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Management of Acute Postoperative Pain in Dogs and Cats

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Alleviation of animal pain is a central value of the veterinary profession as written in the Veterinarian's Oath - '... I solemnly swear to use my scientific knowledge and skills for the benefit of society through the protection of animal health, the relief of animal suffering, ...' In addition, the AVMA's 1987 Panel Report on the *Colloquium on Recognition and Alleviation of Animal Pain and Stress* reminds veterinarians of their responsibility to provide effective management of pain in their patients.⁸ Understanding and management of pain in animals is a challenging and often subjective task that faces practitioners. Factors that contribute to an ambiguous approach for managing pain in animals include, to what extent should pain be treated in an animal, inability of animals to verbally communicate, variation in signs of pain displayed by an animal, complex mechanisms and pathways involved in pain recognition, the variety of analgesic options available for therapy and when to use them, and failure to stay informed of current concepts in management of animal pain. The intent of the following article is to convey some of the recent information and its potential application towards management of acute postoperative pain in animals. Understanding concepts related to mechanisms of pain recognition, assessment of pain, preemptive considerations, and management options, contribute to effective management of acute postoperative pain in dogs and cats.

Mechanisms of Pain Recognition

Pain is a perception and is a result of cortical processing that began as activation of receptors

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by stimuli that are actually or potentially damaging to tissue. This phenomena is referred to as nociception.⁷ Signals generated by noxious stimulation of specific nociceptors are transmitted, via nociceptive afferent nerves, to the central nervous system (CNS) provoking local reflexes in muscles, autonomic reflexes, endocrine responses (pituitary and adrenal effects), and continue as ascending afferent information to the brain stem, thalamus, and cerebral cortex for sensory processing. Processing involves a sensory-discrimination component and an affective-motivational component.¹⁰ Evaluation of sensory input as pain includes the discriminatory component that determines location, intensity, duration, and quality of pain, and the affective-motivational component leading to reactions such as anxiety, suffering, and depression.

Cutaneous nociceptors are the primary components of peripheral mechanisms of pain and can be divided into two broad categories: A δ nociceptors and C-polymodal nociceptors.⁷ A δ nociceptors are referred to as heat-mechanoreceptors and their corresponding A δ fibers (smallest myelinated fibers) have been associated with first, fast, or initial pain which is described as the sharp, stabbing, well-localized quality of superficial pain.¹⁶ These nociceptors are silent in the absence of stimulation although a background of activity can develop if they are stimulated repeatedly.³

C-polymodal nociceptors respond to strong mechanical stimulation, heat, and to irritant chemicals and their corresponding fibers (nonmyelinated) are associated with the second, slow, or delayed pain that is described as a dull, burning, diffusely located quality of superficial pain.¹⁶ Receptive fields of C-polymodal receptors respond to stimuli similar to those of A δ nociceptors. In addition, C-polymodal receptors are excited by endogenously occurring chemicals such as H⁺ ions, serotonin, histamine, bradykinin, prostaglandins, and leukotrienes, which have a causal role in inducing pain after inflammation, trauma, bone tumors, and chronic ischemia.²⁰ Injury to tissue, whether before or

as a result of surgery, causes release of these chemicals and C-polymodal nociceptors become sensitive to stimuli that would not normally have caused them to fire, or they fire spontaneously. This is referred to as peripheral sensitization. Conditioning stimulus to C-polymodal pathways can also cause a change in spinal processing of sensory signals and is referred to as central sensitization. Another neurogenic factor that may be a causative factor in inflammation and pain, or modulate nociceptive spinal pathways, is substance P which is contained in some afferent nerve fibers.⁹ Excitation of nociceptors containing substance P may result in its release which leads to vasodilatation and local edema as a result of increased capillary permeability. Repeated C-polymodal fiber stimulation also results in release of substance P from afferent terminals causing prolonged facilitation of the spinal cord.

Central mechanisms of pain are complex and include neural pathways in the dorsal horn of the spinal cord and medulla oblongata, ascending spinal tracts, thalamus, and cerebral cortex.¹⁷ Nociceptive primary afferents form synaptic junctions with nociceptive interneurons and nociceptive ascending tracts. Most central processing of peripheral nociceptive information occurs in this area of the dorsal horn. Nociceptive afferent fibers have synaptic terminals with interneurons, ascending tract neurons, and motor neurons (as in reflex arcs). Interneurons interact with ascending tract neurons and motoneurons. Some of these connections are excitatory and others are inhibitory that involve mediators such as Gamma-aminobutyric acid, or enkephalin (an opioid peptide). Increased stimulation of cutaneous or deep muscle C-polymodal nociceptors changes spinal processing of sensory information in two ways.¹⁸ First, dorsal horn neurons begin to respond to cutaneous stimuli in regions beyond the original border (receptive-field expansion), and second, the magnitude of responses to stimuli increases and previous nociceptive-selective neurons begin to respond to innocuous cutaneous input (receptive-field expansion in modality domain). Thus, any given stimulus drives more spinal neurons and weak stimuli drive pain signalling spinal neurons. This is central sensitization. Induction and maintenance of central sensitization is dependent on *N*-methyl-D-aspartate (NMDA) subclass of receptors for the excitatory activation of nociceptive spinal neurons.¹⁹ Postoperative pain has been thought to be due

to peripherally sensitized nociceptors activated by weak stimuli when a patient moves, but in addition, surgery strongly stimulates C-polymodal input triggering central sensitization. Central sensitization occurs in an anesthetized patient and, after recovery, weak A δ heat-mechanoreceptor input from the periphery results in hyperalgesia. Blocking this change in spinal processing, induced by surgery and increased C-polymodal input, is referred to as preemptive analgesia.

Assessment of Pain

Assessment of pain begins with a working definition of pain. Pain in animals is an aversive sensory and emotional experience (a perception), which elicits protective motor actions, results in learned avoidance, and may modify species-specific traits of behavior, including social behavior.⁷ Pain is classified as a chronic pain if it exists more than six months and is thought to be a separate syndrome from acute pain.¹⁴ Post-operative pain is a form of acute pain. Traditionally, qualities of acute pain are related to the site of origin of the pain, where somatic and visceral pain form the two major divisions of pain.¹⁴ Somatic pain is further subdivided into superficial pain and deep pain. Subdivisions of superficial pain, referred to as first and second, correspond to A δ and C-polymodal nociceptive mechanisms respectively.

Attempts at assessing pain are often based on an anthropomorphic approach and analgesics are given based on clinical signs. Unfortunately, this can lead to a wide variation in pain management as personal attitudes, with respect to pain in animals, can range from the notion that animals do not feel noxious stimuli as much as humans to the tendency of analgesic overdosing in post-operative animals. There is a tendency for many breeds of dogs and most cats to be stoic which should not be confused with lack of nociceptive mechanisms. Animals that are weak, systemically ill, or debilitated may be unable to respond to noxious stimuli.¹³ Overreaction to noxious stimuli can occur in young animals, males, or frightened animals.

Pain perception in animals is indicated by voluntary actions and physiologic responses.⁵ Response to pain exhibited by dogs upon recovery from surgery may include vocalization (whimpers, whining, or cries), trembling, rolling, thrashing, turning its head toward the site of noxious stimuli, unusual body posture, lame-

ness, and/or restlessness. Animals may vocalize, attempt escape, or become aggressive when the affected region is approached or manipulated. Dogs or cats often attempt to guard or protect the affected area, or may self-mutilate the area by biting, scratching, or licking. Animals may exhibit prolonged recumbency, limb disuse, slow/stiff or reduced movement, decreased appetite, or may appear anxious or nervous. Abdominal pain in animals is indicated by splinting of abdominal muscles and animals with chest pain tend to have a shallow breathing pattern and abduct their elbows. If an animal has been painful over several days, it may not respond to pain and may become quiet and withdrawn. The contribution of pain to shock or masking of pain in animals with altered states of consciousness, especially after surgery or acute trauma, could also be considered. Physiologic responses to pain may include tachypnea (panting), sinus tachycardia, ventricular premature contractions, atrial premature contractions, mydriasis, ptialism, hyperglycemia, hypotension or hypertension, and pallor. The inherent difficulty underlying analgesic therapy based on clinical signs is the requirement of in depth observation of behavioral responses in each patient, and familiarity with the species or even the specific breeds.

Attempts to minimize the subjectivity involved in assessing pain of post-operative animals has led to an Expected Postoperative Pain Response approach.⁵ Generalizations were made based on type of surgery and the corresponding number of nociceptors that would be stimulated to generate an expected level of pain response (Table 1). Procedures such as amputations (transection of large muscle masses and nerves), cervical spine surgery, ophthalmologic surgery, and sternal thoracotomies have a high expected level of postoperative pain response. The expected pain level can be extrapolated to nonsurgical trauma and systemic disease such as meningitis, pleuritis, necrotizing pancreatitis, dissolving saddle thrombi, osteosarcomas, or metabolic bone disease. Given the expected level of postoperative pain response, analgesic therapy is planned and implemented accordingly. Opioid analgesics are indicated for treating postoperative pain and the question becomes-When to initiate treatment? Obviously, in the humane sense, before noxious stimuli and before an animal shows signs of pain. Traditionally, postoperative analgesics are given when surgery is nearing completion or when an animal

shows signs of recovery (palpebral reflex or initiation of swallowing). Although, a less obvious time to administer analgesics would be prior to central sensitization to nociceptive stimuli.

Preemptive Analgesia Considerations

The concept of controlling disease in animals by vaccination and preventing the development of disease is a common practice in veterinary medicine. The same logic may be applied to management of postoperative pain in animals. Central sensitization to pain can result from minimal stimulation of C-polymodal nociceptive pathways, via injury and/or surgical procedures. In an attempt to prevent central sensitization to pain, noxious stimuli should be stopped at the site of injury or blocked at the location of central transmission, which is at NMDA receptors of nociceptive spinal neurons. One approach would be to apply regional analgesia, in the form of nerve blocks or epidural opiates, to block input associated with surgery. Although, sensitization would occur from the surgical field as soon as the analgesia subsided. This would imply a need for a complete regional block from the time of initial incision to wound healing, which is obviously impractical. Numerous clinical trials have been performed to determine the optimal application of preemptive regional analgesia.¹¹ Regional analgesic blocks applied before surgery, or at the completion of surgery with concurrent use of opioids preoperatively and intraoperatively, reduces response to postoperative pain and improves recovery rates. Preventing central sensitization to pain within the first 24 hours after surgery may be the most beneficial in reducing postoperative pain. Preemptive analgesia is one component within an overall plan for postoperative pain management.

Management Options

Clinical management of acute postoperative pain includes minimizing anxiety and stress, use of premedication to reduce anxiety and peripheral and central sensitization, preemptive local anesthetics and epidural opioids, and use of postoperative opioid analgesics.¹²

Anxiety, fear, helplessness, and sleep deprivation all tend to contribute to stress and decrease tolerance to pain. Each of these factors occurs to some extent in the unfamiliar

environment of a veterinary clinic. Any attempt to minimize anxiety and stress prior to surgery is beneficial. Kindness, compassion, and pharmacologic agents should be considered. Pharmacologic agents, or adjuvant analgesics, commonly used to reduce anxiety include xylazine, diazepam, and acepromazine.⁶ Xylazine is a sedative-analgesic that acts on central and peripheral Alpha-2 receptors and is often used in neuroleptanalgesic protocols. Diazepam is a minor tranquilizer that acts as a muscle relaxant with minimal respiratory and cardiovascular effects. Acepromazine, a phenothiazine, can also be used as a sedative but it can produce hypotension. Acepromazine and diazepam can be given to animals before they leave home so peak effect will be reached before gas analgesia is produced.

In addition to reducing anxiety, premedications that inhibit centralization of pain response are also beneficial. Opioids and Alpha-agonists inhibit C-polymodal input at the spinal cord level. Neuroleptanalgesic combinations also work well in healthy patients. Frequently used combinations include acepromazine and one of the opioids meperidine, butorphanol, morphine, or oxymorphone. The choice of opioid to use with acepromazine is based on duration of action matched to duration of procedure. Meperidine, fentanyl and butorphanol have short durations of action (meperidine approximately 45 minutes) and can be used for procedures lasting less than 2 hours. Morphine and oxymorphone are appropriate for procedures lasting 3 to 4 hours. Opioids should be used at lower doses in cats and butorphanol has a longer duration of action in the cat.

Local anesthetics are used to block neuronal pathways from the surgical site and are more effective if applied prior to surgery. Two procedures used are the intravenous regional technique and epidural application of local anesthetics. The objective of these blocks include 1) preemptive analgesic effects on central sensitization, 2) reduce stress response associated with the surgery, and 3) to decrease risk to anesthetized patients. Local regional blocks or epidurals using bupivacaine provides long acting anesthesia for orthopedic cases involving trauma and fractures especially in the pelvic limbs, for incipient shock, cases involving highly altered homeostasis, or when organ function could be further compromised by pain.¹ Intercostal nerve blocks are used in thoracotomy

procedures and improve postoperative ventilation by providing analgesia without respiratory depression.⁴ Preemptive analgesia can also be accomplished using morphine or buprenorphine in the epidural space prior to surgery.² The most frequent indication has been orthopedic surgery involving the pelvis and/or hind limbs. It takes 20 to 60 minutes to reach peak effect and lasts for up to 24 hours because morphine is cleared slowly due to its relative hydrophilic property. Results from clinical use include 1) reduction of inhalation anesthetics and more stable intraoperative parameters, 2) no pain response for several hours and a reduced requirement for postoperative analgesics, 3) anterior analgesia is not as intense as posterior analgesia, and 4) safe without complications (respiratory depression not noted).

Opiate receptors are located primarily in the spinal cord and brain, but are also distributed in muscle, and the gastrointestinal tract.⁶ Interaction of opioids with μ -, and κ -receptors, provides relief from pain. Binding at μ -receptors produces supraspinal analgesia, euphoria, respiratory depression, mydriasis and/or miosis, hypothermia, bradycardia, sedation, and physical dependence. Binding at the κ -receptor results in spinal analgesia, miosis, moderate sedation, mild respiratory depression, and dysphoria. Agonists that are used in veterinary medicine include morphine, oxymorphone, meperidine, codeine, and fentanyl. These agonists have the greatest affinity for, and activation of, μ -receptors, but they also activate κ -receptors. Butorphanol and buprenorphine are agonist-antagonist opioids and result in limited activation of μ -receptors, but have high affinity for, and activation of, κ -receptors. Even though agonist-antagonists do not activate μ -receptors, they have a greater affinity for μ -receptors than agonists, thus they are able to compete with agonists for binding at μ -receptors. Agonist-antagonists also demonstrate a ceiling effect, that is, increasing the dose does not increase analgesia or respiratory depression. Frequently used opioids include butorphanol, oxymorphone, and buprenorphine. Butorphanol provides control for mild to moderate pain and its duration of analgesia is one to four hours. Moderate to high levels of pain can be relieved by use of oxymorphone which has a duration of analgesia of two to four hours. Buprenorphine is a long acting analgesic (due to slow dissociation from μ -receptors) and is effective for control of moderate to high levels of pain. Buprenorphine

is not reversible by use of naloxone. Use of these opioid analgesics should be based on the expected postoperative pain response. Additional postoperative administration of opioid analgesics could be indicated if physiologic parameters are altered due to pain. Another possibility for postoperative control of pain response may include use of transdermally applied fentanyl.¹⁵ Fentanyl passively absorbs across the skin and can be used in permeable membrane patches to maintain blood levels for up to 3 days through controlled sustained release.

Summary

Postoperative pain in dogs and cats is one aspect of animal suffering that can not always be avoided, but is to be minimized to the best of a veterinarian's ability. Unfortunately, the approach to management of postoperative pain is not particularly straight forward and depends on an individual's knowledge and clinical judgement. Understanding the nociceptive mechanism of C-polymodal afferent input and development of central sensitization supports a preemptive approach to management of postoperative pain instead of administering analgesics based on signs of pain. The type and duration of surgical procedure or injury, and expected postoperative level of pain should be considered when assessing the potential for pain and when developing a management protocol. Compassion and possible use of adjuvant analgesics will reduce anxiety and stress associated with a decreased tolerance to pain. Preemptive analgesic options used as premedication include opioids, neuroleptanalgesic combinations, analgesic epidurals, and anesthetic regional blocks. Intraoperative and postoperative opioid analgesics can be used as the final component in an overall management plan. In light of all management options available for prevention of animal pain, success at minimizing acute postoperative pain will not be accomplished if veterinarians do not remain informed of current concepts relating to the alleviation of acute pain in dogs and cats. The decision to read this article was a good beginning.

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Table 1
Expected Postoperative Pain Response

Surgery	Expected Pain Level
Amputatin	High (transection of large muscle masses and nerves)
Cervical spine	High
Ophthalmological	High
Thoracotomy	High (sternal); moderate to high (lateral)
Anorectal	Moderate to high
Head, ear, throat, dental	Moderate to high
Orthopedic	Moderate to high (upper axial segments, e.g., shoulder/humerus and hip/femur, are very painful)
Lumbar and thoracic spine	Moderate
Celiotomy	Mild to high (varies with duration of procedures associated with major pathological changes)