE 4).

Sacroiliac Anatomy
In dogs, the sacroiliac joints are angled about 20° medial to lateral on the sagittal plane (FIGURE 5). The sacroiliac joints can be restricted in several ways, with the most common being the ilium stuck dorsally (posterior/inferiorly [PI]) or ventrally (anterior/superiorly [AS]).

Adjustment
Adjustments are made to correct an AS or ventrally restricted ilium by performing an HVLA ventral to dorsal with a 20° lateral to medial angle. When correcting a PI or dorsally restricted ilium, an HVLA is performed dorsal to ventral with a 20° medial to lateral angle (FIGURE 6).
IMPLEMENTING MANIPULATIVE THERAPIES IN GENERAL PRACTICE

Becoming personally certified or having a certified associate is the ideal way to add chiropractic/spinal manipulation services to a veterinary practice, as the training gives practitioners a better understanding of the specific conditions that can best be helped by chiropractic/spinal manipulation. Several certification programs exist for veterinarians and chiropractors to become trained in animal chiropractic or veterinary spinal manipulation (BOX 1).

If personal certification is not feasible, developing a referral relationship with a local qualified practitioner is a good option. If the practitioner is a chiropractor and not a veterinarian, state law may require direct supervision by a veterinarian, so direct referral may be necessary. Alternatively, he or she could come into the practice on a regular basis (e.g., once a week) to see patients at the facility.

Once the practice is ready to offer chiropractic therapy/spinal manipulation services, promoting it to clients is not difficult. Practices that already offer physical therapy/rehabilitation services or acupuncture may find that they already have a built-in, interested client base.

And, as in the opening scenario, satisfied clients tend to recommend their friends, so the service becomes self-promoting. At the least, it benefits veterinarians to be aware of the possibilities of manipulative therapies to have informed conversations with clients. TVP

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A

B

FIGURE 6. Patient and hand thrust positioning for manipulation of restricted sacroiliac joint.
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