PAIN & ANALGESIA

POWH JMO TEACHING 13/4/21

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PAIN

What is it?

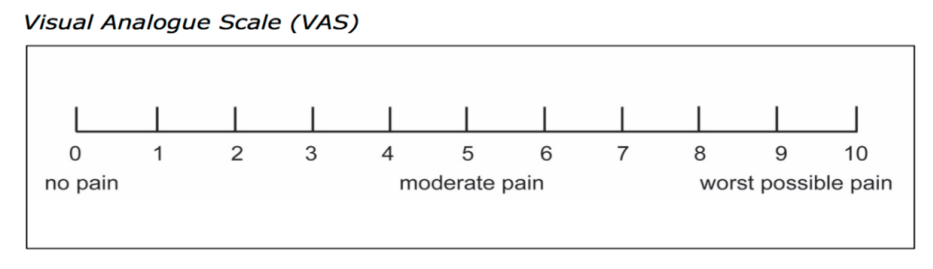
'Pain is an unpleasant sensory and emotional experience associated with potential tissue damage or described in terms of such damage' IASP

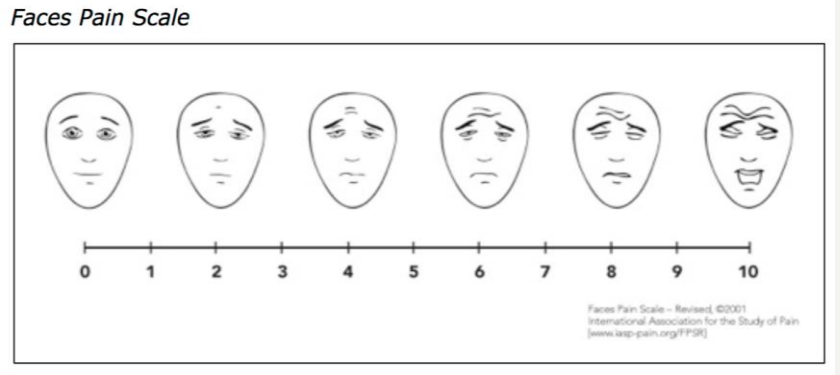
OR

…***'Doctor it hurts'***

PAIN

- **? Severity**
  - Verbal rating Scale
    - Mild / Moderate / Severe
  - Visual Analogue Scale / Numerical Scale
    - Score of 0 = no pain
    - Score of 10 = worst possible pain
  - Faces Pain Scale
  - Functional activity score
    - No / mild / severe limitation to perform an activity





Look at your patient… if they are sleeping, they do not have a pain score of 8.

PAIN

Adverse effects of acute pain:

- **CVS**
  - HTN, tachycardia, increased myocardial oxygen demand (increased risk of ischaemia) secondary to sympathetic overactivity
- **Resp**
  - Decreased lung volumes secondary to diaphragmatic splinting & weakened cough
    - Atelectasis, sputum retention -> pneumonia
    - Hypoxaemia -> widespread end-organ dysfunction
- **GI**
  - N&V
  - Impaired GI motility & gastric emptying
- **Genito-urinary**
  - Urinary retention
- **Endocrine / metabolic**
  - Increased levels of catecholamines, cortisol, aldosterone, growth hormone, ADH, glucagon and insulin
    - -> Hyperglycaemia, Protein Catabolism, sodium & water retention, impairment of wound healing / immune function, increased metabolic rate, increased fibrinogen and platelet activation
- **Musculoskeletal**
  - Increased muscle spasm secondary to inactivity
  - Increased risk of DVT
- **CNS / Psychological**
  - Anxiety, fear, insomnia & fatigue

PAIN

Classification

- Difficult to classify
- Consider ->

  - **How long** has the patient had pain?
    - Acute v. chronic
  - What is the **cause**?
    - Cancer v. non-cancer
  - What is the **pain mechanism**?
    - Nociceptive
      - Somatic
      - Visceral
    - Neuropathic

PAIN

Types of pain

- **DURATION: Acute & Chronic**
  - Acute
    - < 3 months
    - Recent onset, expected limited duration, localized
    - Usually temporal / causal relationship to injury disease & intensity correlated with trigger
    - Protective function
  - Chronic
    - > 3 months
    - Uncoupled from causative event, intensity does not correlate with stimulus
    - Disease in it's own right
    - No protective function

PAIN

Types of pain

- **CAUSE:**
- Cancer v. Non-Cancer pain
- Non-cancer: Surgery, injury, degenerative disease, childbirth, nerve compression or injury…

- **MECHANISM: Nociceptive, neuropathic or mixed**

- Nociceptive
  - Commonest pain following tissue injury
  - Caused by stimulation of pain receptors in tissues that have been injured
  - Somatic v. Visceral
    - SOMATIC: Pain from body wall structures; can be superficial or deep.
    - VISCERAL: Nociceptors from viscera have larger receptive fields; large amount of convergence with nerves from somatic structures & the ANS. Poorly localized, produces non-specific regional or whole-body responses, ANS responses, produces strong effective responses.

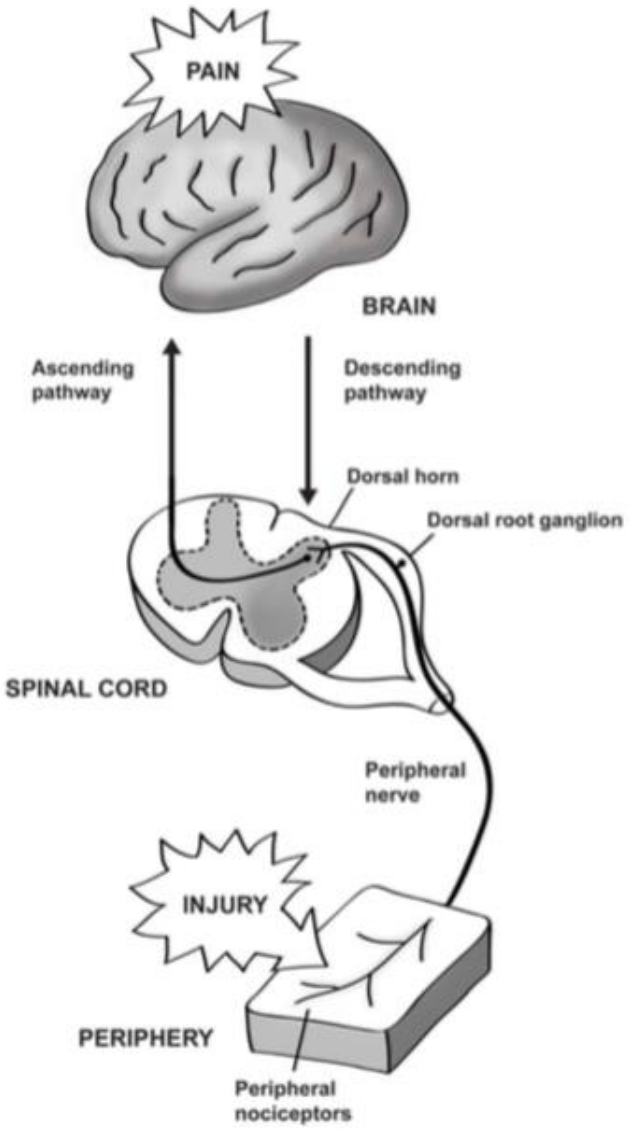
- Neuropathic
  - Abnormal pain signals travelling to the brain & abnormal perception of pain
  - Spontaneously (no stimulus), stimuli that are not normally painful
  - Lesion / disease of nervous system
  - No protective function
  - 'Burning', 'shooting' pain; numbness, pins & needles; poorly localized
  - E.g. Nerve trauma, amputation, diabetic neuropathy, invasive cancer

PAIN PHYSIOLOGY

Pain physiology is complex.

'Normal pain' involves a complicated pathway between the site of injury and the brain. Signals travelling along this pathway can be altered at many points which affects how we experience pain.

In addition, individual patient factors contribute to pain perception. These include but are not limited to psychological factors, beliefs about pain, expectations, personality, social and cultural factors.



PAIN PHYSIOLOGY

ANALGESIA

Start simple… then escalate.

Consider the ‘type of pain’ you are treating and what’s going to be most effective.

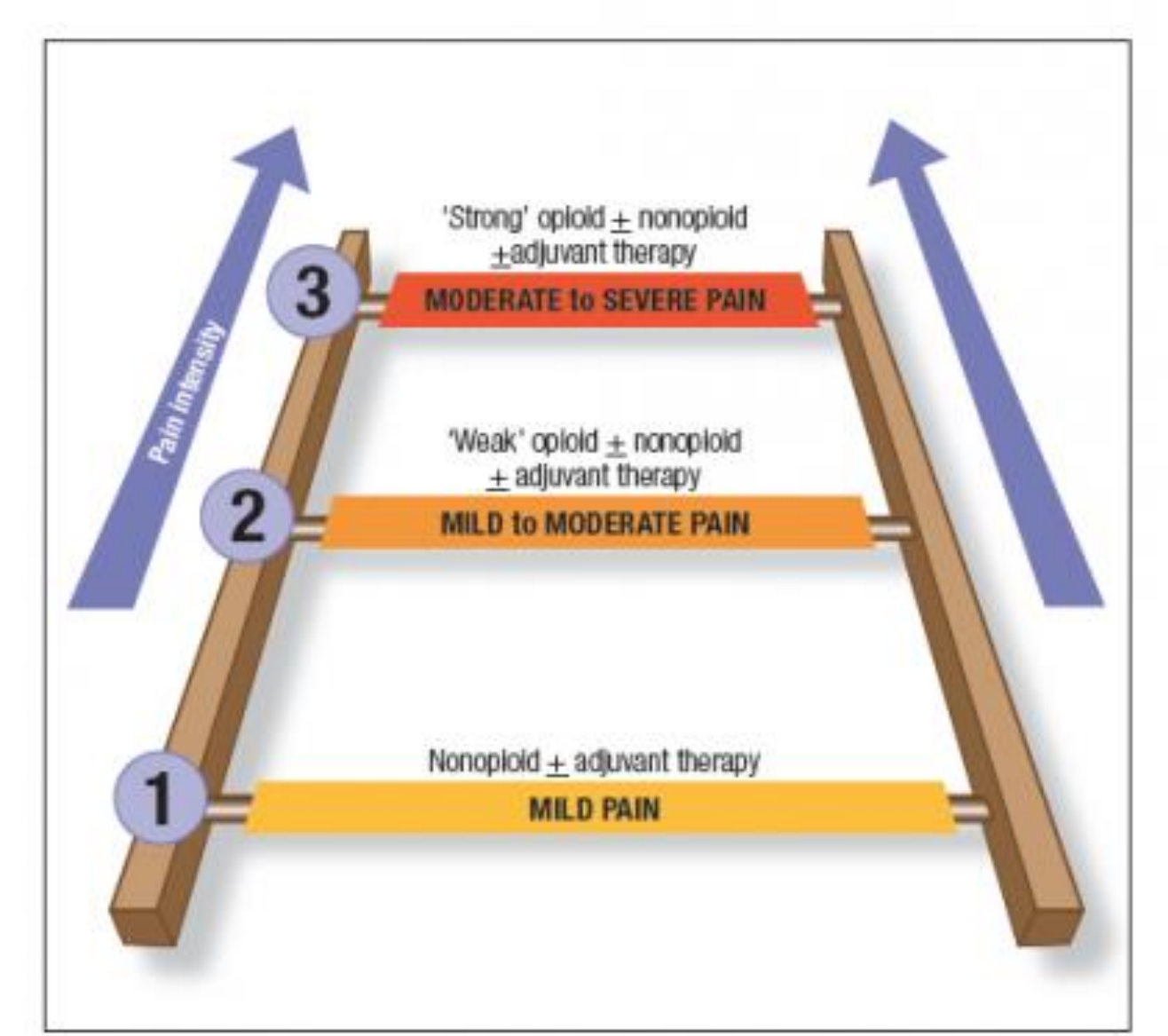
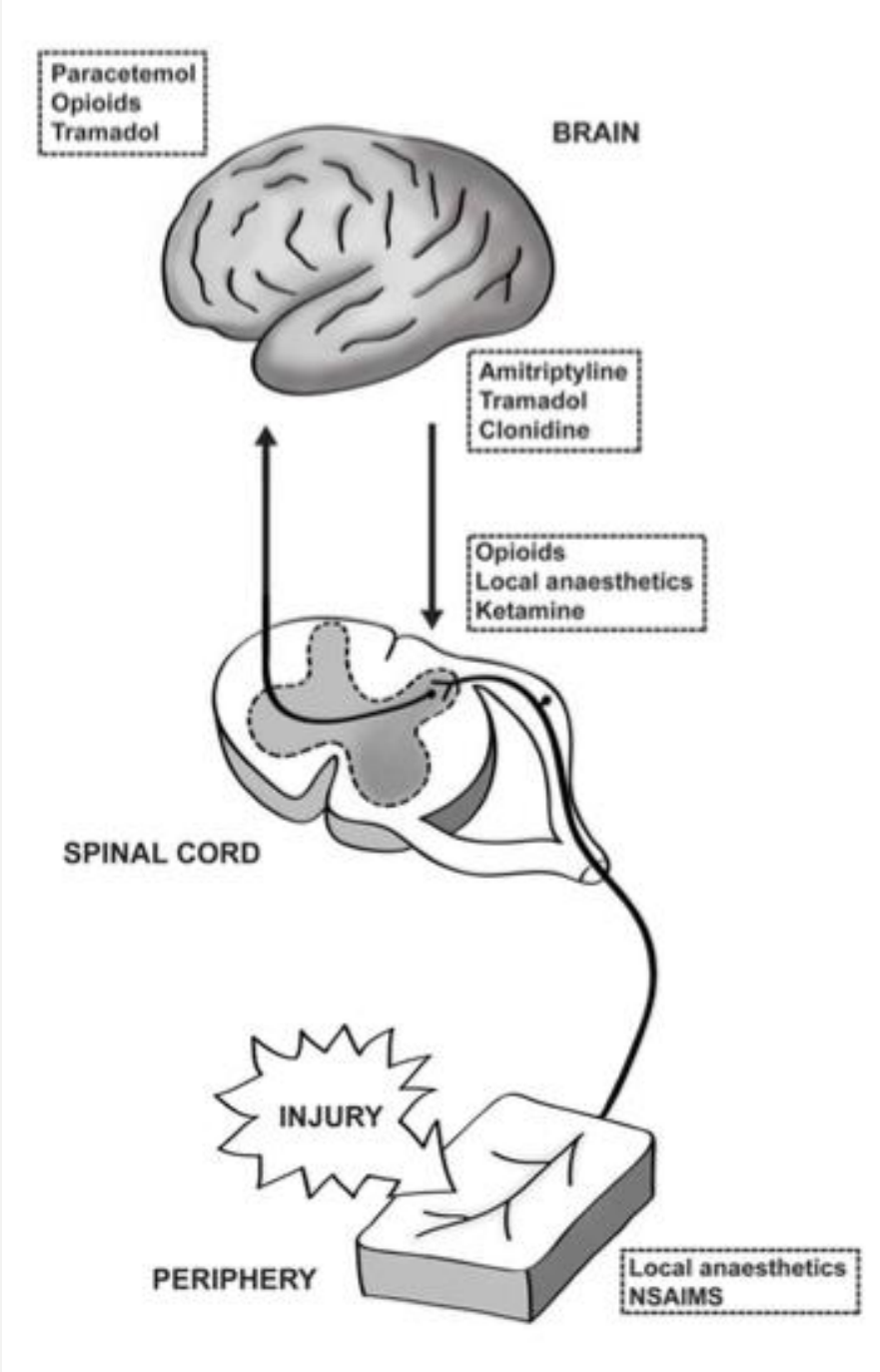


Figure 1. WHO Three-step Pain Ladder. This analgesic step ladder has been the treatment standard most used during the past 3 decades.

https://www.practicalpainmanagement.com/resources/clinical-practice-guidelines/editors-memo-who-pain-treatment-3-step-ladder

ANALGESIA

Simple analgesics	
Paracetamol	Change prostaglandin levels in the brain
NSAIMs	Mainly work by changing prostaglandin levels in the periphery, thereby reducing inflammation
Opioids	
Codeine	Acts on opioid receptors in the brain and spinal cord
Tramadol	Acts weakly on opioid receptors, also increases descending inhibitory signals in the spinal cord
Morphine, pethidine, oxycodone	Act on opioid receptors in the brain and spinal cord
Other analgesics	
Tricyclic antidepressants	Increase descending inhibitory signals in the spinal cord
Anticonvulsants	"Membrane stabilisers", probably work by reducing abnormal firing of pain nerves
Local anaesthetics	Temporarily block signalling in pain nerves in periphery (e.g. infiltration or nerve block) or spinal cord (e.g. spinal block)
Ketamine	Blocks NMDA receptors in the brain and spinal cord (especially in the dorsal horn)
Clonidine	Increases descending inhibitory signals in the spinal cord



PARACETAMOL

Effective analgesia... Not 'just Panadol'; especially when given regularly.

Mechanism of action (MOA):

- Central
- Not entirely clear; ? Decreased central prostaglandin synthesis

Dose

- 1g QID; decrease to TDS for elderly / frail
- Convert to PO as soon as able

Adverse effects

- Few; extremely well tolerated
- Hepatotoxicity
- Pancytopaenia (rare)
- Hypotension post IV administration

NSAIDS

Effectiveness of these analgesics is often underestimated!

- Multiple formulations; in hospital – shorter acting agents safer e.g. Ibuprofen, Celecoxib, Diclofenac, Naproxen
- **MOA**
  - Peripheral
  - Most - Non-specific inhibition of COX-1 & COX-2 which inhibit prostaglandin production
    - COX-2 specific inhibitors e.g Celecoxib
- **Dose**
  - Ibuprofen 200-400mg TDS / QID – max dose 2400mg/day
- **Adverse effects**

These generally prohibit use however, in the right patient, short term therapy of 3-5 days will significantly decrease risk of serious complications and will provide excellent pain relief.

  - GI ulceration
  - Increased bleeding risk (*check with surgical specialty)
  - Bronchospasm in Asthma patients
  - Renal impairment
    - Particularly if pre-existing, dehydration (post-op/diuretics)
  - Thrombotic complications in those with significant pre-existing ischaemic heart disease / cerebrovascular disease

OPIOID COMPARATIVE INFORMATION

Drug	Approximate dose equivalent to 10 mg IM/SC morphine[1]	Approximate duration of action (hours)[2]	Comments
Agonists			
codeine[3] (analgesic only)	200 mg oral	3–4	not recommended (contraindicated in children <12 years)
fentanyl[4]	100–150 mcg SC	1–2	preferred in renal impairment
hydromorphone[5]	1.5–2 mg SC/IM; 6–7.5 mg oral	2–4; 24 (controlled release)	not for first-line use
methadone (analgesic only)	complex	8–24 (chronic dosing)	discuss with a pain or palliative care specialist
morphine[5]	30 mg oral	2–3; 12 or 24 (controlled release)	
oxycodone	15–20 mg oral	3–4; 12 (controlled release)	preferred in renal impairment (adjust dose)
pethidine[5]	75–100 mg IM	2–3	not recommended
tapentadol[6]	75–100 mg oral	4–6; 12 (controlled release)	
tramadol[5]	100–120 mg IM/IV; 150 mg oral	3–6; 12 (controlled release)	
Partial agonists			
buprenorphine (analgesic only)[4]	0.4 mg IM; 0.8 mg sublingual	6–8	not first line for analgesia

https://amhonline.amh.net.au.acs.hcn.com.au/chapters/analgesics/drugs-pain-relief/opioid-analgesics#opioids-table

CODEINE

- **MOA**
  - Produces it's analgesic effect by being metabolized to Morphine
  - Enzyme required for this is P450 2D6
  - Significant inter-individual variability in the activity of this enzyme + many drugs which inhibit / augment -> unpredictable efficacy of codeine
- **Dose**
  - 30-60mg QID; often in combination with Paracetamol or Ibuprofen

Because of its unpredictable efficacy, use in hospital is discouraged… you have much more reliable analgesic options to provide your patients.

TRAMADOL, TAPENTADOL

Tramadol

- **MOA**
  - Binds mu opioid receptors and also inhibits reuptake of Noradrenaline & Serotonin
- **Dose**
  - 50-100mg QID PO / IV; max dose of 400mg / day
  - Controlled release product also available, duration of action 24 hours
- **Adverse effects**
  - CNS stimulation, weakness, sweating , sleep disorder, dizziness, nausea rash
  - Poorly tolerated by elderly
  - Less opioid related side effects
  - *Do not co-prescribe with agents that increase serotonin levels e.g. SSRI due to risk of serotonin syndrome

Tapentadol

- **MOA**
  - Binds mu receptors and inhibits reuptake of Noradrenaline
- **Dose**
  - 50-100mg QID
  - Controlled release product also available, duration of action 12 hours
- **Adverse effects**
  - As for opioids

OXYCODONE (ENDONE)

MOA

- Opioid agonist
- Binds mu receptors, and more weakly to kappa & delta opioid receptors

Dose

- 5-15mg q4-6H; significantly lower doses in elderly patients

Adverse effects (opioids)

- **CNS**
  - N&V
  - Sedation
  - Euphoria, dysphoria, hallucinations
  - Miosis
- **CVS**
  - Vasodilation secondary to vascular smooth muscle relaxation & histamine release resulting in hypotension
- **Resp**
  - Upper airway obstruction secondary to sedation -> snoring
  - Respiratory depression
- **GIT**
  - Constipation – make sure patient is on aperients!
- **RENAL**
  - Urinary retention
- Pruritus

LONG ACTING OPIOIDS

1

Continue long-acting opioids (including Methadone) that patients have been on in the community; this includes post-operatively

2

As a general rule, do not commence long acting opioids for inpatients without seeking senior / expert advice

3

Seek help for patients who are opioid tolerant with acute pain crises / acute post-operative pain

OPIOID OVERDOSE

- Toxic dose of opioids varies considerably with the individual
- Signs of opioid toxicity:
  - Respiratory depression
  - Somnolence -> coma
  - Skeletal muscle flaccidity
  - Miosis
- Give **Naloxone**
  - On PCA chart you will see a standing order for Naloxone:
    - **'100microg x4 every 2-3 minutes'**

OTHER AGENTS

Ketamine

- **MOA**
  - NMDA receptor antagonist
  - Generally used when pain difficult to control e.g. opioid tolerance
- **Adverse effects:**
  - Dysphoria / unpleasant dreams – less common at lose dose infusions
  - HTN & tachycardia
  - Increased muscle tone
  - Lacrimation
  - Hypersalivation
  - N&V

Gabapentanoids

PATIENT CONTROLLED ANALGESIA (PCA)

Doses of opioid +/- background infusion are delivered when the patient presses a trigger (usually hand-held button). The dosage, lockout period between doses, and maximum number of doses per hour can be programmed into the machine. Settings can only be adjusted with key / code.

Important safety feature is that unless the patient is alert enough to press the button they will receive no further analgesia.

Require regular and well documented monitoring including sedation score and respiratory rate.

Key considerations when reviewing a patient on a PCA:

- What is the indication for a PCA? E.g. Surgery, NBM, Colitis & poor absorption etc.
- What drug? What bolus dose? What lockout? Is there a background infusion?
- Does the patient understand how to use it? Are they using it appropriately?
- How many doses has the patient had in the last hour? Last 24 hours? What is the pattern?

PATIENT CONTROLLED ANALGESIA (PCA)

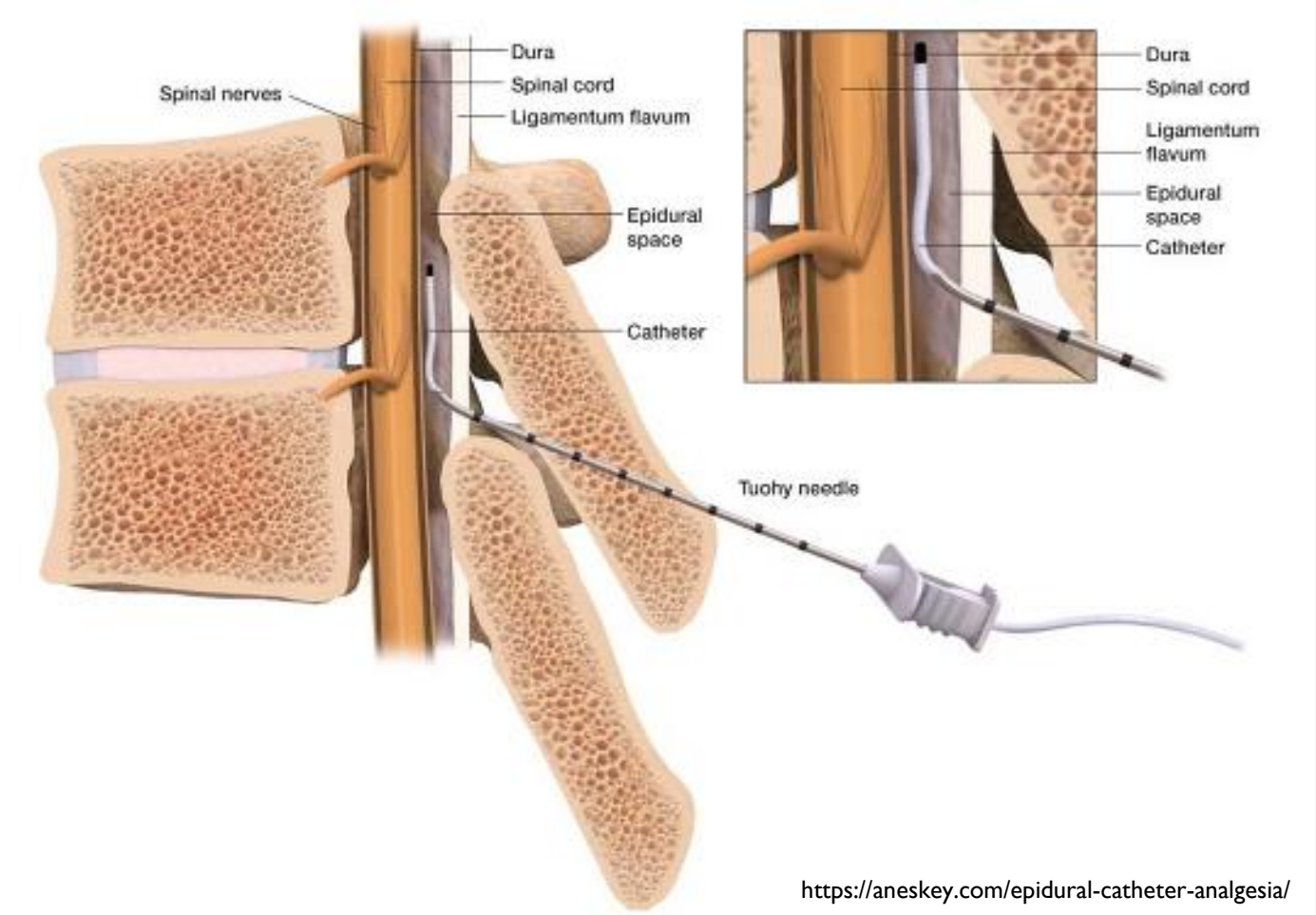
- Most common opioids used:
  - **Morphine**
    - **Diluted to 1mg = 1ml (bolus dose of 1mg)**
    - Advantages: Familiar, cheaper cf other opioids
    - Disadvantages: Sedating, itch, dysphoria
  - **Fentanyl**
    - **Diluted to 10microg = 1ml (bolus dose of 10-20microg)**
    - Advantages: 'Cleaner' drug – better in elderly
    - Disadvantages: Inadequate analgesia if inadequate load
  - **Hydromorphone**
    - **Diluted to 100microg = 1ml (bolus dose of 200microg)**
    - Advantages: Agent of choice in renal failure due to lack of metabolites
    - Disadvantages: Less familiarity

EPIDURAL

Delivery of multimodal agents (Fentanyl, Bupivacaine, Adrenaline) into the epidural space via a catheter for analgesia.

Regular programmed bolus doses +/- additional patient controlled doses.

You are not expected to trouble shoot epidural catheters OR administer additional bolus doses; but should be able to assess a patient with an epidural in-situ and call for help appropriately.



https://aneskey.com/epidural-catheter-analgesia/

EPIDURAL

- **Assessing a patient with an epidural**:
  - Is the **tubing connected consecutively** from the pump to the patient? Is there any leak? Is there swelling at the site of insertion? *Do not take the dressing down.
  - Is the epidural providing **adequate analgesia**?
    - At rest? On deep breathing / coughing? On moving?
  - Check the **level of the block** bilaterally
    - Check sensation to Ice. Do this by demonstrating normal cold intensity in an unaffected part of the body; then assess the level of the block bilaterally by moving the ice caudally / cephalad until normal sensation is reached.
  - Assess **lower limb power**
    - Lower limb weakness should not be present with a thoracic epidural
    - May be present with lumbar epidural; Anaesthetics should be made aware
  - What are the **vital signs**?
    - Hypotension is common. Pt may require vasopressor support; ask for help early.
  - Is the patient on any **anticoagulation**? What agents / doses? Last given?

PAIN CONSULT

- Every patient on a PCA should be known to the Acute Pain Service (APS)

- If patient is known to Palliative Care for pain management e.g. terminal cancer pain; please contact them first.

- **When requesting a pain consult, consider:**
  - What is the **reason for the pain**?
    - Surgical? Cancer? Trauma? Other?
  - **What is the patient on**? Doses? Timing?
    - What has been effective / ineffective?
  - **How does the patient examine**? (you need to see the patient before calling!)
    - Vitals
    - Writhing in pain? Sleeping?
  - Is there a **history of chronic pain**? To whom are they known? What do they normal take – drugs + doses?
  - Is there a **hx of opioid tolerance** e.g. IVDU / ex-IVDU on Methadone / Buprenorphine
  - Any **individual patient factors that are contributing**? e.g. psychological / social / cultural factors

PAIN AT POWH

Designated Pain Team who are very involved with pain management in the hospital and can be of great help to you!

- **In hours:**
  - Acute Pain Service
  - Chronic Pain Service
  - *Palliative Care

- **Out of hours:**
  - Anaesthetic Registrar (pager)

All patients on a PCA will be automatically referred to the Acute Pain Team.

If you wish to start a PCA on a patient please contact the Acute Pain Team in hours OR Anaesthetic Reg out of hours.