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Regional Anesthesia and Analgesia for Oral and Dental Procedures

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Rationale for using multimodal analgesia

We have come a long way in our treatment of animal pain, especially within the last decade. Research has extended to veterinary medicine many of the practices and products previously used in the human field. Incorporating local anesthetic nerve blocks and multimodal analgesia into daily practice is beneficial for the client, the patient, the veterinarian, and the practice.

Pets are becoming members of society's definition of a nuclear family, and as such, clients are looking for the same level of care for their pet as they would expect for themselves. Concern for their pet's comfort or safety may cause a client to decline a necessary procedure or to have the procedure done elsewhere. Veterinarians who emphasize analgesic care are perceived as sensitive, caring, and more competent, which reassures the client. A pet that is appropriately managed should be comfortable at home and should need smaller quantities of analgesics and less frequent dosing. This means cost savings and less stress for the client. All these benefits translate to a satisfied client.

The patient benefits from nerve blocks and preemptive analgesics because they lead to a decrease in intraoperative and postoperative pain. Less pain means that a lighter anesthetic plane can be used, which translates to more stable vital organ function, a smoother recovery, and earlier discharge from the hospital. Preemptive analgesia means that less potent and smaller amounts of analgesics are needed in the postoperative period, which means less "work" for the animal's system and lower risk of toxicity. A comfortable recovery, hospital stay, and recuperation mean that the animal is less likely to self-traumatize and that pain-induced immune suppression,

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cardiac rhythm disturbances, hypertension, inappetence, and cachexia are avoided, which translates to better healing. A less stressful experience at the veterinary clinic means that the animal is also less distressed about visiting the clinic in future, a fact that the client certainly notices.

Your business should benefit from offering this service, because clients are more willing to allow current and future procedures to be performed on their pet if they perceive such procedures to be pain-free. The equipment needed to perform nerve blocks is already available in a veterinary hospital, and only one type of regional anesthetic agent is needed for all species of all sizes. Using analgesics leads to saving on anesthetic injectables and gases and, if billed out appropriately, can actually generate revenue. A decrease in the dosing frequency of postoperative analgesics translates to savings of your technician's time. A comfortable patient is also significantly safer to work with because such patients have calmer recoveries and are less likely to show aggression when they are handled after surgery. This all translates to increased revenue for the clinic.

The benefits to you, the practitioner, include a safer working environment, expansion of your skill base, and the comfort of knowing that you are offering humane medicine.

Process of pain generation

Effective treatment of clinical pain depends on understanding the mechanisms involved in the formation and maintenance of a pain experience. Noxious stimuli are converted into electrical activity at sensory nerve endings. The neural impulses are then transmitted via the dorsal root ganglia to the dorsal horn of the spinal cord. The electrical signals are sent on to the cerebral cortex. Once the impulses reach the cortex, perception occurs in the conscious patient.

The noxious stimuli that begin the pain process can arise in two phases. The first phase is the sensory input, which arises directly from oral manipulations. The second and more prolonged phase of noxious stimulation is the result of inflammation caused by the surgery. Inflammatory mediators cause the impulse threshold in the sensory nerve endings to decrease, resulting in a state of hyperalgesia and peripheral sensitization. Disproportionate numbers of impulses are sent on to the dorsal horn, where *N*-methyl-D-aspartate (NMDA) receptors respond to this repeated exposure by accelerating their own rate of pulse discharge, further amplifying the signal. This increased NMDA receptor activity is known as central stimulation or "wind-up." Dorsal horn neurons stay "wound up" even after the original noxious stimuli stop [1], causing non–pain-related signals to be interpreted as painful. Research has found that central sensitization can last days, weeks, or possibly a lifetime, such that a single painful insult early in life may have such long-lasting effects as to lower a patient's pain threshold permanently [2]. The purpose of preemptive analgesia is thus to

prevent the transmission of noxious impulses to the brain and to stop central sensitization from developing.

The pain experience can be modulated in several ways. A local anesthetic block shuts down the formation of a painful sensation by preventing neural impulses from reaching the spinal cord. All other medications modulate or ameliorate a pain impulse that has been allowed to form or the conscious perception of that pain. Acute pain impulses are well controlled with opioids because these agents modulate wind-up and conscious perception of pain. Chronic pain requires aggressive multimodal therapy as soon as it is diagnosed, because physiologic changes in neuronal nociceptive processing occur and may lead to "resistance to treatment" [3] or opioid tolerance [4]. The degree of pain and its source (eg, somatic, neuropathic) combined with duration, species of interest, and country of residence determine the most appropriate therapeutics to add to the opioid.

There are many grading systems for determining how much pain an animal is in; however, mild to moderate or moderate pain should be expected with most dental procedures. For stomatitis, multiple extractions, fracture repair, head trauma, cancer, and mucositis after radiation therapy, expect moderate to severe or severe pain. Severe to excruciating pain should be anticipated with bone cancer, especially after a biopsy [5]. The World Health Organization (WHO) has a recommended analgesia ladder (Table 1) [4].

Most dental pain is somatic (ie, arising from diseased tissues), but cancer or trauma, for example, may cause neurogenic distress by directly insulting the nervous tissues. Therapeutics, such as tramadol (Ultram; Ortho-McNeil Pharmaceutical, Raritan, NJ) or gabapentin (Neurontine; Park-David Division of Pfizer, New York, NY) may be useful in these cases. Most dental cases have some degree of chronicity to them by the time the patient is presented for treatment. The actual dental manipulations cause acute stimulation, but chronic pain should be expected with odontoclastic resorptive lesions, stomatitis, and cancer, for example. NMDA receptor blockers are helpful. Species of interest can be a factor in choosing medications, because cats react poorly to many drugs that are used safely in dogs and many products are not labeled for use in cats. Country of residence can affect access to drugs, such as buprenorphine (Buprenex; Reckitt

Table 1
The World Health Organization recommended analgesia ladder

Mild pain	Treat with NSAIDs, acetaminophen
Moderate pain	Treat with NSAIDs + mild opioids
Severe pain	Use a stronger opioid, perhaps added to NSAID
Refractory pain	Control may require alternative routes of delivery, interventions, blocks, neural stimulations, neurolysis.

Adapted from Veterinary Information Network. Pain management in cancer patients. Available at: www.vin.com/Members/Proceedings/Proceedings.plx?CID=WVC2004&PID=pr05460&O=VIN. Accessed September 2004.

Benckiser Pharmaceuticals, Richmond, VA), which is not available in Canada.

Local anesthetic agents

Local anesthetic agents prevent or retard the conduction of afferent pain impulses by entering and occupying ion channels in a nerve cell membrane, preventing depolarization. Uptake into the membrane is improved with a higher concentration of agent or a larger volume. Blood flow through the area decreases the quantity and concentration of agent situated around the nerve. Duration of effect is thus improved if vasoconstrictors are added to the injectable product. Cell membrane uptake is poor; therefore, blockade is decreased in an infection or in an acid environment.

The most commonly used local anesthetic agents are mepivacaine, lidocaine, and bupivacaine. Time to onset of sensory blockage is fastest for mepivacaine (Carbocaine; AstraZeneca, Wayne, PA) and slowest for bupivacaine (Marcaine; AstraZeneca, Wayne, PA). Mepivacaine is effective for 1.5 to 2.0 hours, whereas bupivacaine begins to attenuate after 6 hours. Lidocaine (Xylocaine; AstraZeneca, Wayne, PA) produces onset of analgesia in 2 to 5 minutes and lasts 20 minutes (without epinephrine) to 2 hours (with epinephrine). These products can be purchased in ampules that contain 1.8 mL or in larger multidose bottles.

The maximum safe dose of local anesthetic agent for a dog or cat is 2 mg/kg divided between the necessary sites. If the patient is small, the total volume allowed could be quite limited. In these animals, lidocaine can be diluted with saline [6] or the 0.25% solution of bupivacaine can be used, because larger volumes can be infused without reaching toxic doses. In practice, bupivacaine 0.5% at a rate of 0.25 mL per site is adequate to achieve full desensitization in a cat. Even in large dogs, 1 mL per site is sufficient to achieve complete analgesia. A 1- or 3-mL syringe with a 0.625- to 1.5-inch, 25-gauge needle is usually adequate for placing the blocks.

Facial innervation

The pain receptors in the dental hard and soft tissues are free nerve endings. A-δ fibers transmit sharp localized pain; A-β fibers conduct touch and pressure; and C fibers provide the sensations of burning, aching, and throbbing [7]. These fibers are incorporated into nerves that form the sensory branches of the trigeminal (fifth cranial) nerve. The branches of concern to oral and dental surgeons are the maxillary and mandibular divisions.

The maxillary division leaves the trigeminal ganglion and exits the brain case through the foramen rotundum, courses through the periorbita, and enters the infraorbital canal. Just before entering the caudal limit of the

infraorbital canal, the nerve sends off branches that become the major and minor palatine nerves. These nerves innervate the hard and soft palates, their mucosa, and the nasopharynx. These branches are desensitized with the maxillary nerve block. The maxillary division also gives off the caudal maxillary alveolar nerve, which supplies the maxillary molars and their associated soft tissues and is blocked with the caudal infraorbital nerve block. After giving off the caudal maxillary alveolar nerve, the maxillary nerve enters the infraorbital canal and is now called the infraorbital nerve. While the infraorbital nerve is traversing the infraorbital canal, it gives off two more branches that exit ventrally from the canal. The middle maxillary alveolar nerve innervates the premolars and associated buccal gingiva. The rostral maxillary alveolar nerve supplies the canine, incisors, and associated buccal gingiva. The remaining fibers of the infraorbital nerve then exit the cranial extent of the infraorbital canal to innervate the lateral and dorsal cutaneous structures of the rostral maxilla and upper lip. The middle maxillary alveolar, rostral maxillary alveolar, and infraorbital nerves are blocked by the cranial infraorbital nerve block.

The mandibular division of the trigeminal nerve arises from the trigeminal ganglion, exits the cranium via the foramen ovale, and divides into multiple branches. One such branch is the mandibular, or alveolar, nerve. The mandibular nerve enters the mandible on the lingual side via the mandibular foramen. The nerve then courses rostrally within the bone to innervate the mandibular teeth to the mesial midline; this nerve can be blocked with the mandibular nerve block. At the level of the second premolar (dogs) or rostral to the third premolar (cats), the mandibular nerve gives off mental nerve branches. These branches exit through the mental foramina and serve the cutaneous areas of the chin, lip, and rostral buccal gingiva and mucosa. These nerves, and possibly the mesial portion of the mandibular nerve, can be blocked with the mental nerve block.

Sites for regional anesthetic placement

Cranial and caudal infraorbital nerve blocks

The cranial end of the infraorbital foramen is located apical to the distal root of the third premolar just ventrorostral to where the zygomatic arch meets the maxillary bone. The anesthetic needle should be directed slightly dorsal to horizontal and slightly mesiad to the long axis of the maxilla. The block anesthetizes the ipsilateral premolar, canine, and incisor teeth as well as associated soft tissues. If the needle is advanced deep into the foramen or if digital pressure is placed over the cranial end of the infraorbital canal after injection, a caudal infraorbital nerve block has been accomplished, and the caudal maxillary nerve is also desensitized. This block anesthetizes all ipsilateral dentition and soft tissues, including the molars (Figs. 1B, 2B, and 3).

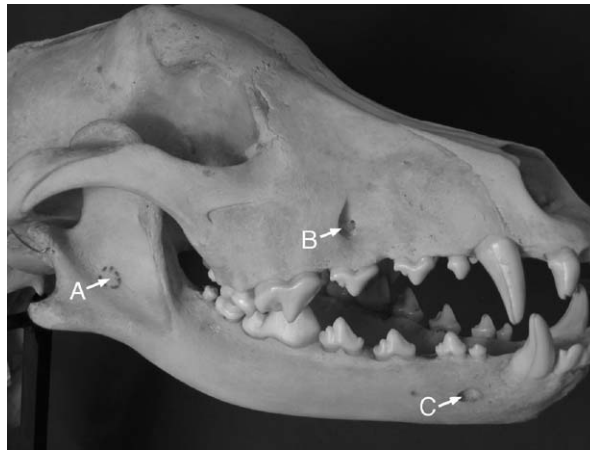

Fig. 1. Foramina of interest in the canine skull. The arrow next to A points to the position of the mandibular foramen on the lingual side of the mandible. The arrow next to B points to the infraorbital foramen. The arrow next to C points to the middle mental foramen.

Maxillary nerve block

The maxillary nerve block desensitizes the complete hemimaxilla, including the soft tissues, dentition, and palate. If an approach is made as though for a caudal infraorbital nerve block but is carried slightly further, the needle should approximate the orbital end of the infraorbital canal. The major and minor palatine nerves are in the immediate vicinity; thus, depositing anesthetic agent to diffuse throughout the area is likely to

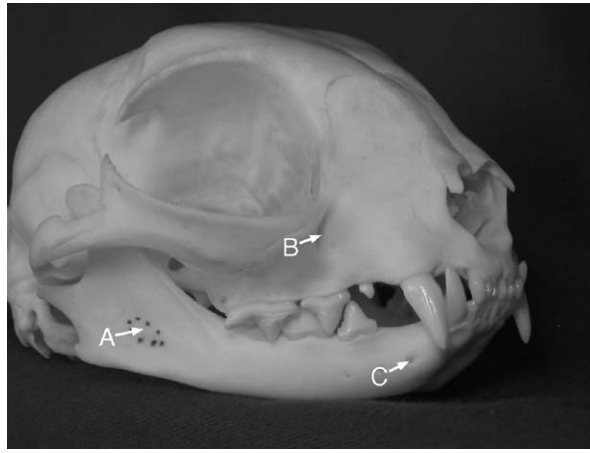

Fig. 2. Foramina of interest in the feline skull. The arrow next to A points to the position of the mandibular foramen on the lingual side of the mandible. The arrow next to B points to the infraorbital foramen. The arrow next to C points to the middle mental foramen.

desensitize these nerves as well (see Fig. 3). Alternatively, there is an extraoral technique by which the needle is inserted through the skin perpendicular to the long axis of the head, under the rostroventral limit of the zygomatic arch, at the dorsocaudal limit of the hard palate (Figs. 4 and 5).

Mental nerve block

In the dog, the middle mental foramen is palpated ventral to the mesial root of the second premolar. In the cat, the foramen is located under the lip frenulum approximately equidistant between the third premolar and the canine. If the needle enters the foramen, the block should anesthetize the ipsilateral soft tissues, the canine and incisor teeth, and possibly the premolars. If the anesthetic is deposited outside the foramen, only the buccal soft tissues from the canine forward to the midline receive analgesia (Figs. 1C and 2C; Fig. 6).

There is concern that penetration of a foramen with a needle may cause trauma to the nerve. This is especially possible when attempting to block the mental nerve in a small animal. For this reason, it is recommended to use the mandibular nerve block in cats and small dogs, which should provide blockade for the area while avoiding iatrogenic trauma (Fig. 7).

Mandibular (alveolar) nerve block

The mandibular nerve block can be done intraorally or extraorally. The foramen is a depression located on the medial side of the ramus of the mandible. It is approximately equidistant between the mesial and distal borders of the ramus and at a height midway between the dorsal and ventral

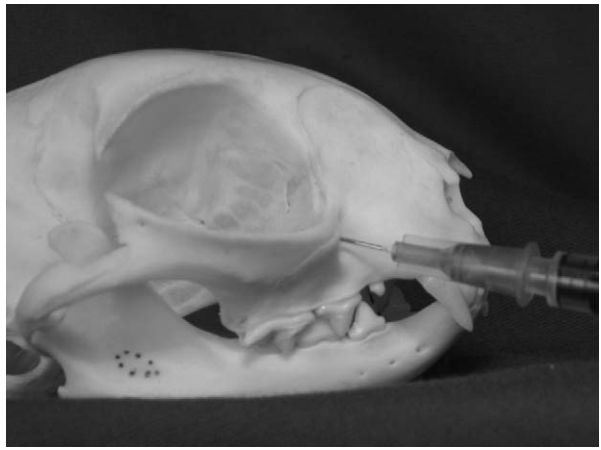

Fig. 3. Placement of an infraorbital nerve block.

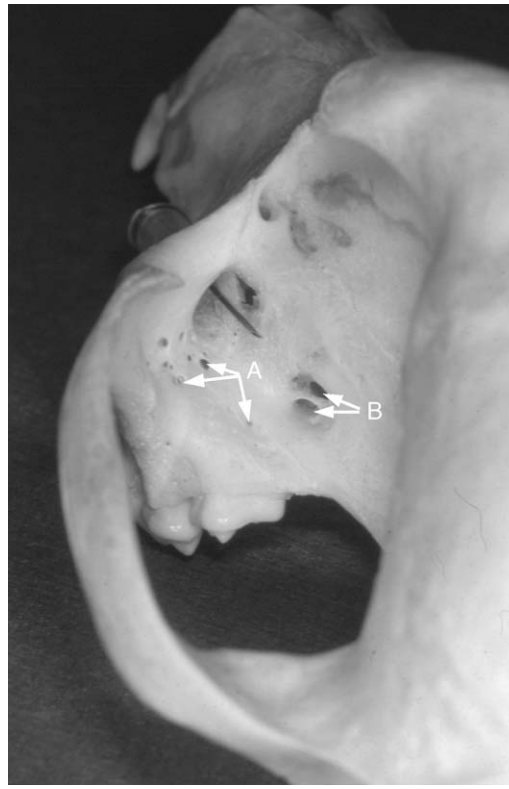

Fig. 4. Maxillary nerve block via the infraorbital foramen. The needle is placed deep into the infraorbital canal. The arrows next to A point to foramina leading to the distal premolars and molars. The arrows next to B point to the foramina for the major and minor palatine nerves.

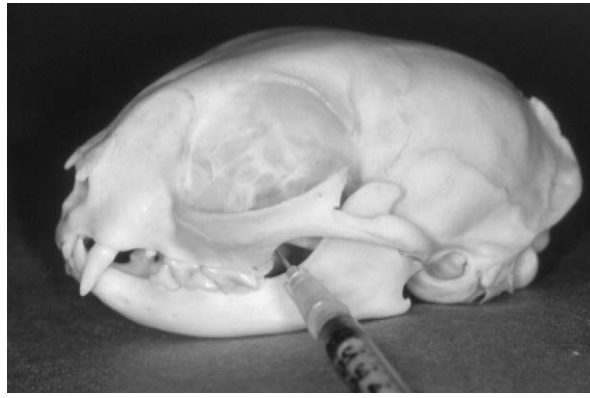

Fig. 5. Extraoral approach for a maxillary nerve block.

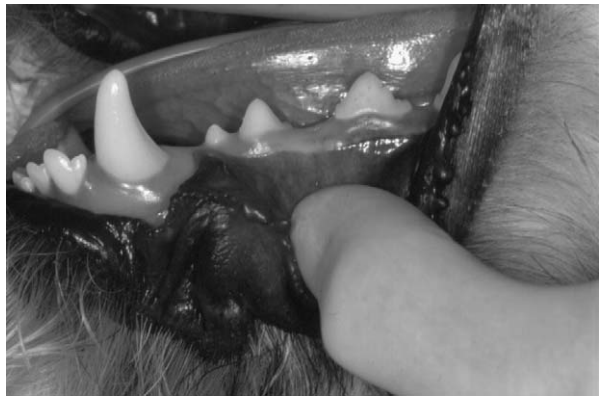

Fig. 6. Location of the middle mental foramen in the dog.

edges of the body of the mandible. The nerve is anesthetized before it enters the mandible and blocks all the soft tissues and dentition on that side of the mouth. The intraoral approach involves directing the syringe across the tongue from the opposite side of the mouth and placing the anesthetic agent in proximity to the foramen. In the extraoral approach, the needle is inserted through the skin at right angles to the ventral border of the mandible. The foramen is on a line drawn from the lateral canthus of the eye, through the midpoint of the zygomatic arch, to the ventral aspect of the mandible. In the dog, this contact point should be approximately 0.5 to 1.0 cm mesial to the angular process. With a finger inserted into the animal's mouth and palpating the foramen, the needle should be walked off the medial edge of the mandible and advanced dorsally until it can be felt in proximity to the foramen. Anesthetic agent is deposited around the nerve as it enters the foramen (see Figs. 1A and 2A; Fig. 8).

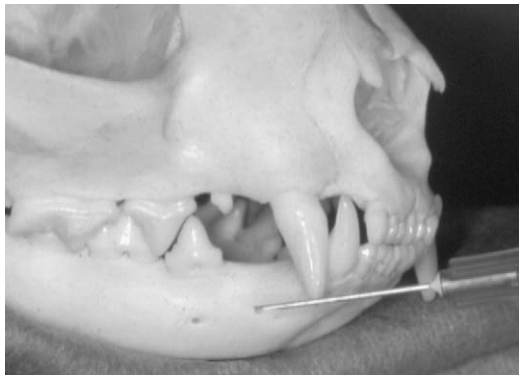

Fig. 7. A 25-gauge needle and the middle mental foramen in the cat. Iatrogenic trauma may occur from trying to place a mental nerve block in cats and small dogs.

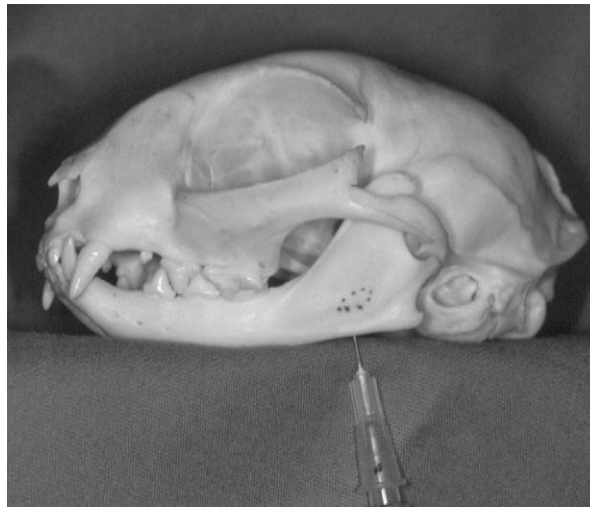

Fig. 8. Extraoral approach for a mandibular nerve block.

A less technique-sensitive method for blockading a limited area is with infiltration of that area. Anesthetic agent is placed under the soft tissues on the buccal and medial sides of the tooth adjacent to the bone. The agent diffuses into the bone and desensitizes the tooth. This technique only works for maxillary teeth, because the cortical bone is too dense in the mandible for infusion to occur. Intraosseous anesthesia does not have this latter limitation. A specially designed intraosseous needle (Dentsply Canada Ltd., Woodbridge, Ontario, Canada) is placed directly into the interproximal bone without predrilling, and agent is delivered. Analgesia is immediate, and an injection port stays available should additional anesthetic agent be needed.

A third method of desensitizing a single tooth uses intraligamentary anesthesia. Anesthetic agent at a maximum dose of 0.2 mL per root is injected into the periodontal ligament space using a special syringe. Onset of analgesia takes 10 to 15 minutes. This injection provides periodontal ligament, gingival, and apical afferent sensory nerve analgesia [7]. This technique does not work in the presence of infection or severe periodontal disease. For these cases, intraosseous anesthesia or a specific nerve block should be considered.

Opioids

Opioids are appropriate for controlling short-term pain. Pharmaceuticals with μ-agonist activity provide excellent analgesia for moderate to severe pain. Combined with nonsteroidal anti-inflammatory drugs (NSAIDs) or NMDA receptor antagonists, for example, opioids can also treat chronic or refractive conditions.

The response to narcotics is species dependent. The feline reaction is possibly the result of a different opiate receptor population. Dogs show central nervous system (CNS) depression, hypothermia, and miosis, whereas cats may develop CNS excitation, hyperthermia, and mydriasis. Cases of ''morphine mania'' are usually the result of giving excessive quantities of a pure μ-agonist to a nonpainful cat. Coadministration of a tranquilizer, such as acepromazine (PromAce, Fort Dodge Animal Health, Madison, NJ; 0.05 mg/kg administered subcutaneously or intramuscularly) [8] can reduce this side effect, as does giving smaller quantities of μ-opioid.

Commonly used narcotics are listed in the following sections, including their salient features.

Morphine

Morphine (Morphine; AstraZeneca, Wayne, PA) is the standard by which all opioids are measured, but it has variable intensity and duration of effect in the cat. Given intravenously, morphine causes histamine release and frequently causes vomiting if given to a nonpainful animal. Nausea does not occur if pain is present.

Methadone

Methadone (Methadone; AAIPharma, LLC, Wilmington, NC) is similar to morphine in duration and degree of analgesia but is less likely to induce vomiting. Methadone also affects NMDA receptors by means of a non-competitive mechanism [9]. It is a relatively expensive drug.

Oxymorphone

Oxymorphone (Numorphane; Endo Labs, Chadds Ford, PA) is also an expensive opioid and has recently been a victim to supply problems. It can be used intravenously without histamine release and can be absorbed across mucous membranes. This latter characteristic means that oxymorphone can be sent home for intranasal application at a dose of 0.05 to 0.1 mg/kg administered every 4 to 6 hours. Oxymorphone is not effectively antagonized by naloxone in cats; thus, overly narcotized patients are difficult to reverse.

Fentanyl

Fentanyl (Duragesic; Janssen Pharmaceuticals, Titusville, NJ) has a short duration when given systemically, usually lasting only 30 to 60 minutes. It is most useful as a constant rate infusion (CRI) or in a transdermal patch.

Transdermal patches are useful for control of significant pain of longer duration. Uptake of fentanyl through the skin varies between species, even within a species, but seems to be faster in cats [8]. The time required to reach steady-state plasma concentrations fluctuates, but comfort seems to begin between 6 and 12 hours after application, and analgesia can last for 4 to 5

days. Because of the variation in absorption, all patients with a transdermal patch should be observed for breakthrough pain. Animals weighing less than 2.5 kg may absorb too high a dose even from the smallest patch; thus, an alternative analgesic protocol should be considered.

The use of a fentanyl patch is off-label for a veterinary patient. If sending an animal home with an active patch, ensure that the pet is going to a "safe" household (eg, the clients are trustworthy, children and other pets do not have access to the patch).

Hydromorphone

Hydromorphone (Dilaudid; Abbott Laboratories, Abbot Park, IL) does not have a ceiling to its analgesic effects [10]. Animals demonstrating severe pain that is refractory to a single dose of hydromorphone may experience relief when the dose is increased. Analgesia can last for up to 6 hours [8]. Hydromorphone is not licensed for use in animals but is an economic alternative to oxymorphone. It often induces vomiting when given to a non-painful animal (ie, as premedication before surgery).

Butorphanol

Butorphanol is useful for mild to moderate pain. It has a ceiling effect for its analgesia, meaning that higher doses do not produce better analgesia. It is not appropriate for significant somatic pain [11], and some researchers question whether it is analgesic in animals at all [12].

Buprenorphine

Buprenorphine is a partial μ-agonist that is good for mild to moderate pain. It has a tremendous affinity for the μ-receptor and competitively inhibits other μ-agonists from binding [13]. Buprenorphine can be used to antagonize morphine or fentanyl without loss of analgesia but is difficult to reverse and may cause residual blockade even after it is no longer systemically active. Residual blockade may be a concern if buprenorphine is used after surgery while waiting for a fentanyl patch to take effect.

Buprenorphine is 100% bioavailable via the transmucosal route. Combined with an acceptable taste and a small dose volume (0.02 mg/kg = 0.066 mL/kg), these properties make it an excellent option for feline use. A 3-day transdermal [3] buprenorphine patch is available in human medicine, and a 7-day patch is under development.

Codeine

Historically, codeine has been more useful for home therapy than for in-hospital treatment. Codeine is metabolized to morphine, but cats can only convert less than 10% of a given dose, [14] and a rare animal may show excitation after treatment.

Codeine has an unpleasant taste and is only 60% bioavailable by the oral route [14]. Flavoring makes syrups more acceptable. A transdermal paste has been formulated that can be applied to the pinnae, which seems to be effective, is easier to use, and does not require oral manipulation, making it ideal for postdental patients.

Tramadol

Tramadol is a synthetic derivative of codeine and is one of the most useful drugs available to veterinarians for treating chronic and neuropathic pain. It is not technically an opioid; therefore, it is not controlled. Tramadol does cause μ-receptor stimulation, but it is also a monoamine (ie, serotonin) reuptake inhibitor. This inhibition enhances dorsal horn downregulation of pain impulses and produces mild antianxiety effects [13]. Tramadol has been used short term in cats, but the safety of long-term use is unknown. It should not be used with other tricyclic antidepressants (TCAs), selective serotonin reuptake inhibitors (SSRIs), or monoamine oxidase (MAO) inhibitors, and, [13,15] because it may lower seizure thresholds, caution is advised with epileptics.

Naloxone

Naloxone is a μ-antagonist used to correct overzealous opioid use. Cats do not react predictably to this agent [14,16]. It has a short duration of effect, which may necessitate retreatment.

α_2-Agonists

The α_2-agonists can be ideal adjuncts to general anesthetics. They provide sedation, analgesia, and muscle relaxation and significantly reduce the quantities of injectable and inhalant anesthetic agents needed to induce and maintain anesthesia. Xylazine, medetomidine, and romifidine are in this class. Studies have been completed with medetomidine, which, although off-label for use in cats, can work synergistically with opioids in the perioperative period [17–19]. Preoperative sedation and preemptive analgesia are achieved with drastically less volume than label recommendations. In the postoperative period, ultralow doses, alone or with opioids, can enhance and prolong the analgesic effects of the opioids, with limited effect on cardiovascular function. Synergy means the dose of opioid needed is also reduced, usually by half. Because of possible side effects, all α_2-agonist use should be limited to healthy animals. Atipamezole reverses the sedative and analgesic effects of medetomidine.

Nonsteroidal anti-inflammatory drugs

Opioids are effective before and after surgery when surgical inflammation has not fully developed. After the initial period of insult or in cases of

chronic disease, NSAIDs may prove to be advantageous for pain control via their combined anti-inflammatory and analgesic actions.

The principal mode of action of all NSAIDs is to block prostaglandin production by binding and inhibiting cyclooxygenase (COX). Although the result of this effect is mainly a reduction in inflammation and peripheral nociceptor sensitization, wind-up is also reduced or prevented. There is some evidence that certain NSAIDs, such as acetaminophen, have a central analgesic action [13], possibly against a new variant of COX enzyme [20]. Acetaminophen can be used in dogs as a first-line therapy when NSAIDs cannot be used.

NSAIDs should only be used in healthy, young, normotensive, normovolemic animals without evidence of gastric ulceration, bleeding diathesis, or compromised renal function. Ulcer prophylaxis can be used in a high-risk animal. Of interest, misoprostol, a synthetic prostaglandin analogue that prevents and helps to heal gastrointestinal ulceration caused by NSAIDs also enhances the anti-inflammatory and analgesic effects of NSAIDs [14]. Caution should be exercised when using NSAIDs in cats because of the drugs' prolonged half-life and the potential for toxicity. Few studies have been performed to examine the feline response to these compounds; thus, close monitoring should accompany long-term therapy. Before using an NSAID for cats, it should be checked for labeled use in this species.

N-methyl-D-aspartate receptor blockers

Blocking NMDA receptors impairs the wind-up phenomenon; therefore, acute and chronic pain is better managed [8]. Controlling central sensitization allows other analgesics to be more effective, but NMDA receptor antagonists also act to increase opioid receptor sensitivity, reduce opioid tolerance, and minimize rebound hyperalgesia (the phenomenon of markedly increased pain that occurs when an opioid wears off) [13].

Ketamine is the best-known NMDA receptor blocker. It is most effective as a CRI. The antiviral agent amantadine is also included in this category of agents [20]. It is most effective when used as an adjunctive therapy to an NSAID. A less frequently referenced NMDA receptor antagonist is dextromethorphan [21]. Commercial dextromethorphan products are only cost-effective in a smaller dog or cat. Compounding is necessary for use in a larger animal.

Analgesic adjuncts

Gabapentin

Gabapentin is an anticonvulsant with purported analgesic activity. Its mechanism of action is unclear; it may act on NMDA receptors [20], or it may inhibit postsynaptic neuron firing in general [13]. It has been used for

Table 2
Analgesic agents for use in dogs and cats

Drug	Dosages
Acetaminophen	C: Contraindicated D: 10–15 mg/kg PO q 8–12 h
Amantadine	C: 3 mg/kg PO SID D: 3–5 mg/kg PO SID or 1–2 mg/kg BID
Amitriptyline	C: 0.5–2.0 mg/kg PO SID D: 3–5 mg/kg PO SID
Bupivacaine	1–2 mg/kg q 6–8 h
Buprenorphine	C: 0.01–0.02 mg/kg SQ, IM, IV, sublingual q 4–6 h D: 0.005–0.02 mg/kg SQ, IM, IV q 4–8 h
Butorphanol	0.2–0.4 mg/kg SQ, IM, IV q 1–2 h 0.5–1 mg/kg PO q 4–8 h
Carprofen	C: 1–2 mg/kg SQ q 12–18 h D: 2 mg/kg PO BID or 4 mg/kg PO SID
Codeine	0.5–1.0 mg/kg PO TID
Deracoxib	D: 3–4 mg/kg PO SID × 7 days, then 1–2 mg/kg SID
Dextromethorphan	0.5–2 mg/kg PO TID–QID
Etodolac	C: Not recommended D: 5–15 mg/kg PO 24 h
Fentanyl	2–5 μg/kg/h transdermal C: Loading dose: 1–2 μg/kg IV, then CRI: 1–4 μg/kg/h IV D: Loading dose: 1–2 μg/kg IV, then CRI: 5–10 μg/kg/h IV
Gabapentin	C: 2–10 mg/kg PO SID–BID D: 3–10 mg/kg PO SID–BID
Hydromorphone	C: 0.02–0.1 mg/kg SQ, IM, IV q 4–6 h D: 0.05–0.2 mg/kg SQ, IM, IV q 4–6 h
Imipramine	C: 2.5–5.0 mg PO BID D: 1–2 mg/kg q 8–12 h
Ketamine	Loading dose: 0.2–0.5 mg/kg IV, then CRI: 2–10 μg/kg/min IV during surgery, then 2 μg/kg/min after surgery for up to 18 hours (this works out to 60 mg of ketamine in 1000 mL of LRS given at 2 mL/kg/h)
Ketoprofen	C: 2 mg/kg SQ once, then 1 mg/kg SID D: 2 mg/kg SQ, IM, IV once, then 1 mg/kg SID C: 2 mg/kg PO once, then 1 mg/kg SID, maximum 5 days D: 2 mg/kg PO once, then 1 mg/kg SID
Lidocaine	C: 2–6 mg/kg, maximum 2 mL total D: 2–6 mg/kg
Medetomidine	1.0 μg/kg, with equal volume of butorphanol, IV (this produce's heavy sedation and is not recommended if planning on going on to a GA) Before surgery with atropine + opiate C: 5–10 μg/kg IM D: 2–5 μg/kg IM After surgery alone C: 4–8 μg/kg IM D: 2–4 μg/kg IM

(*continued on next page*)

Table 2 (*continued*)

Drug	Dosages
	After surgery with opiate C: 2–4 μg/kg IM D: 1–2 μg/kg IM (After surgery, opiate is given at one half the dose used in premedication (eg, butorphanol at 0.2–0.4 mg/kg in premedication is 0.1–0.2 mg/kg after surgery)
Meloxicam	0.2 mg/kg SQ once C: 0.2 mg/kg PO SID × 1 day, then 0.1 mg/cat PO q 1–3 days D: 0.2 mg/kg PO SID × 1 day, then 0.1 mg/kg q 24 h
Meperidine	C: 5–10 mg/kg IM q 2–3 h D: 3–5 mg/kg IM q 2–3 h
Methadone	C: 0.05–0.5 mg/kg SQ, IM, IV q 4–6 h D: 0.1–1.0 mg/kg SQ, IM, IV q 4–6 h
Misoprostol	C: 1–3 μg/kg PO q 8 h D: 1–5 μg/kg PO q 8 h
Morphine	C: 0.1–0.5 mg/kg SQ, IM q 4–6 h D: 0.5–1.0 mg/kg SQ, IM q 4–6 h
	C: 0.02–0.05 mg/kg IV q 1–2 h D: 0.05–0.1 mg/kg IV q 1–2 h
Morphine oral	C: 0.2–0.5 TID–QID D: 0.5–2.0 TID–QID
Morphine oral (sustained release)	C: Not available D: 0.5–1.0 PO BID–TID
Naloxone	0.04 mg/kg diluted with 10 mL saline, give 1 mL/min IV until symptoms resolve, then q 45–180 min
Oxymorphone	C: 0.05–0.1 mg/kg SQ, IM q 2–4 h D: 0.05–0.2 mg/kg SQ, IM q 2–4 h
	C: 0.03 mg/kg IV q 45–60 min D: 0.06 mg/kg IV q 45–60 min
Piroxicam	C: 1 mg/cat PO SID maximum 7 days[a] D: 0.3 mg/kg PO q 48 h
Tolfenamic acid	4 mg/kg SQ, IM once C: 4 mg/kg PO SID for 3–5 days D: 4 mg/kg PO SID for 5 consecutive days per week
Tramadol	C: 2–4 mg/kg PO BID D: 1–2 mg/kg PO BID to QID, maximum 10 mg/kg/d
Vedaprofen	D: 0.5 mg/kg PO 24 h, maximum 28 days

All doses and labelling for use should be verified by the practitioner before use.

Abbreviations: BID, twice daily; C, cat; CRI, continuous rate infusion; D, dog; GA, general anesthetic; h, hours; IM, intramuscular; IV, intravenous; LRS, lactated Ringer's solution; min, minutes; PO, orally; q, every; QID, four times daily; SID, once daily; SQ, subcutaneous; TID, three times daily.

[a] After compounding, drug is only stable for 10 days.

chronic and neuropathic pain. It is an expensive drug; therefore, it is not suggested as a first choice for chronic pain [8,13]. Once pain is controlled, the patient should be weaned off the drug slowly [8].

Tricyclic antidepressants

Anxiety lowers pain thresholds [16], which is why TCAs have been used in human beings and animals as adjuncts to other analgesics (especially opioids) for chronic pain [3]. TCAs act to inhibit serotonin and norepinephrine reuptake, part of the biochemistry of wind-up. They may have other analgesic effects as well, including possible actions at opioid receptors and on nerve transmission [13]. Amitriptyline and imipramine are the most commonly used drugs in this class.

Other adjuncts

Acupuncture, physiotherapy, and nutraceutical agents may also be used to provide comfort, especially in chronic pain cases.

Summary

It is beneficial to provide local anesthetic nerve blocks and multimodal analgesia. Nerve blocks arrest a pain impulse before it is formed. The most commonly used blocks for oral and dental surgery are the infraorbital, maxillary, mental, and mandibular blocks. Source of pain, duration, and subject species, for example, can all be factors in determining the therapeutics used for acute and chronic pain control. Opioids, NSAIDs, NMDA receptor blockers, TCAs, and other adjuncts can all be used (Table 2).

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