OTC Pain Management

University of Hawai‘i Hilo Pre-Nursing Program
NURS 203 – General Pharmacology
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Objectives

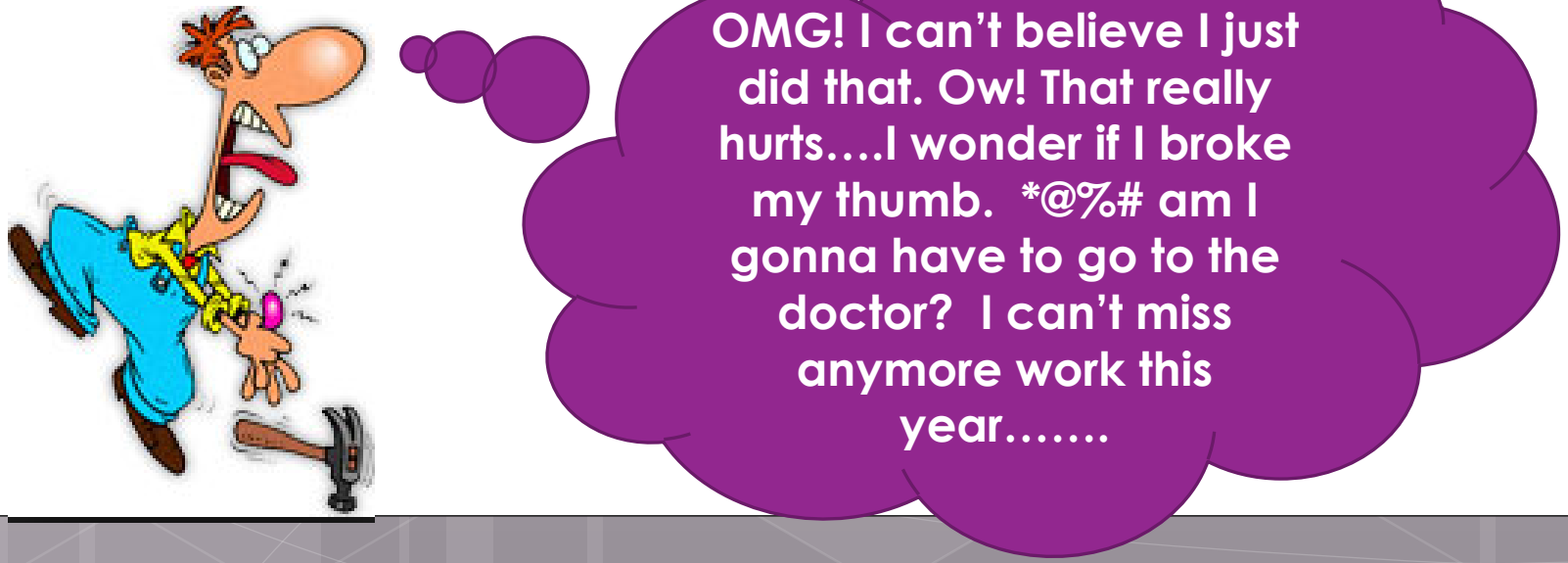
- Understand the definition of pain and the components of that definition
- Know the MOA for OTC pain medication
- Know the major ADRs & interactions of OTC pain medications
- Know indication for OTC pain medications

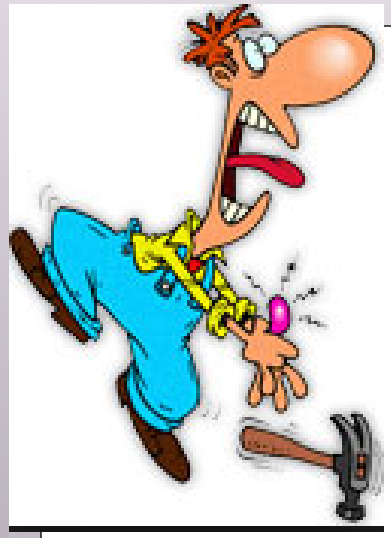
Overview

- NSAIDS
  - Ibuprofen
  - Aspirin
  - Naproxen
- Acetaminophen
- Combinations
  - Excedrin®
- Topical
  - Menthol
  - Capsaicin

Pain

- What is pain?
  - Pain is made up to 2 components
    - Physical – sensation of pain
    - Psychological – emotional reaction to that sensation

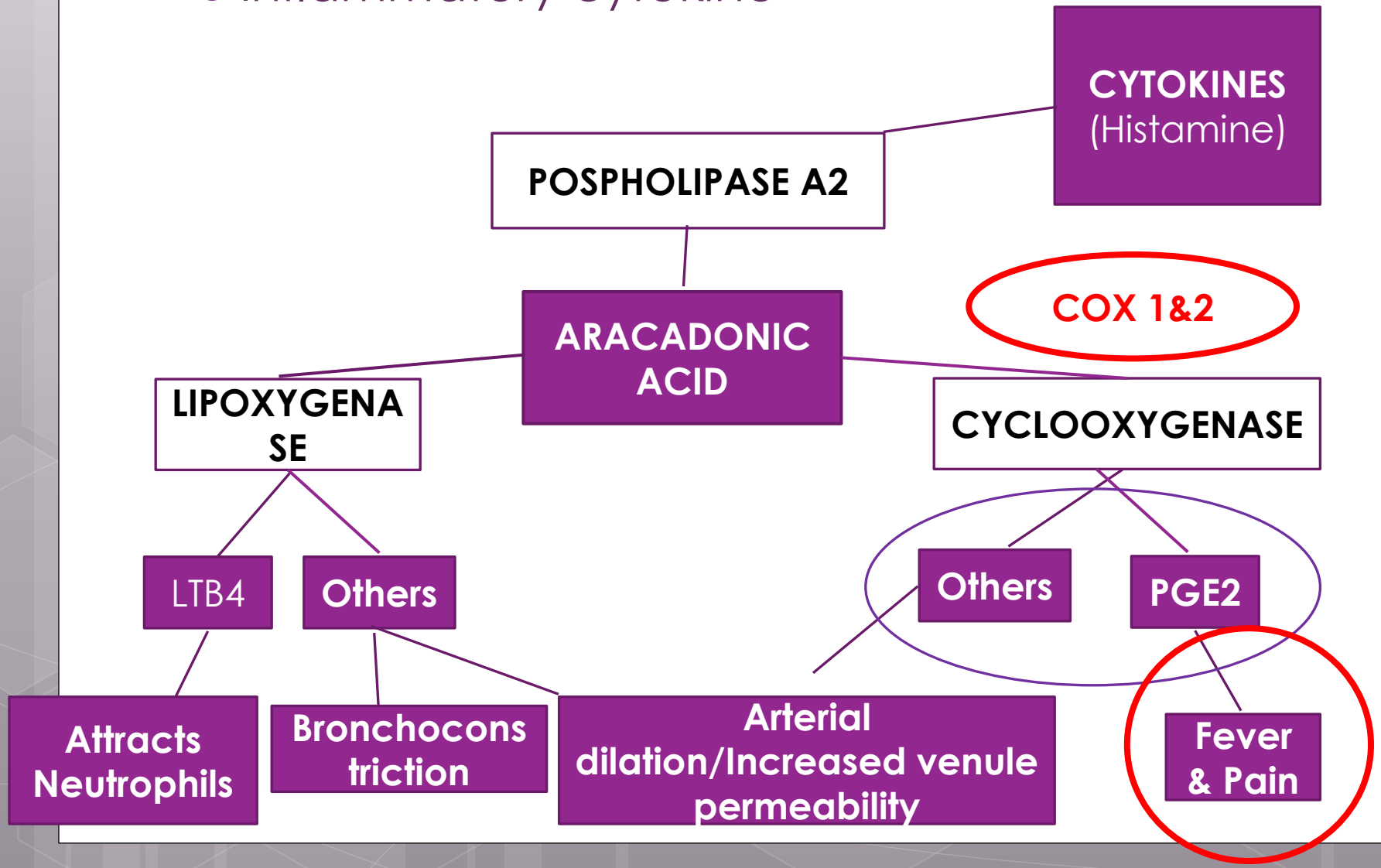


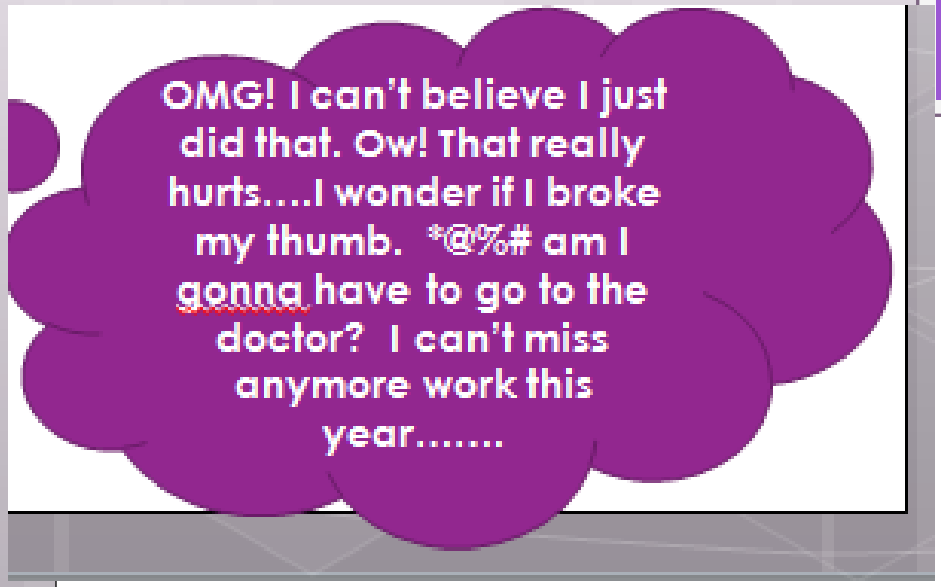


Physical component

- Nociceptors
  - Nerve cell ending that initiate the sensation of pain
    - Frequency of firing (action potentials)determines the intensity of the pain
- Prostaglandins
  - Important local hormones important in the sensation of pain
    - PGE2 – vasomotor tone, capillary permeability, smooth muscle tone, platelet aggregation, endocrine & exocrine functions, and CNS

- Inflammatory cytokine





Psychological component

- Age
- Gender
- **Anxiety/Emotions**
- Experiences
- Culture

Emotions Involved in Tolerance

Which is **TRUE** of pain/pain management

- A patients perception of chronic pain can be paired with vital sign changes
- Severe chronic pain cannot be effectively controlled
- Opioids are addictive and a treatment of last resort because of unmanageable adverse effects
- The goal of chronic pain management is to keep the dose of medication as low as possible
- Studies show that women are at a greater risk of being undermedicated for pain

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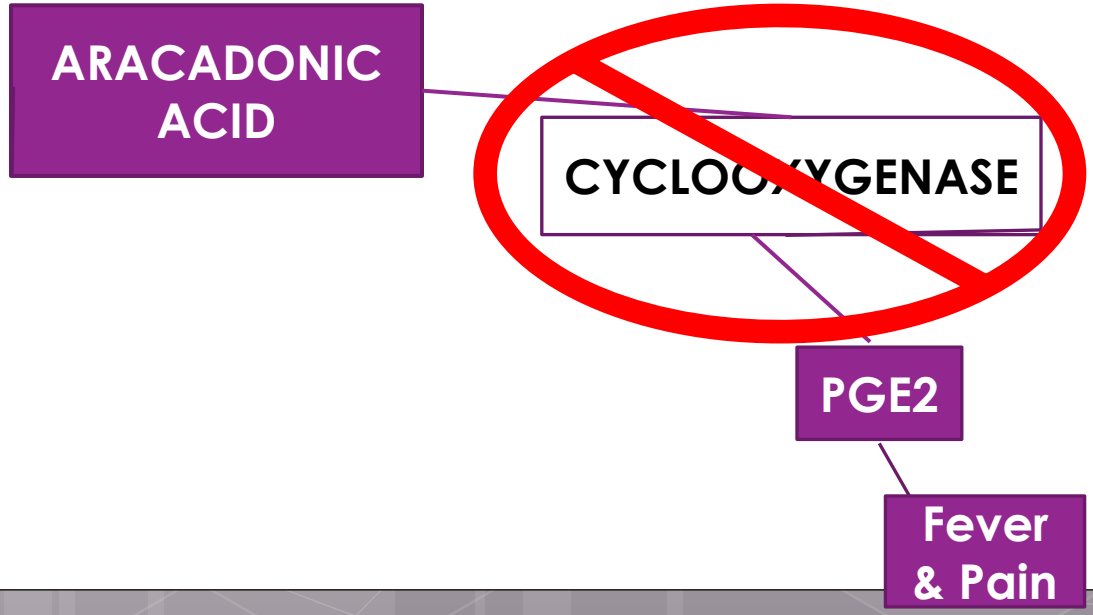
Treatment of Pain

- Ibuprofen

Ibuprofen – Advil/Motrin

- MOA - **Reversibly inhibits cyclooxygenase-1 and 2 (COX-1 and 2) enzymes**, which results in decreased formation of prostaglandin precursors; has antipyretic, analgesic, and anti-inflammatory properties. Other proposed mechanisms not fully elucidated (and possibly contributing to the anti-inflammatory effect to varying degrees), include inhibiting chemotaxis, altering lymphocyte activity, inhibiting neutrophil aggregation/activation, and decreasing proinflammatory cytokine level

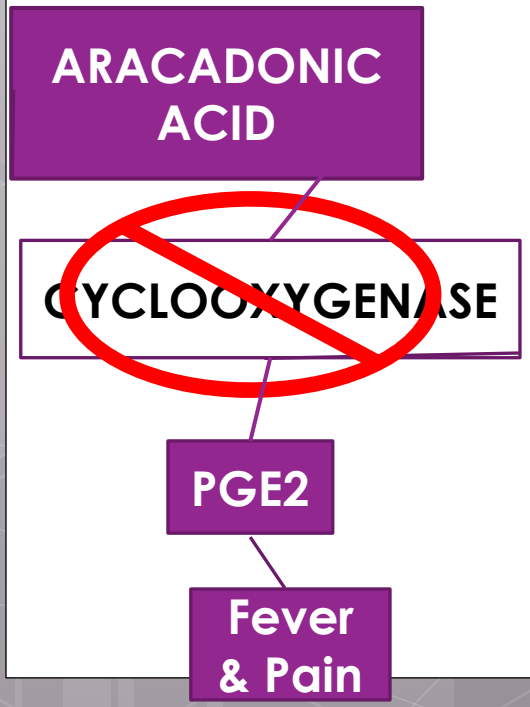


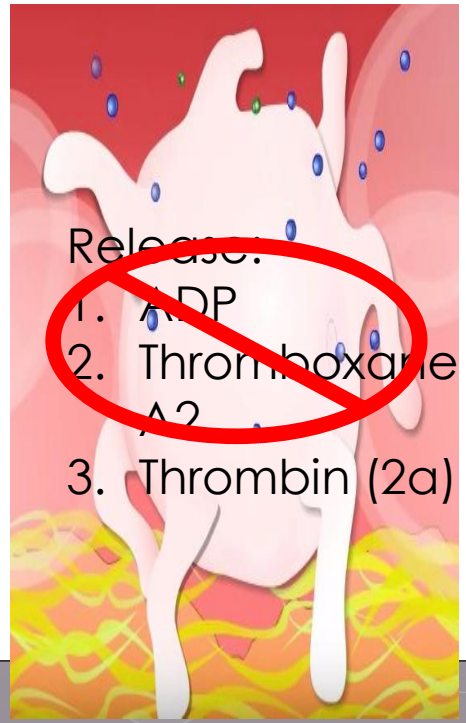
Ibuprofen

- Kinetics
  - Onset – 30-60 minutes (oral)
  - Duration 6-8 hours
  - Highly protein bound (>99%)
  - Metabolized – liver
  - Excreted – urine (only 1% unchanged drug
- ADRs
  - Epigastic pain, heartburn, & nausea (take with food)
  - Tinnitus
  - CV – edema & fluid retention
- Pregnancy
  - Not recommended
- Interactions
  - Anticoagulants, ACEI & ARBs, & others

Aspirin

- MOA - Irreversibly inhibits cyclooxygenase-1 and 2 (COX-1 and 2) enzymes, via acetylation, which results in decreased formation of prostaglandin precursors; irreversibly inhibits formation of prostaglandin derivative, thromboxane A_2, via acetylation of platelet cyclooxygenase, thus inhibiting platelet aggregation; has antipyretic, analgesic, and anti-inflammatory properties



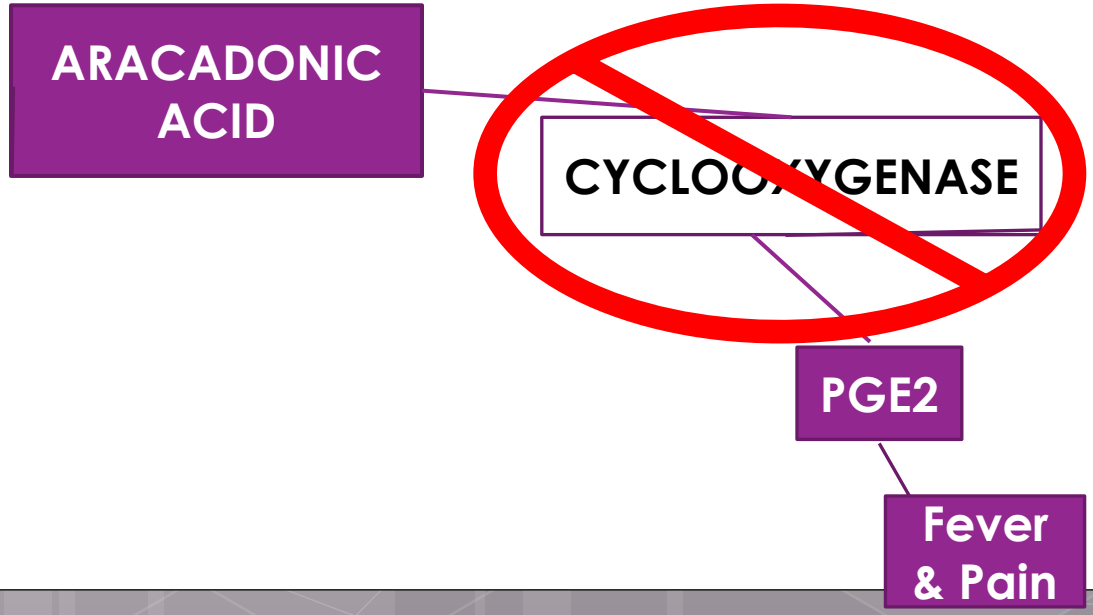


Aspirin

- Kinetics
  - Absorbed – Rapid
  - Duration – 4-6 hours
  - Distributes – into most fluids & tissues readily
  - Metabolized – liver
  - Excreted - Urine
- ADRs
  - Bleeding
  - CV – edema, arrhythmia, hypotension
  - GI – ulcer, heartburn, nausea, stomach pain
- Pregnancy
  - Not recommended
- Interactions
  - Anticoagulants, ACEI & ARBs, other salicylates

Naproxen - Aleve

- MOA - Reversibly inhibits cyclooxygenase-1 and 2 (COX-1 and 2) enzymes, which results in decreased formation of prostaglandin precursors; has antipyretic, analgesic, and anti-inflammatory properties. Other proposed mechanisms not fully elucidated (and possibly contributing to the anti-inflammatory effect to varying degrees), include inhibiting chemotaxis, altering lymphocyte activity, inhibiting neutrophil aggregation/activation, and decreasing proinflammatory cytokine levels



Naproxen

- Kinetics
  - Duration - >12 hours
  - Onset – 30-60 minutes
  - Protein binding - >99%
  - Half life – 12-17 hrs (increased in renal impairment)
  - Metabolized – liver
  - Excreted - Urine
- ADRs
  - CV – edema
  - Dizziness, drowsiness, HA
  - Bruising, itching, & rash
  - Tinnitus
  - Shortness of breath
- Pregnancy
  - Not recommended
- Interactions
  - Anticoagulants, ACEI & ARBs, others

Acetaminophen - Tylenol

- MOA - Although not fully elucidated, believed to inhibit the synthesis of prostaglandins in the central nervous system and work peripherally to block pain impulse generation; produces antipyresis from inhibition of hypothalamic heat-regulating center

- Theories
  - COX inhibitor
  - Endocannabinoid
  - Serotonin

Acetaminophen

- Kinetics
  - Onset - < 1hr
  - Duration 4-6 hrs
  - Protein binding only 10-25% at therapeutic doses (> at toxic)
  - Metabolism – glucuronidation & CYP2E1
  - Half life – changes with age & renal fx
  - Excretion - urine
- ADRs
  - Nausea & vomiting
  - Rash & hypersensitivity
  - Generally well tolerated
- Pregnancy
  - Recommended
- Interactions
  - Minor
  - Avoid in EtOH abuse & liver disease

WAIT!!!

Acetaminophen

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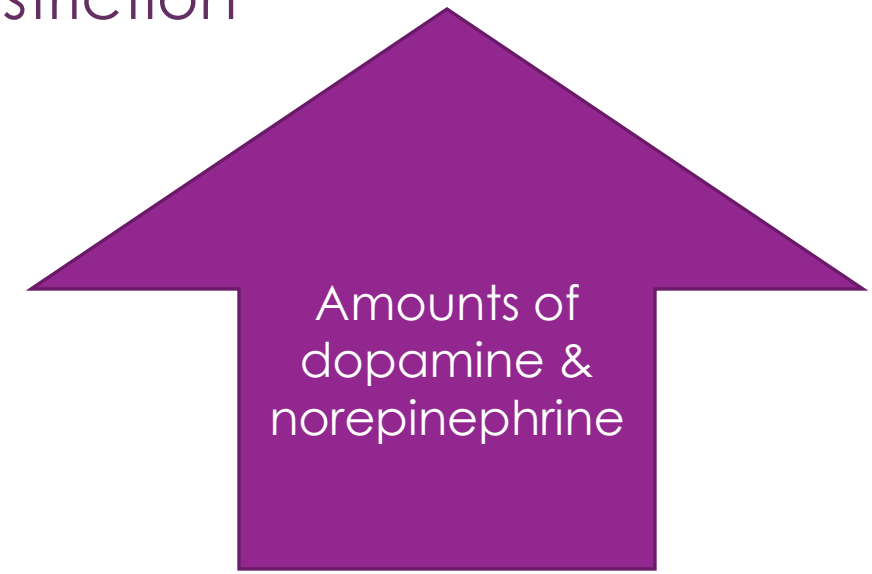
  - Half life – changes with age & renal fx
  - Excretion - urine

This is important!!!!
ADULT PATIENTS MUST NOT EXCEED MORE THAN 4 GRAMS IN 24 HOURS.
Accumulation of a dangerous metabolite via the CYP2E1 route. (NAPQI)
Glucuronide metabolism is saturable.
Liver damage
Liver failure
Death

- Interactions
  - Minor

Excedrin – acetaminophen, aspirin, & caffeine

- MOA – Caffeine – adenosine antagonist/sympathomimetic/cerebral vasoconstriction



Excedrin

- Indications
  - Minor aches & pains
  - Headache (migraine)
- Administration
  - Take with food to avoid GI upset
- Dosing
  - Available with different strengths of each component
- ADRs
  - GI upset, hepatotoxicity, hypersensitivity, & skin reactions
  - Rebound headache
- Interactions
  - Similar to individual components
    - Anticoagulants, ACEI & ARBs, etc.

Topicals

- Menthol

- Capsaicin

Topicals

- Menthol
  - When applied to the skin **menthol dilates the blood vessels, causing a sensation of coldness followed by an analgesic effect.** It relieves itching and is used in creams, lotions, or ointments in pruritus and urticaria. It has also been applied to the forehead, presumably as a counter-irritant, for the relief of headache.
- Capsaicin
  - Causes depolarization of nociceptive nerve fibers, initiation of action potential, and pain signal transmission to the spinal cord; capsaicin exposure results in **desensitization of the sensory nerve and inhibition of pain transmission initiation**. In arthritis, capsaicin induces release of substance P, the principal chemomediator of pain impulses from the periphery to the CNS , from peripheral sensory neurons; after repeated application, **capsaicin depletes the neuron of substance P** and prevents reaccumulation. The functional link between substance P and the capsaicin receptor, TRPV1, is not well understood.

Topicals

- Menthol
- ADRs
  - Local
    - Contact dermatitis
  - Systemic
    - Toxic if too much consumed – severe abd pain, N&V
- Interactions
  - No known drug interactions

- Capsaicin
- ADRs
  - Local
    - Redness & pain
  - Systemic
    - Some – well tolerated
- Interaction
  - No known drug interactions

Questions

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