How I Use (and Dose) Essential Medications for Chronic Pain
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Chronic pain
Management
Peripheral sensitization
Release of compounds by noxious stimuli can lead to lowering of nociceptor threshold; tissue inflammation = “sensitizing soup”:
Histamine, HT-5, BK, K+ions, PG, Cytokines, etc = reduced nociceptor thresholds = primary hyperalgesia

Hyperalgesia
Central sensitization
- nociceptive neurons in the dorsal horn of the spinal cord become sensitized by peripheral tissue damage or inflammation
- Dorsal horn neurons become hyper-responsive; sending more signals to the brain
- cells that fire together, wire together
- activation of NMDA receptors; reduced endogenous opioids levels; reduced serotonin level; expanded receptor fields
  (pain in neighboring areas not subjected to injury, or secondary hyperalgesia)

Maladaptive pain
Pain that serves no useful purpose, and significantly impairs the quality of life of the patient.

Consequences of chronic pain
Hindrances to chronic pain management
Pain scales
Helsinki Chronic Pain Index; Canine Brief Pain Index; Cincinnati Orthopedic Disability Index
Glasgow Composite Pain Scale; AAHA
NSAID’s -- remain the mainstay of therapy for chronically painful patients.
NSAID Precautions
- Use only 1 NSAID at a time
- Never combine NSAIDS with glucocorticoids
  - Gastric Ulceration
  - Change to alternative NSAID if initial response is not favorable

Steroids
Decrease pain by decreasing inflammation
Intra-articular: Triamcinolone < 25 KG = 5 mg > 25 KG = 10 mg
Stop NSAIDs for 4 days pre & post injection.
Protective effects of corticosteroids on cartilage lesions and osteophyte formation in the Pond-Nuki dog model of osteoarthritis.
In vivo protective effects of prophylactic treatment with tiaprofenic acid or intraarticular corticosteroids on osteoarthritic lesions in the experimental dog model. J Rheumatol Suppl. February 1991;27(0):127-30. J P Pelletier1; J Martel-Pelletier
What do we do with those patients that stop becoming responsive to our NSAID therapy?
Do we switch to a different NSAID? NO!
Re-evaluate the patient and the treatment strategy. Start a multi-modal approach
Central Sensitization → Allodynia
NMDA receptor: N-methyl-D-aspartate
- NMDA receptors are “silent” until persistent or large scale release of glutamate
- by blocking the activation of these receptors, a reduction in CNS hyperresponsiveness can be achieved, allowing other analgesics to function more effectively
- adjunctive drug to improve the control of pain
- act to increase opioid receptor sensitivity
- Constant Rate Infusion: Opioid: Morphine or Hydromorphone or Fentanyl with Lidocaine/Ketamine
- Use for 48 – 72 hours in severe chronic pain patients.
Opioid
The most powerful analgesics available, with actions at peripheral, spinal and supraspinal levels.

Main advantage of giving opioids as a CRI is the avoidance of peaks and valleys seen with intermittent injections. A lower dose of opioid can be used, which can reduce the unwanted side effects.

Morphine
Loading dose 0.25 mg/kg IV followed by 2 – 6 ug/kg/minute

Hydromorphone
Loading dose 0.05 mg/kg IV followed by 0.4 - 1.2 ug/kg/minute

Fentanyl
Loading dose 0.002 mg/kg IV followed by 0.02 – 0.08 ug/kg/minute

Lidocaine
- Local anesthetic that provides excellent systemic analgesia.
- Dosage: Loading dose 2 mg/kg IV followed by 20–50 µg/kg/minute.
- IV lidocaine is extremely short acting and can be discontinued without residual effect.
- Typically lidocaine is not recommended for use in cats due to potential for cardiotoxic effects.

Ketamine
- Dissociative anesthetic and an N-methyl-D-aspartate (NMDA) antagonist.
- Dosage: Loading dose 0.5 mg/kg IV followed by 2 - 10 ug/kg/min
- Always be given in combination with an opioid.

Amantadine

Gabapentin

Amitriptyline

Acetaminophen
- 10 – 15 mg/kg q 8 – 12 hours
- CAUTION in hepatic or renal disease.
- Break through pain
- Can use with corticosteroids or NSAIDs
- Do not use in cats
- Helpful to bridge from one NSAID to another

Tramadol
- Dual mode of action: monoamine reuptake inhibition and mu agonist – NOT DOGS!
- controlled substance → august 18, 2014
- Combining tramadol with other analgesics (NSAID’s, mu agonists) further enhances tramadol’s efficacy, producing a multimodal pain relieving action.

Non-pharmacologic therapies
- Rehabilitation Therapy: Joints need to stay in motion
- Acupuncture: Pain management – endogenous opioids “homeostasis”
- Electrical nerve stimulation procedures : Gate Theory: inhibit pain signals in dorsal horn

Laser therapy
- cartilage stimulation
- fibroblast production
- enhancement of immune cells
- increases the vascularity of healing tissue
- may help with Myofascial Pain

Pulsed magnetic field therapy
- Enhanced cartilage repair
- Increased collagen synthesis of tendon fibroblasts
- Cell proliferation
- Synthesis of glycosaminoglycans by growth plate chondrocytes
- Decrease production of inflammatory cytokines

Superficial heat
- Heat the skin and subcutaneous tissues to a depth of 1-2 cm.
- The tissue is usually heated to 40 to 45C for 15 to 20 minutes.
- Increases blood flow to the area, promotes tissue extensibility, decreases pain, muscle spasm and joint stiffness, and causes general relaxation.
- Heat is contraindicated if swelling or edema are present.