

Clinical Decision Making in Emergency Pain Management

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Overview

- Scope of the problem
- Mechanisms of pain
- Management options
- Future directions: Is there a need to change practice

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Key Learning Points

- Management of pain must be placed in the context of the clinical presentation
 - Acute vs chronic; nociceptive vs neuropathic
 - Underlying mechanism of pain impacts approach to managing the pain
- Treatment should not be delayed pending a diagnosis
- IV titration is generally the preferred approach for severe pain
 - Treat early, front-load, around the clock
 - Acute management must be linked to the continuum of care
- Opioids are not always best and NSAIDs are not benign
- Anxiolysis plays an important role in the pain response

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Background

- Many physicians do not understand pain and its management
 - Many patients come to the ED out of desperation
- Pain is the most common reason people come to the ED
 - Accounts for 70% of ED visits
 - Children and the elderly are commonly undermedicated
- Pathways
 - Nociceptive: activation of primary peripheral pain receptors (A-delta and C fibers)
 - Neuropathic: aberrant signal processing in the peripheral or central nervous system

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Myths

- Fear of adverse reactions
 - Rare and generally preventable
- Fear of masking critical clinical findings
 - Questionable and unlikely if judgement used
- Fear of inducing addiction
 - Rate of 1/3,000 pts in Boston study
- Patients will request pain medication if they need it
 - 70% of pts will not request Tx despite pain
- IM treatment saves time and money
- You can assess severity of pain by looking at the patient and the vital signs

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Pain Treatment Options

- Eliminate mechanical and environmental factors
- Block opiate receptors
- Block inflammatory mediators
- Block transmission to the CNS: local anesthetics
- Modulate central 5-HT pathways
- Modulate the "close gates" at dorsal horn: TENS, acupuncture
- Decrease anxiety
- Maximize placebo effect

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Delivery systems

- IV
- IM
- IN
- TD
- PO
- PR
- PS

Analgesics

- Acetaminophen: no antiplatelet effect, no anti-inflammatory effect; acts in CNS
- NSAIDS
 - Inhibit prostaglandin synthesis by interfering with cyclooxygenase (COX) enzymes
 - Cause platelet dysfunctions
 - Can precipitate renal failure
 - Increase risk of GI bleeding
 - COX-2 agents preferentially inhibit the COX-2 enzyme that is induced by inflammatory stimuli and is responsible for the activation and sensitization of nociceptors

Is Ketorolac Contraindicated in Perioperative or Trauma Patients?

- Toradol is contraindicated as prophylactic analgesic before any major surgery, and intraoperatively whenever hemostasis is critical¹
 - Does have significant antiplatelet effects in clinical trials²
- Large case-control study did not show increased bleeding when given peri-op to surgical patients³

¹Physicians' Desk Reference (PDR®), ed. 56, Montvale, NJ: Medical Economics Co. 2002.

²Novack RJ, et al. *Clin Drug Invest*. 2001;21:465-476.

³Strom BL, et al. *JAMA*. 1996;275:376-382.

NSAIDs in Perspective

- No NSAID has been proven significantly more efficacious than another, when given in equivalent doses
 - Select agents based on toxicity profiles?
 - Side-effect rates generally parallel half-life profiles
- Pt. response can vary between agents
 - Multiple categories of agents
- No difference in efficacy by mode of administration

Opioids

- Agonists:
 - Rule of ten
 - 0.1mg fentanyl (Duragesic)
 - 1 mg hydromorphone (Dilaudid)
 - 10 mg morphine
 - 100 mg meperidine (Demoral)
 - Codeine (metabolized to morphine / high nausea)
 - Methadone
 - Oxycodone (Oxycontin)
 - Oxymorphone (Numorphan)
- Agonists – Antagonists
 - High dysphoria rates)
 - Ceiling analgesia and respiratory depression
 - Buprenorphine (Buprenex)
 - Butorphanol (Stadol)
 - Nalbuphine (Nubain)
 - Pentazocine (Talwin)
- Other
 - Tramadol (Ultram)
 - Weak binding to the opiate receptor
 - Inhibits reuptake of both NE and 5-HT

Opioids: Meperidine (Demerol)

- Many EDs no longer stock it
 - Metabolism prolonged in renal or hepatic disease
 - Metabolite (normeperidine) is a CNS toxin
 - Can induce the Serotonin Syndrome
- Highest rate of associated euphoria
 - Problematic pts often request it

Opioids: New strategies

- Less meperidine and morphine
- Early, rapid control with fentanyl
 - Titrate IV
 - Limit total dose
- Maintenance with hydromorphone
 - Start 5 -30 minutes later
 - Well tolerated
 - No maximum dose

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Pain Therapy: Point Injections

- “Trigger” or other point injections may represent an attractive and viable option in selected patients
 - Lower cervical injections for headache relief.

Mellick GA, Mellick LB. Headache 2001.41(10): 992

- Pericranial injection of local anesthetics in the ED management of resistant headaches

Brofeldt, Panacek. Acad Emer Med. 1998.

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Pain Therapy: Other Options

- Patient controlled analgesia (PCA)
- Nitrous oxide
- Moderate procedural sedation
- Deep procedural sedation

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Pain Therapy: Anxiolysis

- Catecholamines and other stress responses play an important role in the experience of pain
- Anxiolytics can have independent benefits, as well as decreasing total opioid requirements

Do not underestimate the benefits of physician reassurance

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Centrally acting agents

- 5HT receptor modulators
 - Phenothiazine
 - Triptans
- Tricyclics
- Carbamazepine
- Gabapentin
- Valproic acid

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Future directions

- Improve physician understanding of mechanisms of pain
 - Improve physician / patient communication
- Improve strategies for choosing the right intervention for the right patient
- Well designed comparative clinical trials
- Improve analgesic delivery systems
- Improve strategies for providing a continuum of pain management after discharge from the ED

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Conclusions: Key Learning Points

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