Managing Wound Pain & Inflammation To Promote Healing

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NCCHC NATIONAL COMMISSION ON CORRECTIONAL HEALTH CARE

Disclosures

I have the following relevant financial relationship with a commercial interest:

I work as an Independent Researcher for tropical developing countries, and as the Clinical Research, Education, & Charity Liaison for Ferris Mfg. Corp. (makers of PolyMem dressings and SportsWrap)

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Learning Objectives

1: Describe how pain and inflammation interact to slow wound healing

2: Identify appropriate tools for assessing pain and interpret the results

3: Design a care plan using a nonprescription intervention to help a patient manage wound pain and inflammation

Outline

Overview of Pain

I. Benefits and Disadvantages of Pain & Inflammation

II. How Injury ⟶ Inflammation & Pain

III. The Role of the Nociceptors in Healing

IV. Wound Pain Assessment

With strategies to address each finding

V. Additional Interventions for Managing Pain

Why is Managing Wound Pain Important?

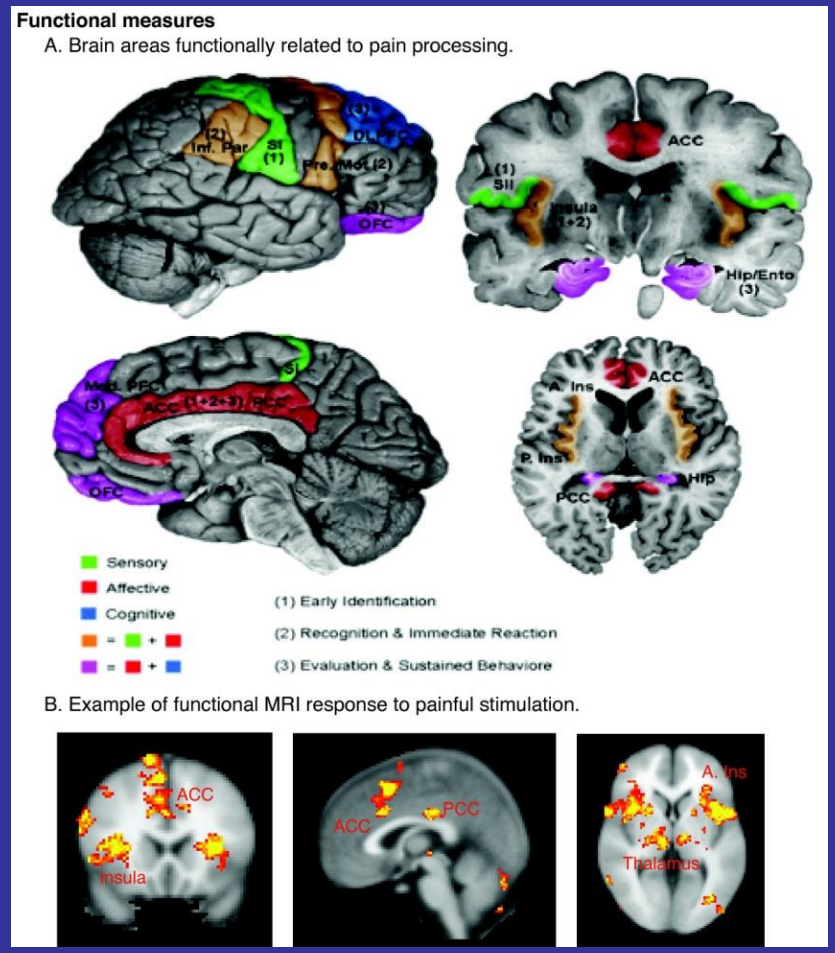
- Wound pain prevalence and incidence is very high
- Constitutional rights (standard of care)
- Compliance (patient satisfaction, avoiding penalties)
- Cost containment
  (decreased burden on patients & the health care system)
- Compassion (quality of life)

- Wound pain DIRECTLY inhibits wound healing!

International Association for the Study of Pain (IASP) Definition

*"**Pain** is a **sensory** and **emotional** experience associated with actual or potential **tissue damage**, or described in terms of such damage"*

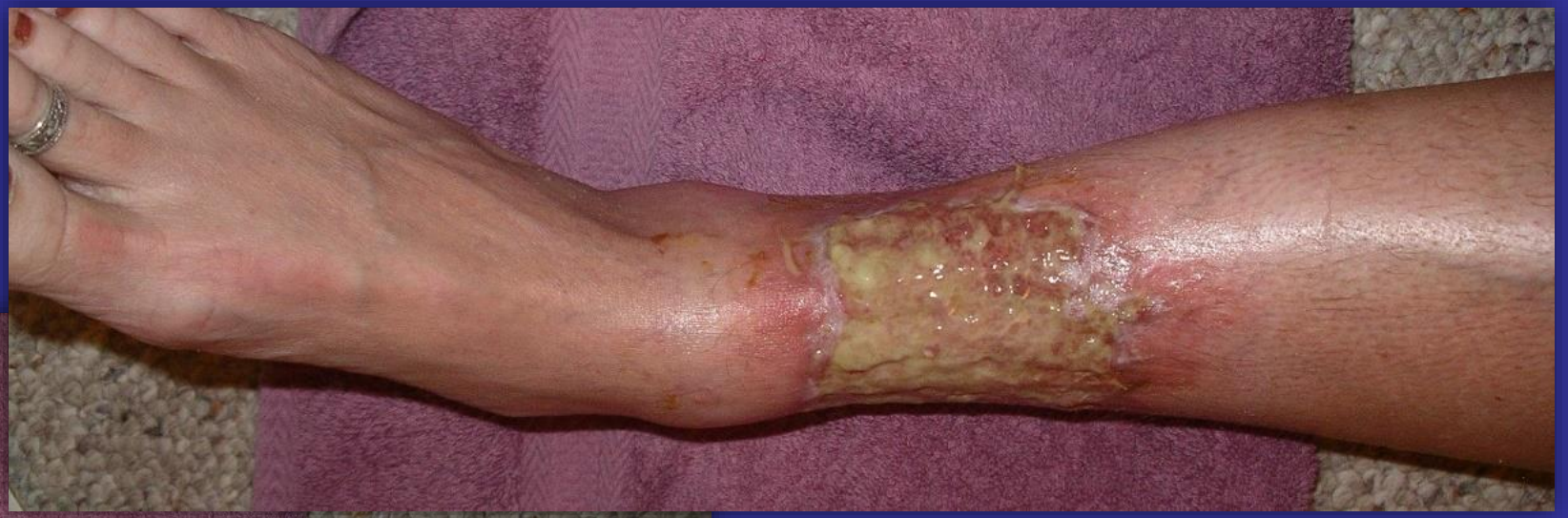
Pain is what the patient feels. Period.

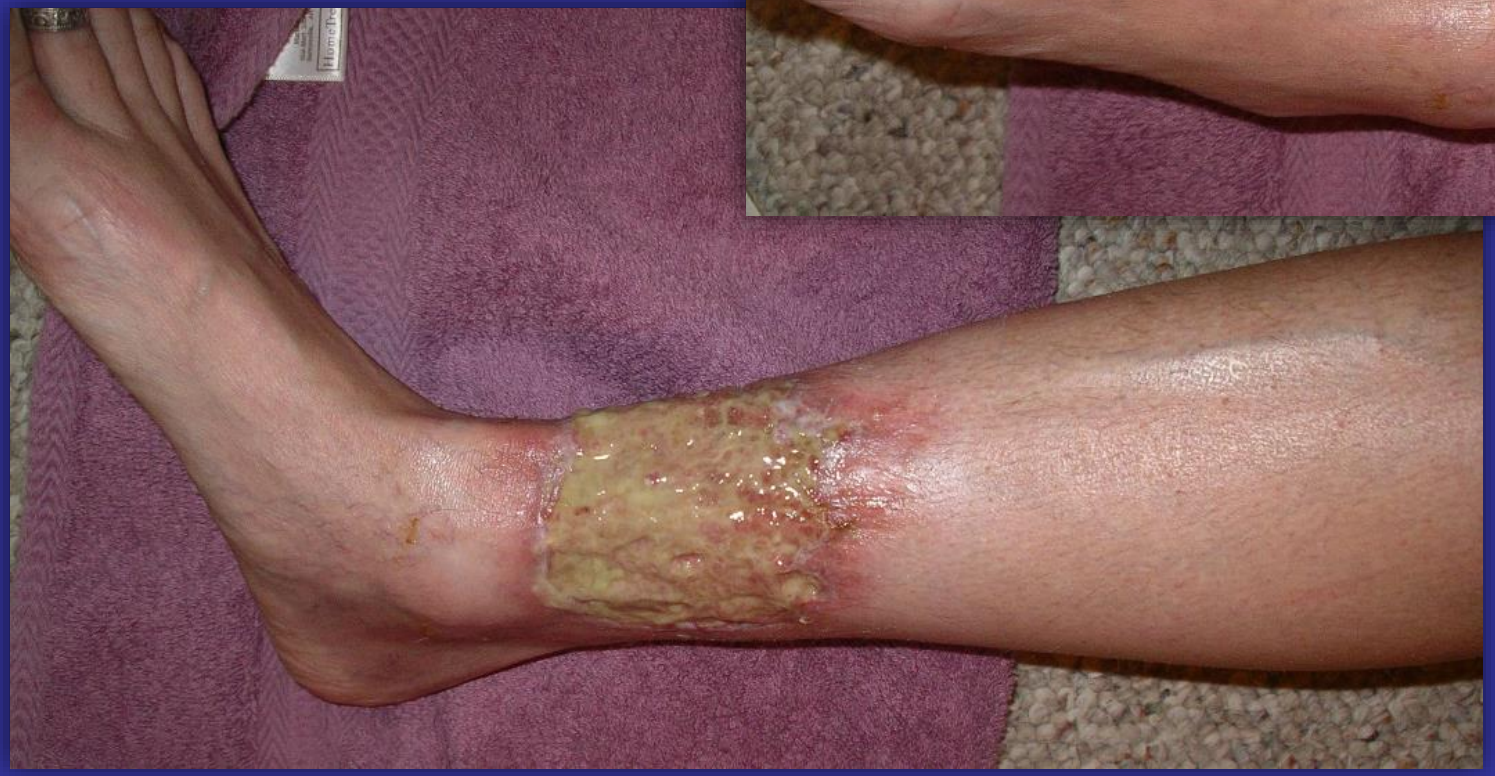


Creative Commons Image By Borsook D, Moulton EA, Schmidt KF, Becerra LR. Schematic_of_cortical_areas_involved_with_pain_processing_and_fMRI

FOCUS ON JUST ONE PATIENT

GOAL: to restore function to an acceptable level





Acceptable to whom?

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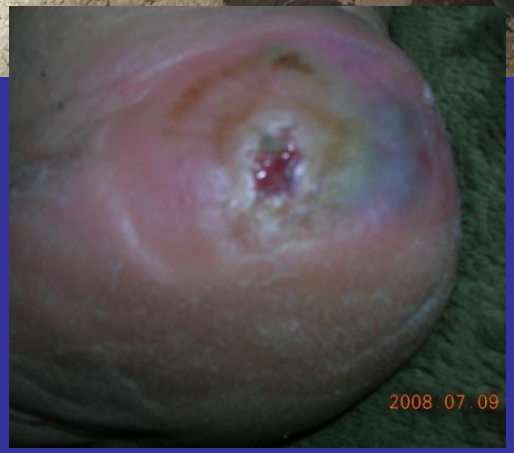
“Pain is God’s greatest gift to mankind”

– Dr. Paul Brand

1. Helps us avoid injury
2. Often first sign of injury
3. Character & intensity - diagnostic
4. Helps prevent re-injury
5. Increased pain can signal infection
   (due to the inflammation)
6. Persistent pain, persistent problem?

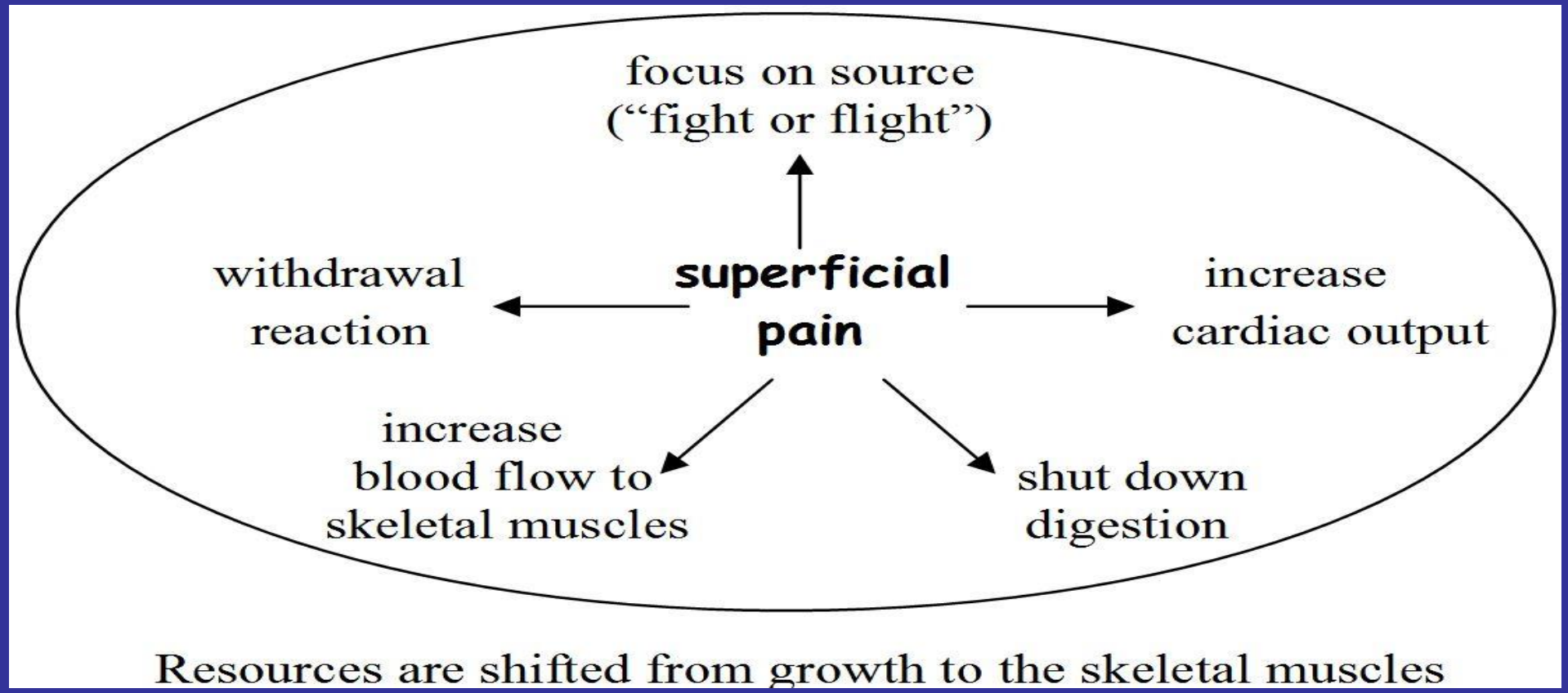


Disadvantages of Pain

Catabolism: Tissue Breakdown or Anabolism: Tissue Building

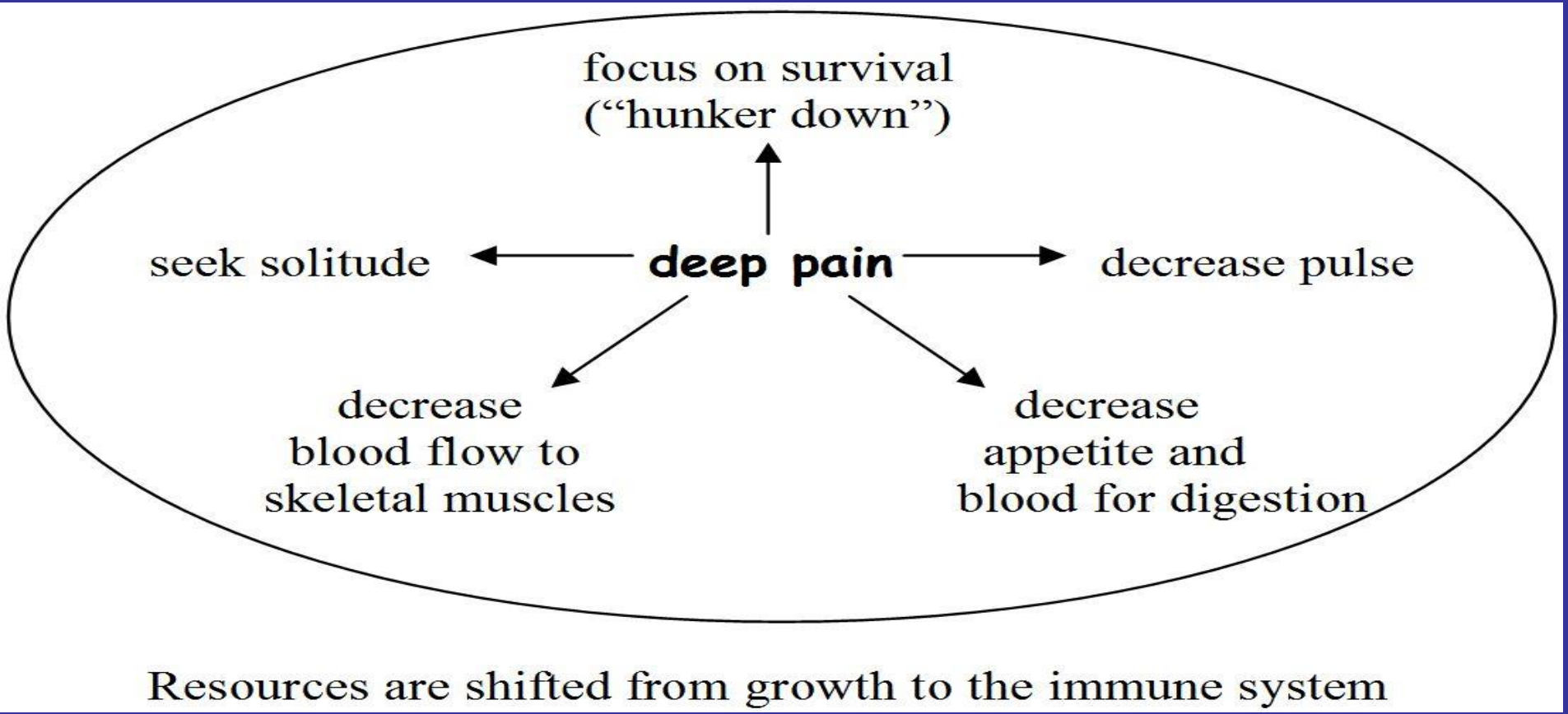
Inflammation Proliferation

Superficial Pain Inhibits Wound Healing



Superficial (most wound) pain ⟶"fight or flight" (cortisol, adrenaline)
Puts body into a catabolic (tissue breakdown),
NOT an anabolic (tissue building), state

Deep Pain Also Inhibits Wound Healing

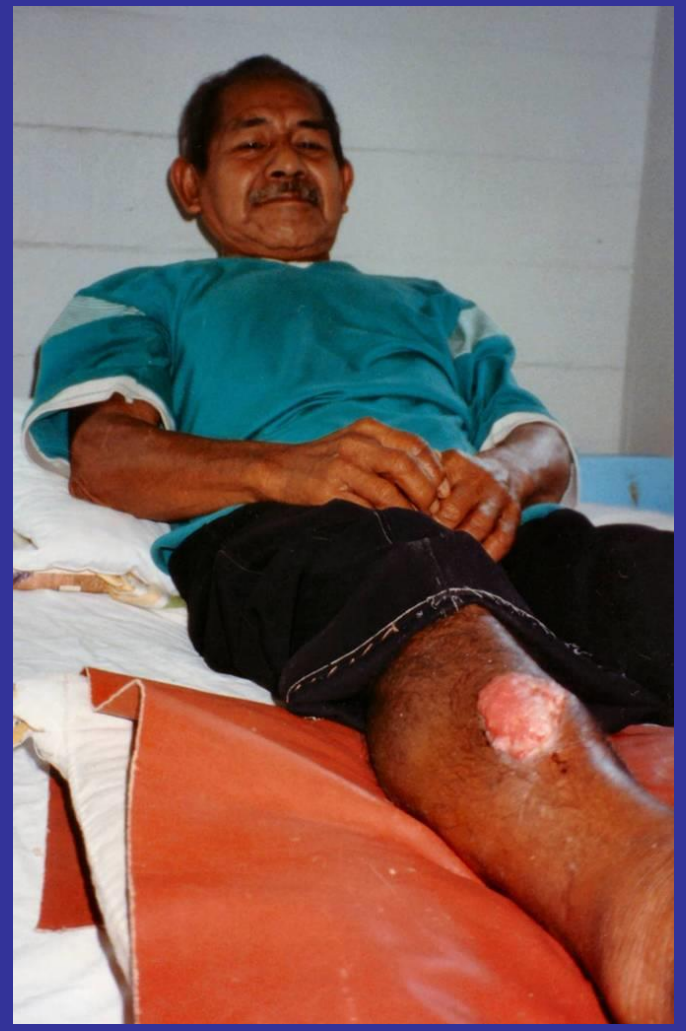


Deep pain ⟶ "hunker down" ⟶ prevent sepsis

Puts body into a catabolic (tissue breakdown),
NOT an anabolic (tissue building), state

All Chronic Pain Inhibits Healing

a. The body stays in a catabolic state
b. Resources are shifted away from growth
c. Digestion is impaired (poor nutrition)
d. Patient withdraws from healthy activities
e. Leads to a long-term inflammatory state



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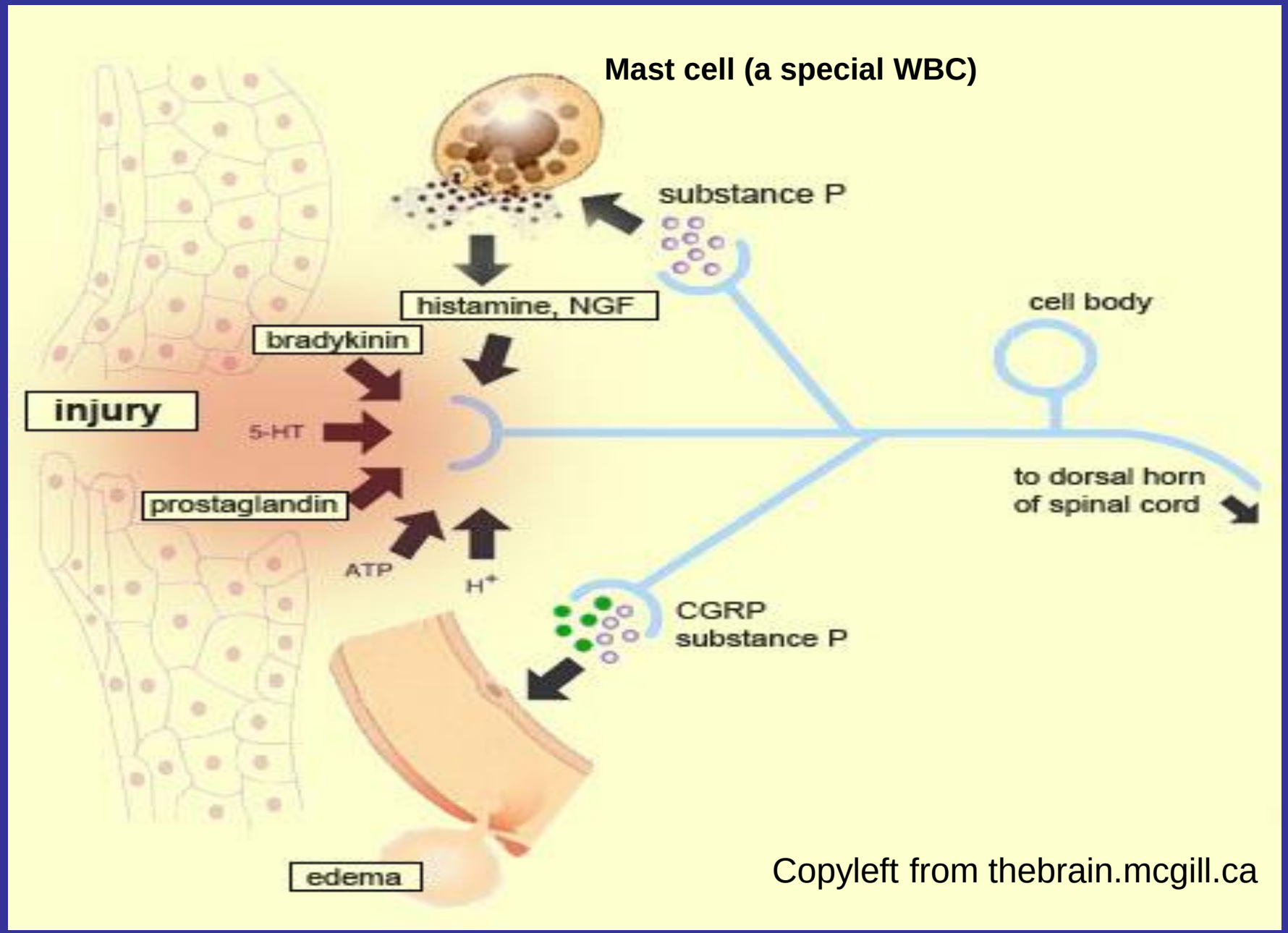
Some of the Chemicals Involved in Wound Inflammation

Excitatory (Increase Pain and Inflammation Overall)			
Chemicals	**Released by**	**Acts on**	**To Cause**
Cell-derived Inflammatory Mediators: **Histamine** **Prostaglandin** **Bradykinin**	➢ Mast cells (histamine only) ➢ Macrophages (triggered by bacteria, H+ & K+ from injured cells) ➢ Tissues at injury area ➢ Platelets ➢ Brain via nociceptors (Prostaglan, bradykinin)	Capillaries & Arterioles Peripheral Terminals of the Nociceptors	Vessels leak & get wider, (edema, heat, redness, bruising) Recruit MMPs & Growth Factors Smooth muscles contract (prevents hemorrhage) Nociceptors release P & CGRP (increase inflammation) Brain feels pain and/or itch
Pro-Inflammatory Cytokines: **Substance P** **CGRP**	Nociceptors (vesicles signaled by histamine at the injury site)	Mast cells (Substance P → release histamine) Capillaries & Arterioles	Brain feels pain Vessels leak & get wider Inflammation increases
Inhibitory (Decrease Pain Overall)			
Modulators: **Endorphins** **Seratonin**	Nociceptors, when signaled by brain	Inhibit Prostaglandin and Bradykinin	Endorphins: Decreased pain and inflammation Seratonin: Decreased pain, but can prolong inflammation



Copyleft from thebrain.mcgill.ca

Nociceptor (a pain sensing nerve)

Local Effects of Inflammatory Mediators

1. Increased blood vessel permeability (intentional leaking)
   + WBCs enter tissue to kill microbes, prevent infection
   + Enzymes arrive to clean up area (recycle microbes & tissue)
   – Albumin escapes → Edema (which can impair circulation)
   – Red Blood cells escape → Bruising, pain
2. Vasodilation (widening of vessels, increased circulation)
   + Repair substances (MMPS, growth factors, etc.) arrive quickly
   – Warmth and redness (easily mistaken for infection)
3. Smooth muscle contraction → + No hemorrhage; – Cramps

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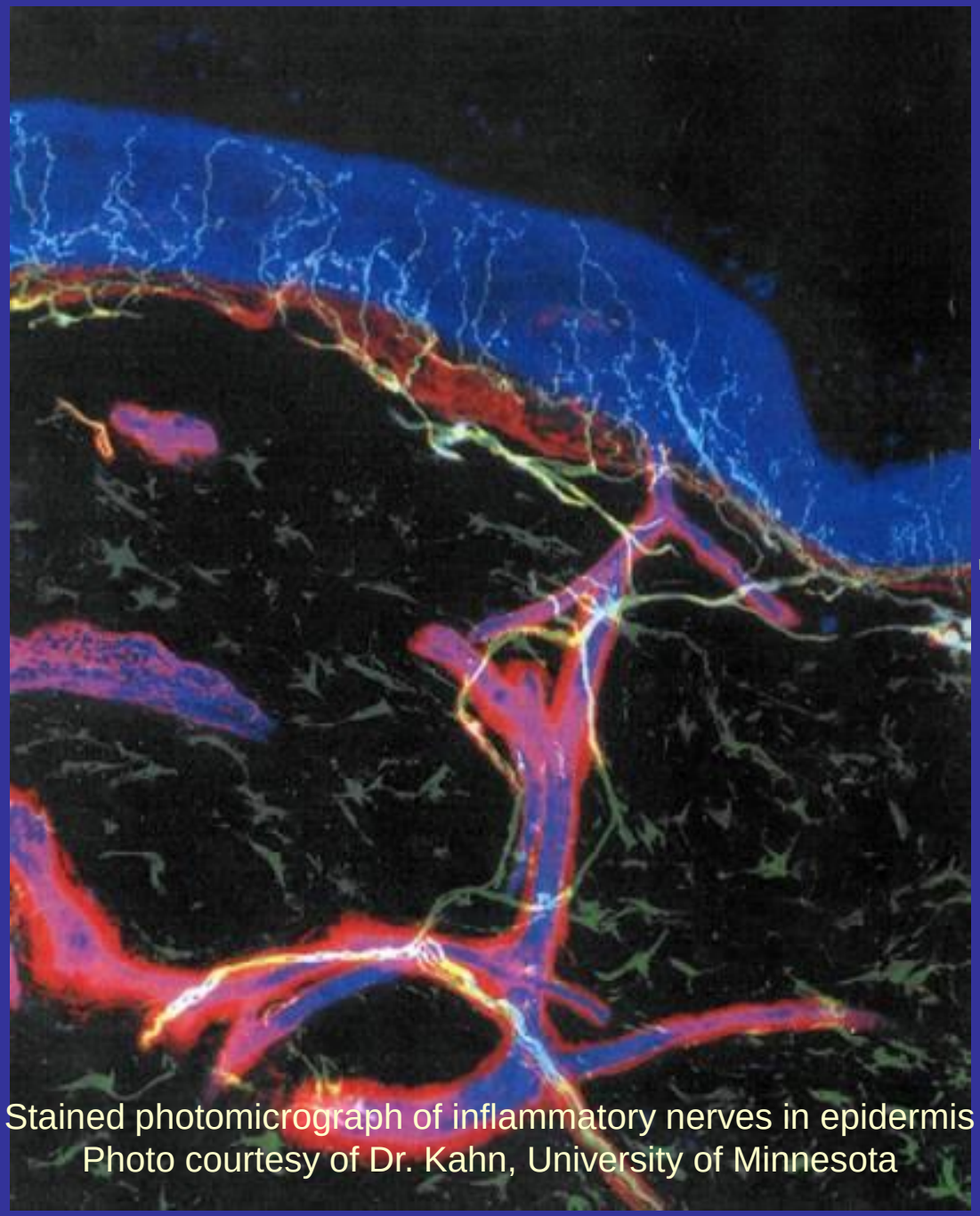
A. Nociceptor response to injury

B. How this affects healing

IV. Wound Pain Assessment

V. Interventions to Diminish Wound Pain



Stained photomicrograph of inflammatory nerves in epidermis
Photo courtesy of Dr. Kahn, University of Minnesota

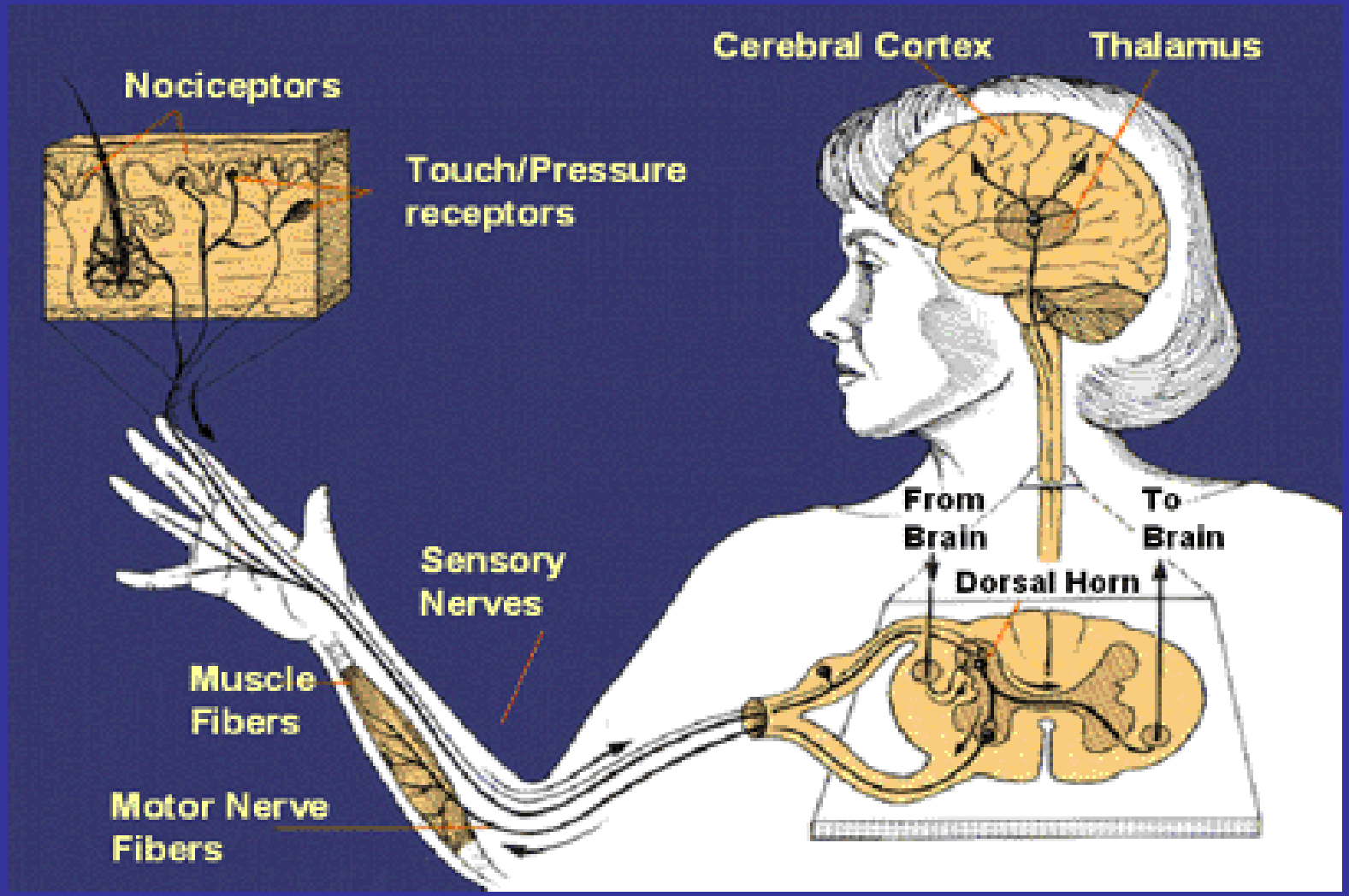
The yellow & white lines that look like lighting bolts are the nociceptor (pain nerve) fibers.

Epidermis

Main Roles of Nociceptors:

1. Maintain homeostasis
2. Sense pain & itch

The Nociceptors



Nociceptors report pain and itch to the brain.

Sensory nerves are activated even if heat, cold, or pressure is too mild to trigger nociceptors

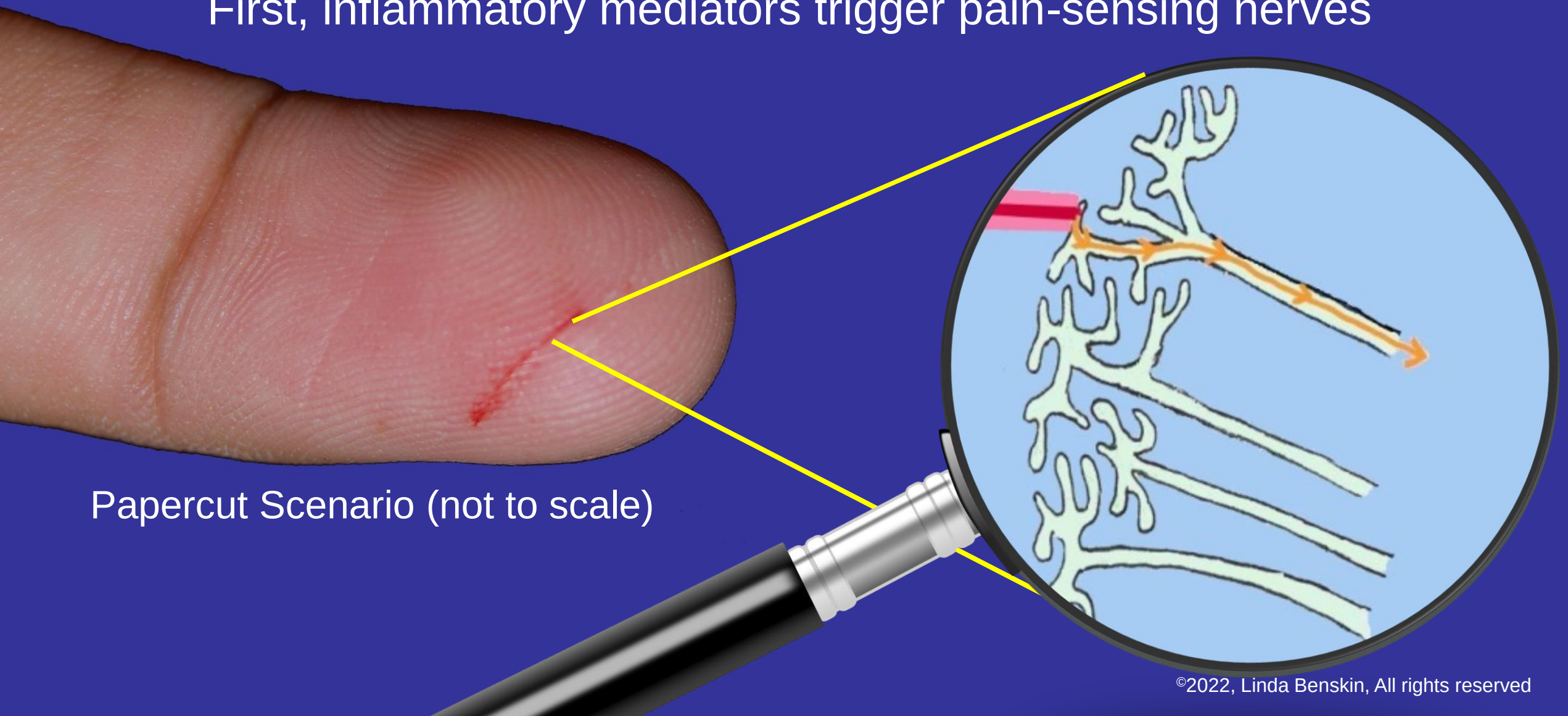
Three sets of nerve cells transfer information to the brain

Pain messages go to both the somatosensory cortex and the limbic system (hub of emotions)

The longest nerves are most easily damaged; neuropathy usually starts in the feet

Nociceptor Response to Injury

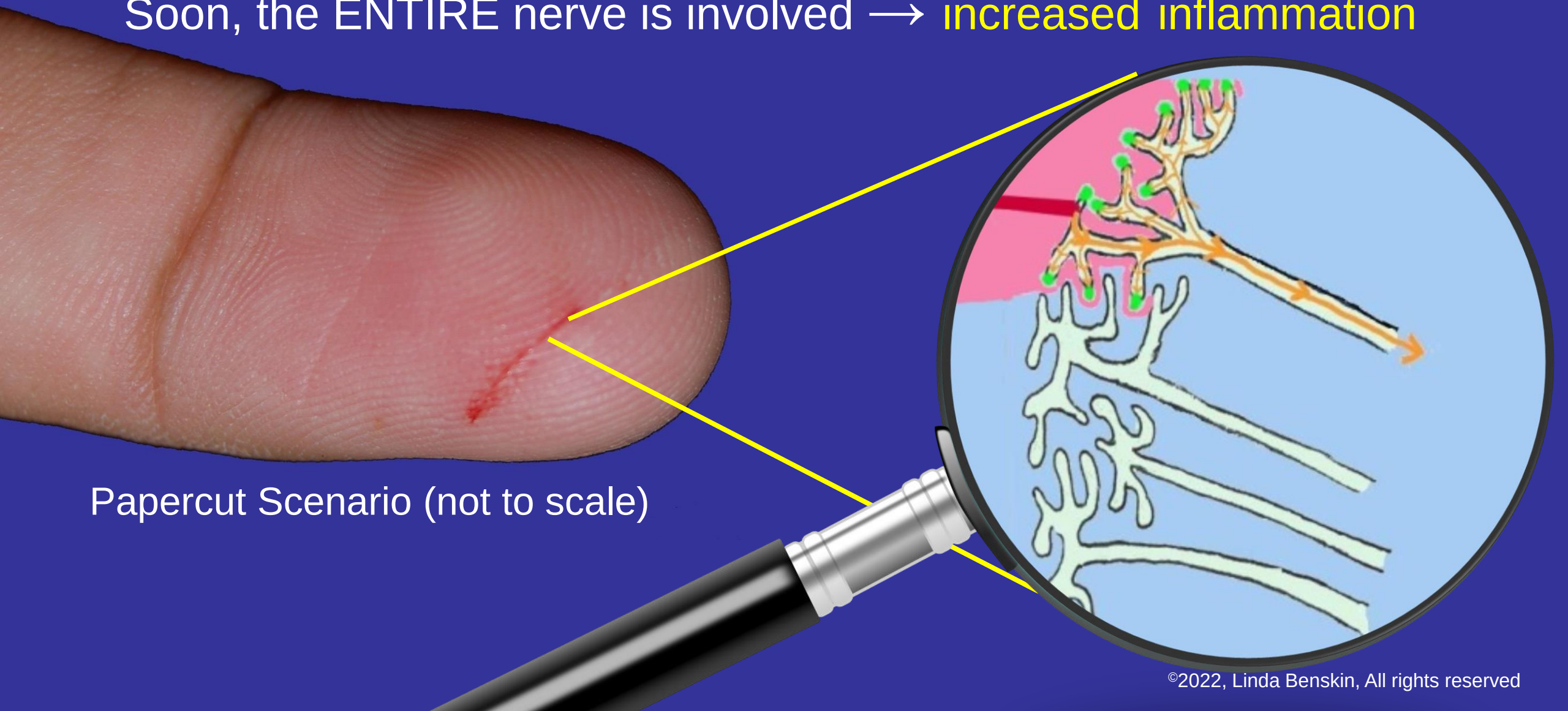
First, inflammatory mediators trigger pain-sensing nerves



Papercut Scenario (not to scale)

Nerves Increase the Inflammation

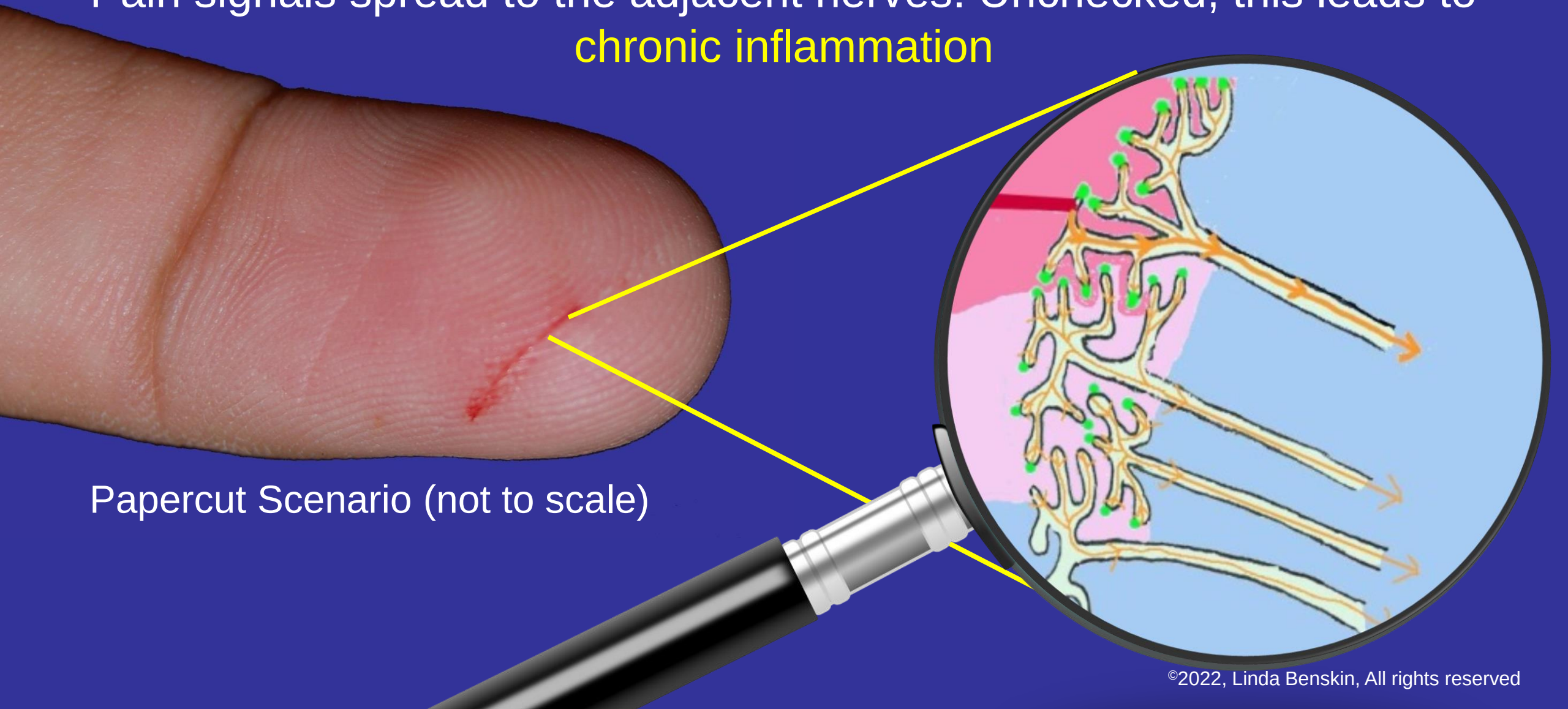
Soon, the ENTIRE nerve is involved ⟶ increased inflammation



Papercut Scenario (not to scale)

Neurogenic (Secondary) Inflammation

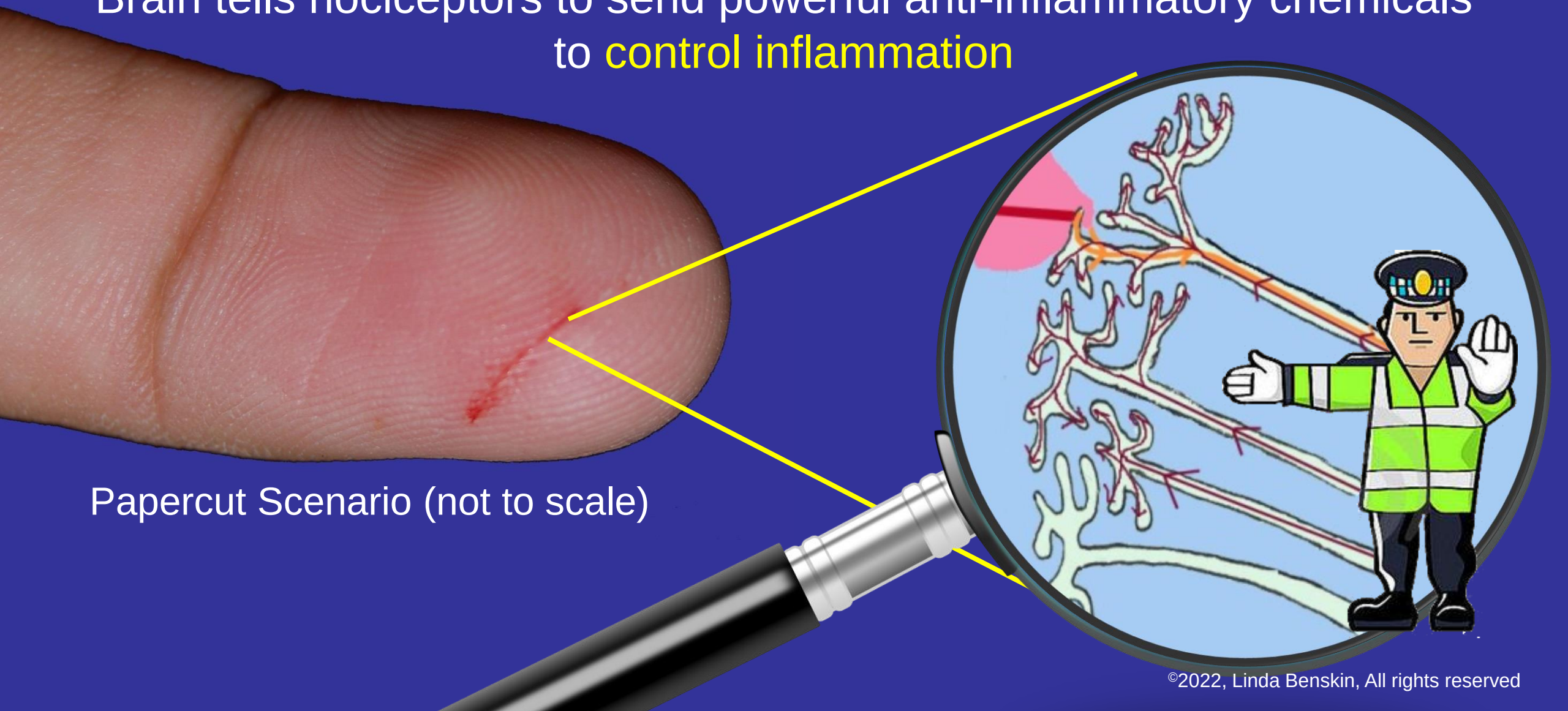
Pain signals spread to the adjacent nerves. Unchecked, this leads to chronic inflammation



Papercut Scenario (not to scale)

Healthy (Focused) Inflammation

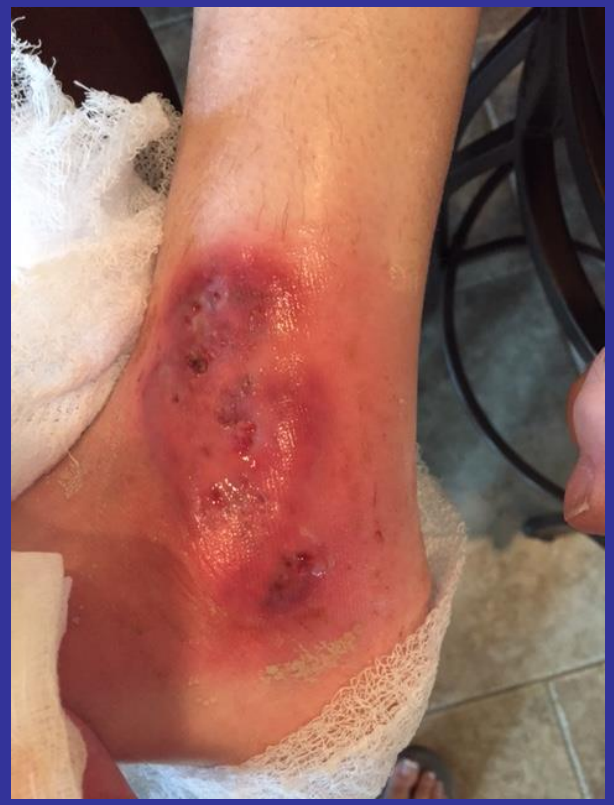
Brain tells nociceptors to send powerful anti-inflammatory chemicals to control inflammation



Papercut Scenario (not to scale)

Classic Signs of Inflammation

a. Redness – from increased circulation
b. Swelling – from capillary leakage
c. Heat – from increased circulation
d. Pain – from nerves being stimulated
e. Immobility – from all of the above



Inflammation must be constantly supported with new resources or it will subside

Outline

Overview

I. Benefits and Disadvantages of Pain

II. How Injury → Inflammation & Pain

III. The Role of the Nociceptors

A. Nociceptor response to injury

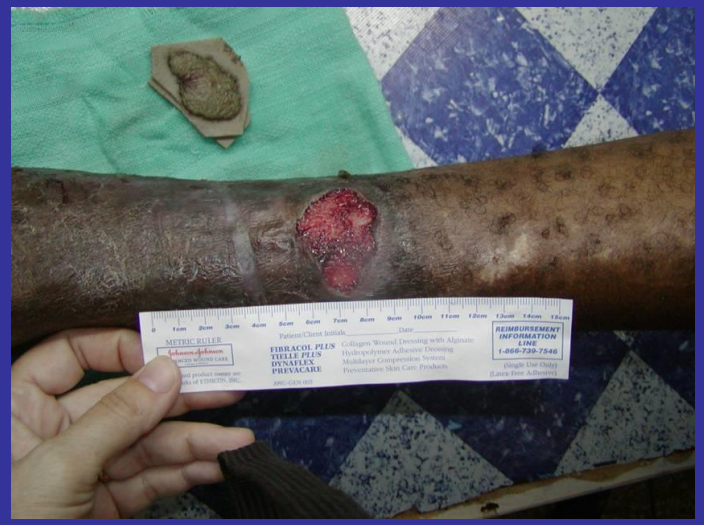
B. How the nociceptor response affects healing

IV. Wound Pain Assessment

V. Interventions to Diminish Wound Pain

1. Chronic Pain & Inflammation leads to Nerve Damage

- Hyperalgesia (helps locate wound and prevent re-injury)
  The nociceptors are extra sensitive
- Allodynia
  Even normally non-painful stimuli are painful
- Neuropathic Pain (or absence of sensation)
  Nerves are actually permanently damaged



Neuropathy can be caused by prolonged pain, but also by ischemia, injury to the nerve itself, or chemical damage (such as from diabetes)

2. Results of Chronic Excess Nociceptor Activity

a. Increases pain to force decreased mobility

   → Hyperalgesia, allodynia, neuropathy

b. Causes edema → secondary ischemic injury

c. Leads to a catabolic state → delays healing

   → Break down tissue, no building or angiogenesis (new blood vessels)

d. Keeps patient sedentary

   → Affects Quality of Life, glycemic control, depression, etc.

e. Increases scarring due to prolonged inflammation

3. Uncontrolled Inflammation Can Cause the Development of Chronic Wounds

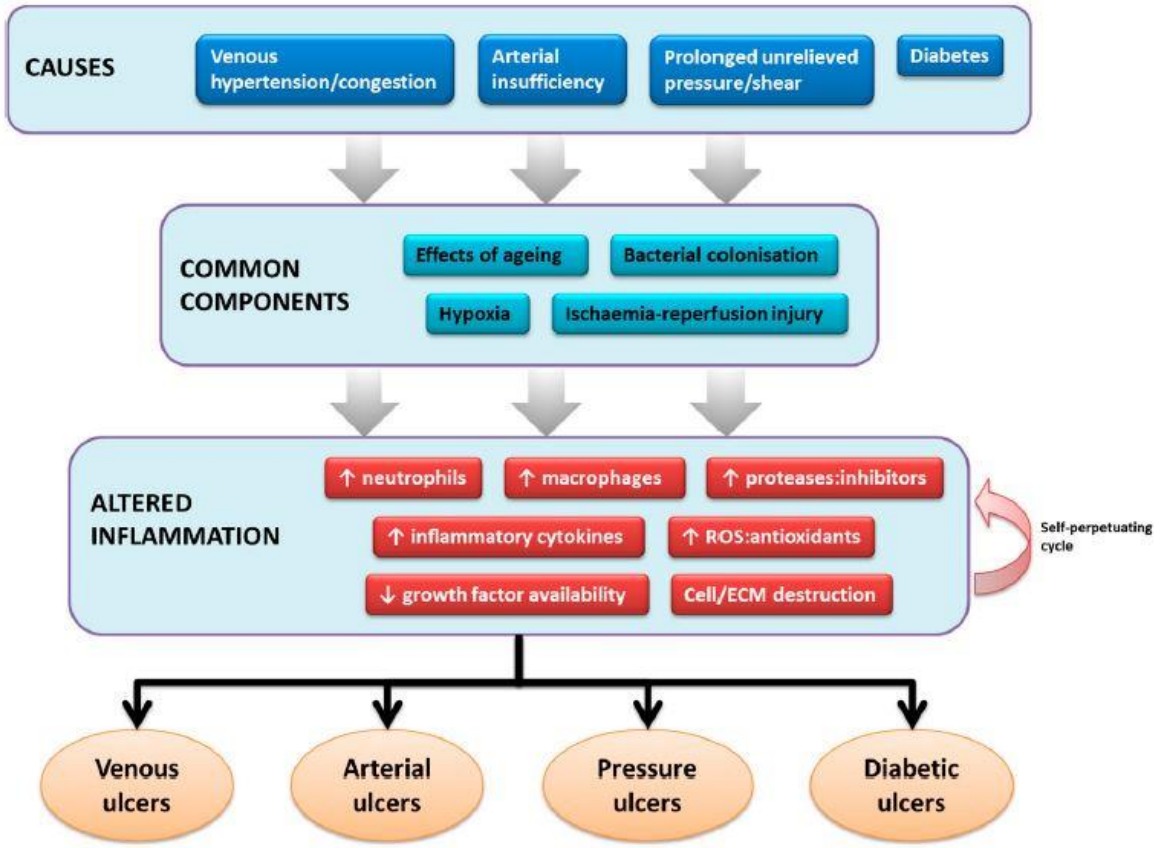
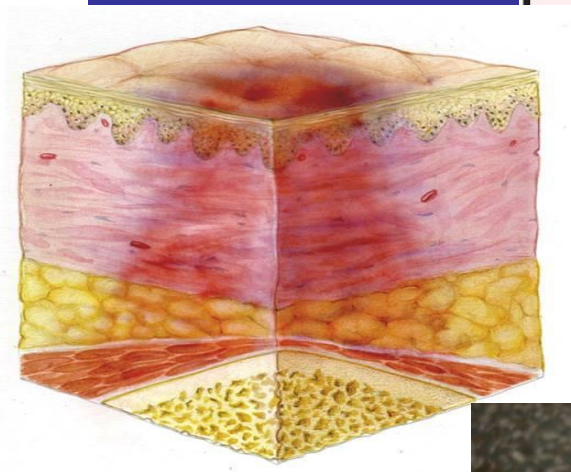
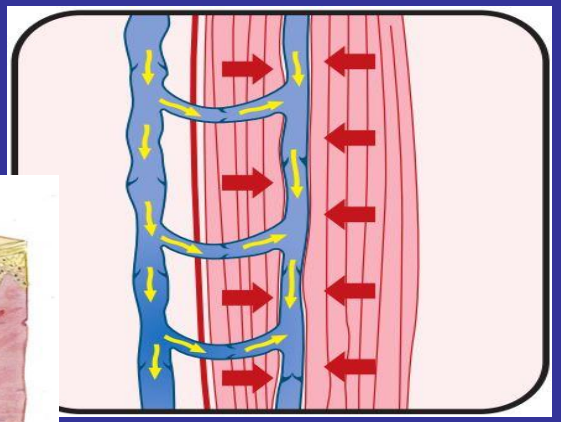


Figure 1. The role of inflammation in the pathophysiology of common chronic wounds. ROS = reactive oxygen species, ECM = extracellular matrix.





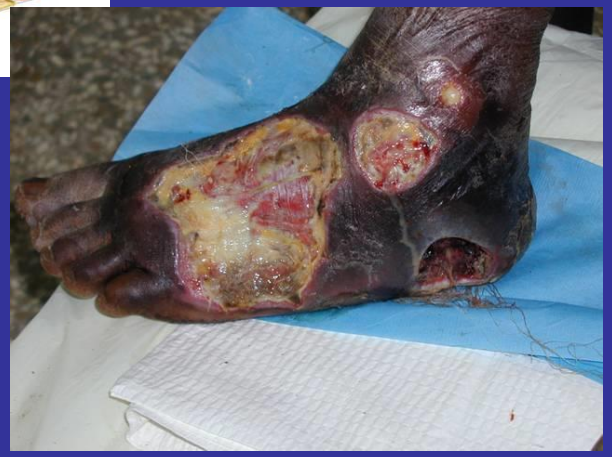
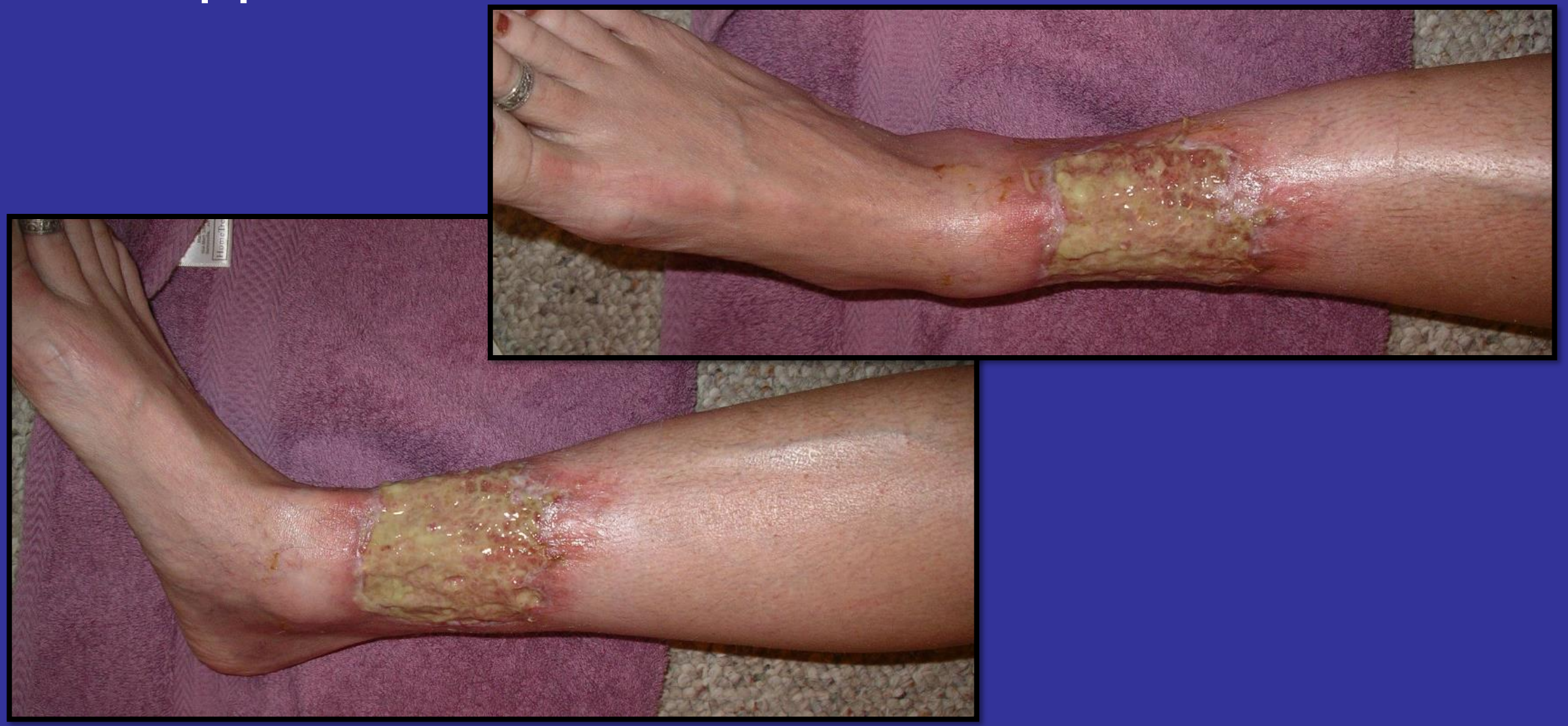


Figure from: Zhao R, Liang H, Clarke E, Jackson C, Xue M. Inflammation in Chronic Wounds. Int. J. Mol. Sci. 2016;17:2085.

Application: Our ONE Patient



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V. Interventions to Diminish Wound Pain

1. Procedural Pain

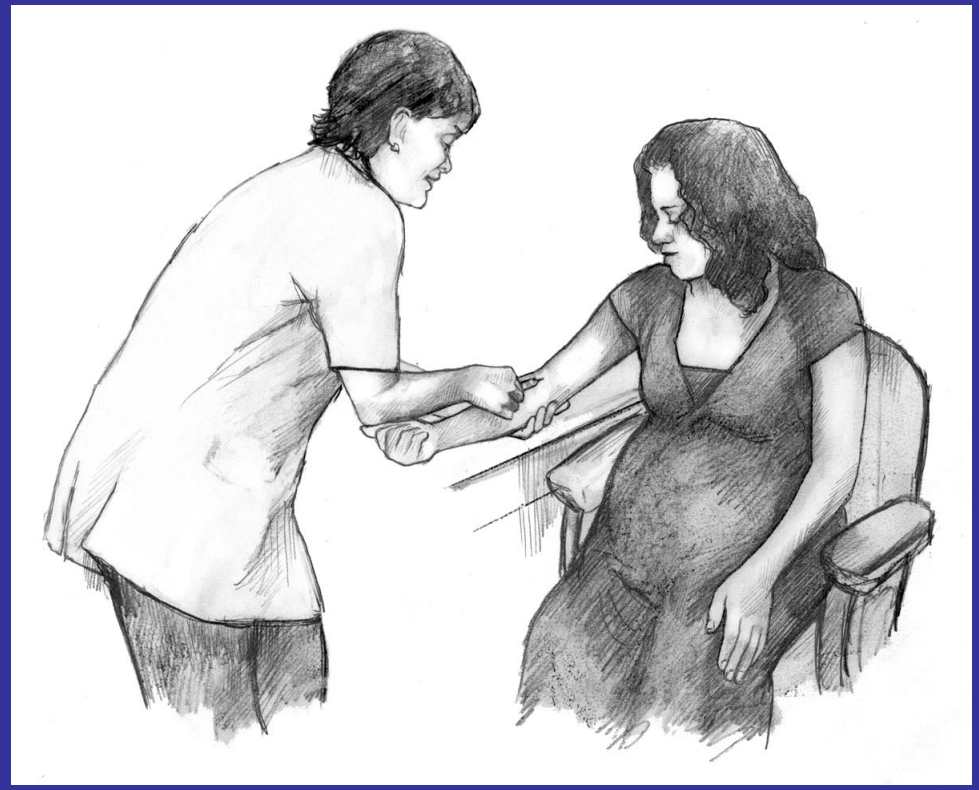
Provide Pre-Op Meds Early If They Will Be Needed

a. Patient handling – be gentle!

- Turning
- Getting out of bed
- Ambulating

Explain, to ↓ anxiety

- Blood draws
- IV starts
- Catheterization



- Splint before eliciting cough & deep breath

1. Procedural Pain – Wound Specific

b. Debridement – pre-op medications???

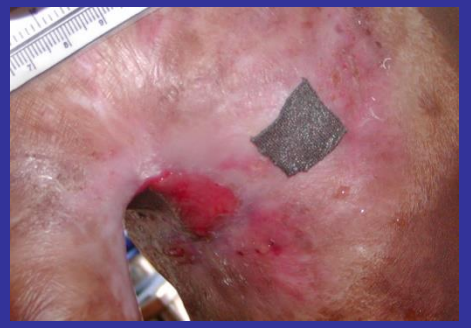
- Mechanical (wet-to-dry, scrubbing) – traumatic! ⟶ infection
+ Conservative sharp – selective IF skilled clinician
+ Autolytic is least painful, most selective – must protect periwound

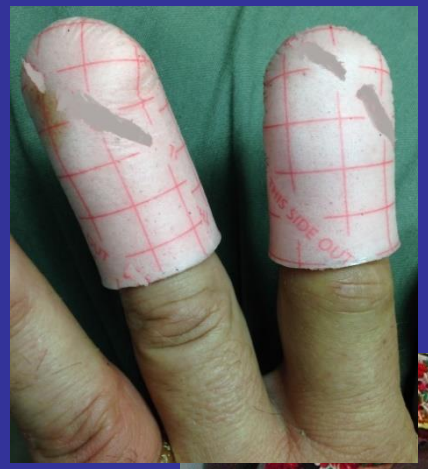
  Moderate immunosuppression is NOT a contraindication

+ Polymeric membrane dressings and honey augment autolytic
- Larval – sometimes painful, $, not faster, removes some bacteria
- Enzymatic – selective, $, not faster, often really autolytic

1. Procedural Pain – Wound Specific

c. Dressing changes





- + Nonadherent, moist dressings
- + AVOID in-growth of tissue!
- + Prevent Medical Adhesive-Related Skin Injury:
  - + Removal techniques (push-pull, parallel)
  - + Use skin barriers or adhesive removers
  - + Stretch netting, special dressing configurations
  - + Special tapes (silicone, cloth stretch)
  - + modern “Montgomery straps” with adhesive film

1. Procedural Pain (Wound Specific)

d. Wound Cleansing: Gently!

- Major stressor for patients

Initially:

+ Irrigation (4 - 15 PSI) with LOTS of fluid – until it runs clear
+ Warm saline, water, or a non-toxic cleanser
- NOT scrubbing! Even wiping can cause or spread infection

At dressing changes:

+ Rinse only if needed, using above methods
+ Use dressings that do not leave contaminants behind
- Rinsing washes away nutrients, cools, triggers hyperalgesia

2. Activity Pain

Assess/Advise: Is the pain protective?

a. Measure: How far can the patient walk?

Edema decreases with walking

b. Compression decreases causes of pain

c. Walking even helps with PAD pain

d. Patients with DFUs: offload and walk

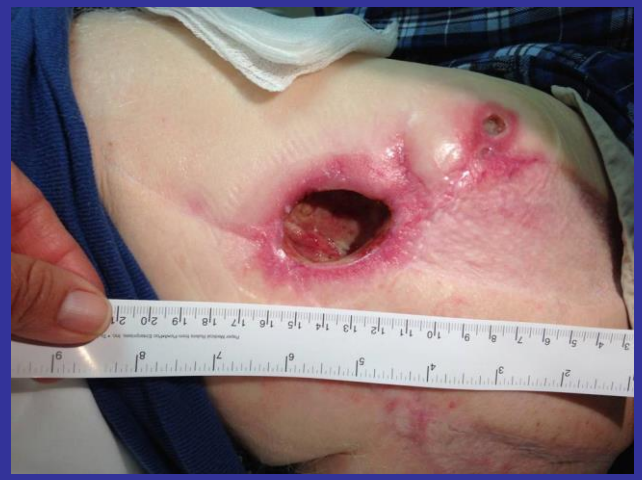
e. Assess all diabetic patients for

Peripheral Neuropathy (Ipswich Touch)

Peripheral Arterial Disease (PAD) - ABIs

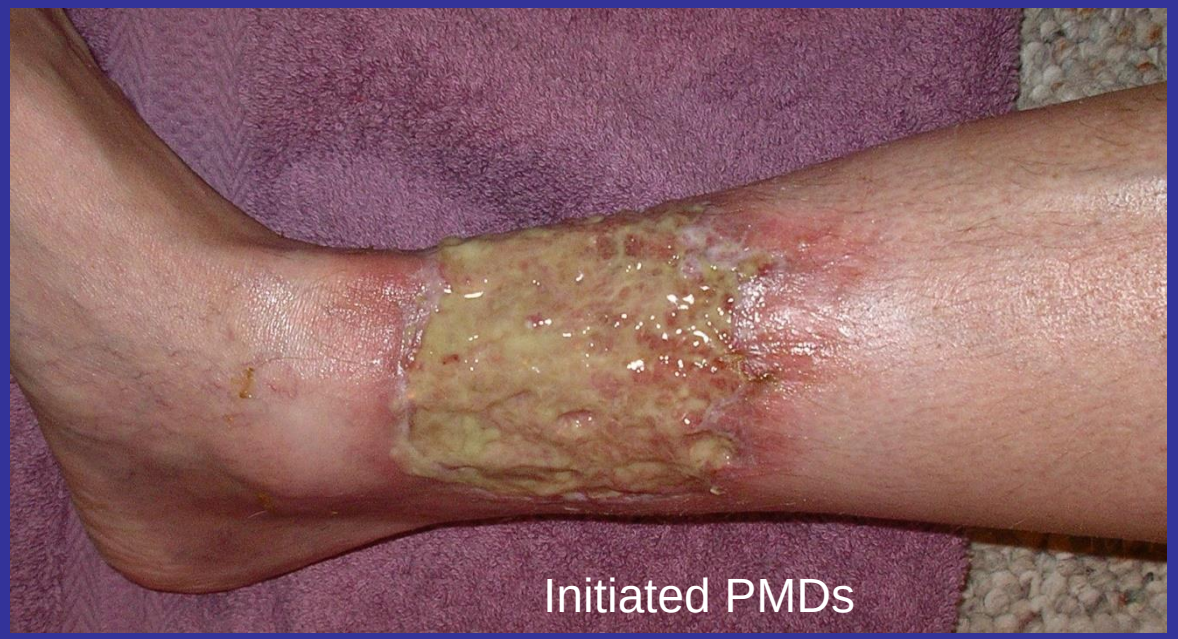
3. Persistent (Continuous, Background) Pain

a. Address Desiccation, Pressure, & Inflammation
b. Evaluate/address Infection
c. Avoid Painful Treatments
    i. NPWT
    ii. Larvae
    iii. Honey

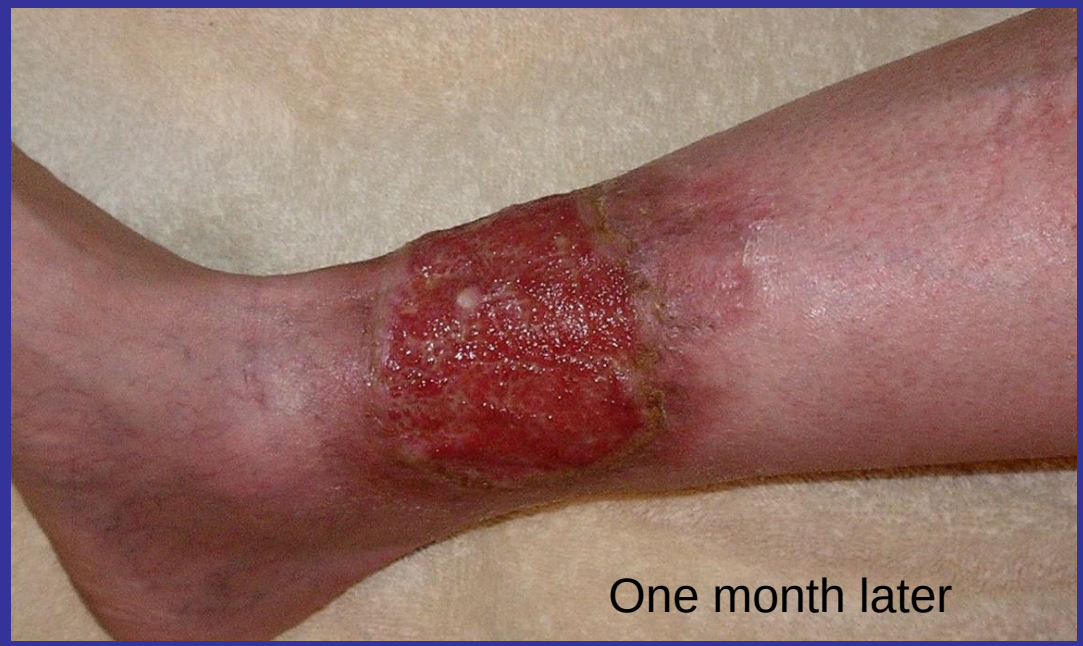


ONE Patient: Decrease Procedural Pain from Wound Cleansing

Compare procedural pain with these two methods:



Very painful soaking; no improvement



No cleaning, minimal pain, closing

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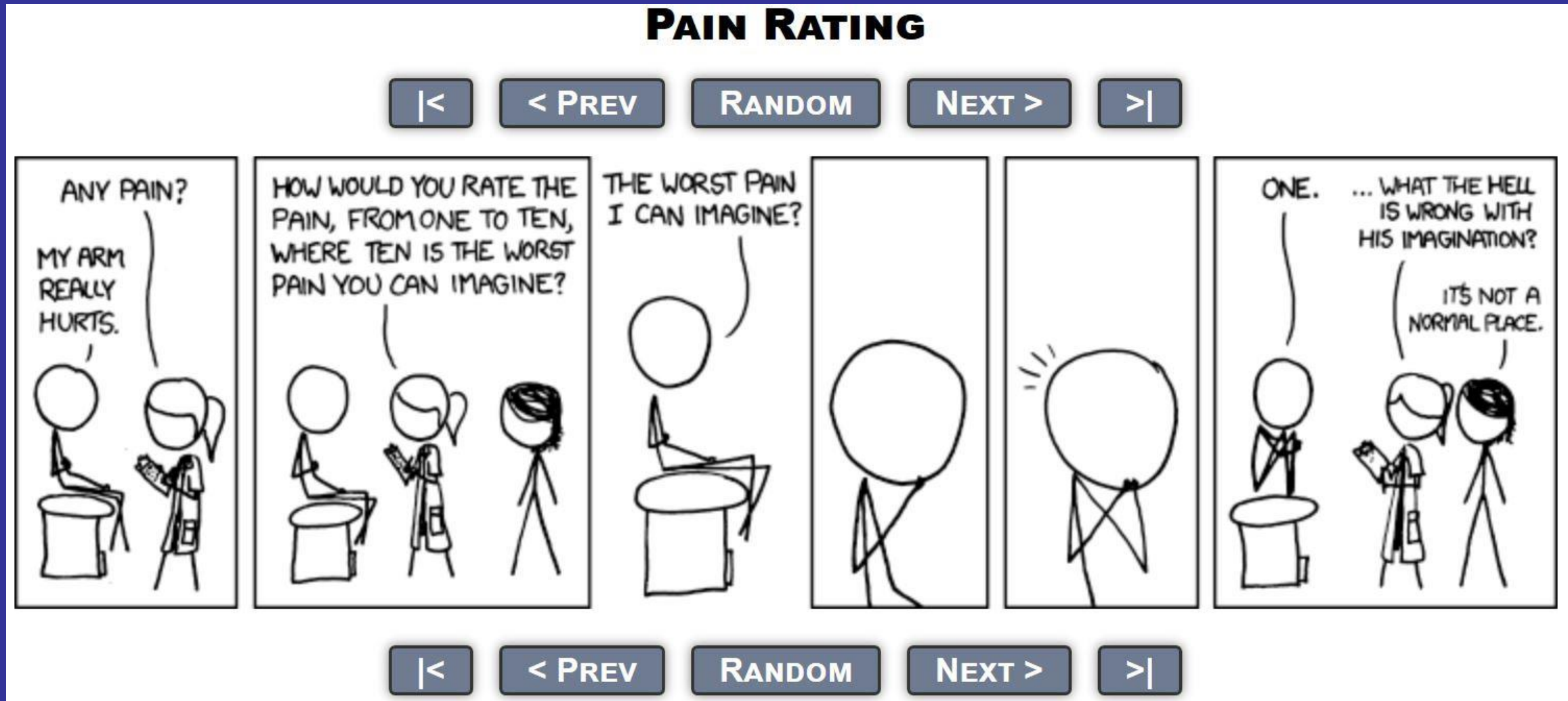
C. Subjective Quantitative Assessment

2.b. Validated self-report scales, usually 0 – 10

2.d. Faces Pain Scale-Revised (below) from IASP de-emphasizes

1. Never Compare One Patients' Pain Scores with Another's



2.e.,f. Subjective Quantitative Assessment

Subjective validated scales, 0 – 10 no self-report

The Pain Assessment in Advanced Dementia (PAINAD) Scale*

Items	0	1	2	Score
Breathing independent of vocalization	Normal	Occasional labored breathing. Short period of hyperventilation.	Noisy labored breathing. Long period of hyperventilation. Cheyne-Stokes respirations.	
Negative vocalization	None	Occasional moan or groan. Low-level speech with a negative or disapproving quality.	Repeated troubled calling out. Loud moaning or groaning. Crying.	
Facial expression	Smiling or inexpressive	Sad. Frightened. Frown.	Facial grimacing.	
Body language	Relaxed	Tense. Distressed pacing. Fidgeting.	Rigid. Fists clenched. Knees pulled up. Pulling or pushing away. Striking out.	
Consolability	No need to console	Distracted or reassured by voice or touch.	Unable to console, distract or reassure.	
			Total	

**Warden V, Hurley Ac, Volicer L. Development and psychometric evaluation of the pain assessment in advanced dementia (PAINAD) scale. J Am Med Dir Assoc. 2003; 4:9-15.*

FLACC Scale	Score
Face 0 - No particular expression or smile 1 - Occasional grimace or frown, withdrawn, disinterested 2 - Frequent to constant quivering chin, clenched jaw	
Legs 0 - Normal position or relaxed 1 - Uneasy, restless, tense 2 - Kicking, or legs drawn up	
Activity 0 - Lying quietly, normal position, moves easily 1 - Squirming, shifting back and forth, tense 2 - Arched, rigid or jerking	
Cry 0 - No cry (awake or asleep) 1 - Moans or whimpers, ocassional complaint 2 - Crying steadily, screams or sobs, frequent complaints	
Consolability 0 - Content, relaxed 1 - Reassured by occasional touching, hugging or being talked to, distractible 2 - Difficult to console or comfort	
Total Score	

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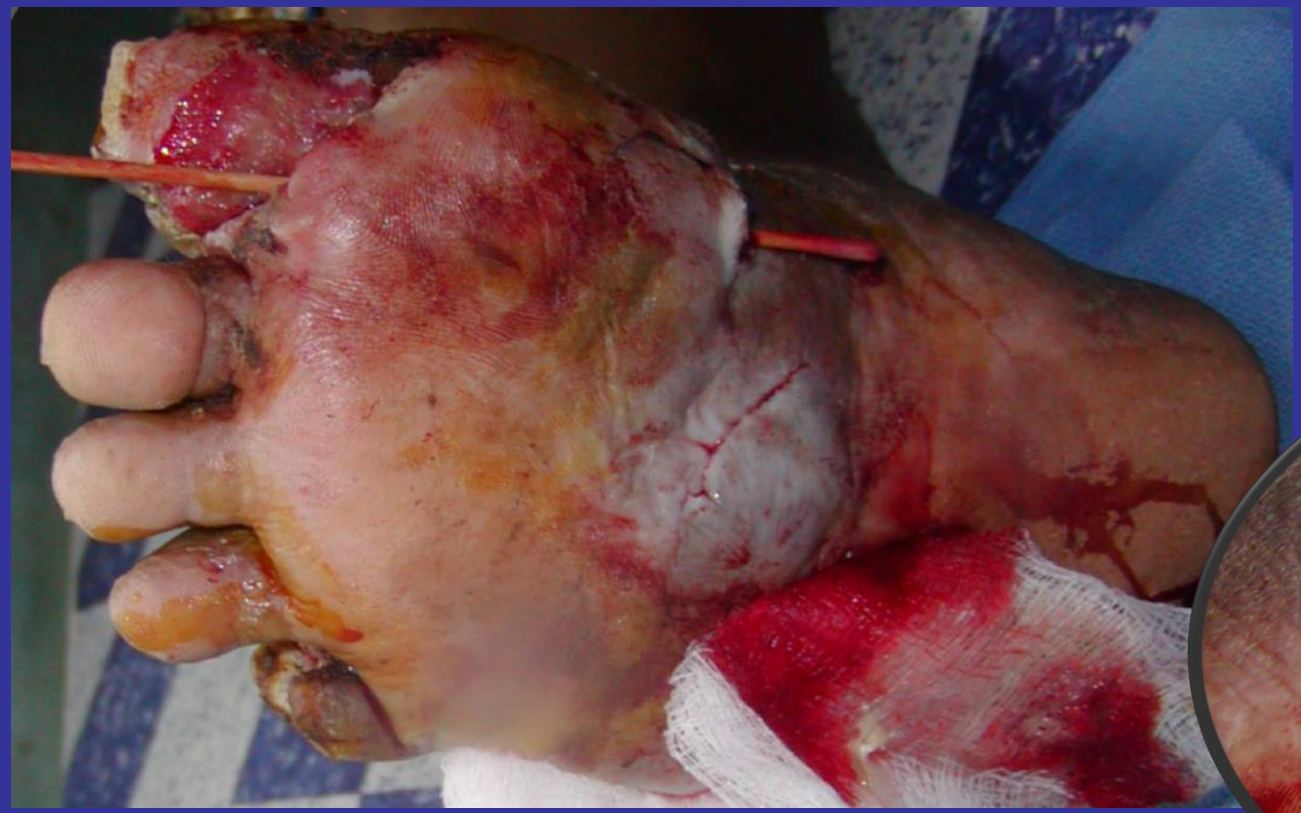
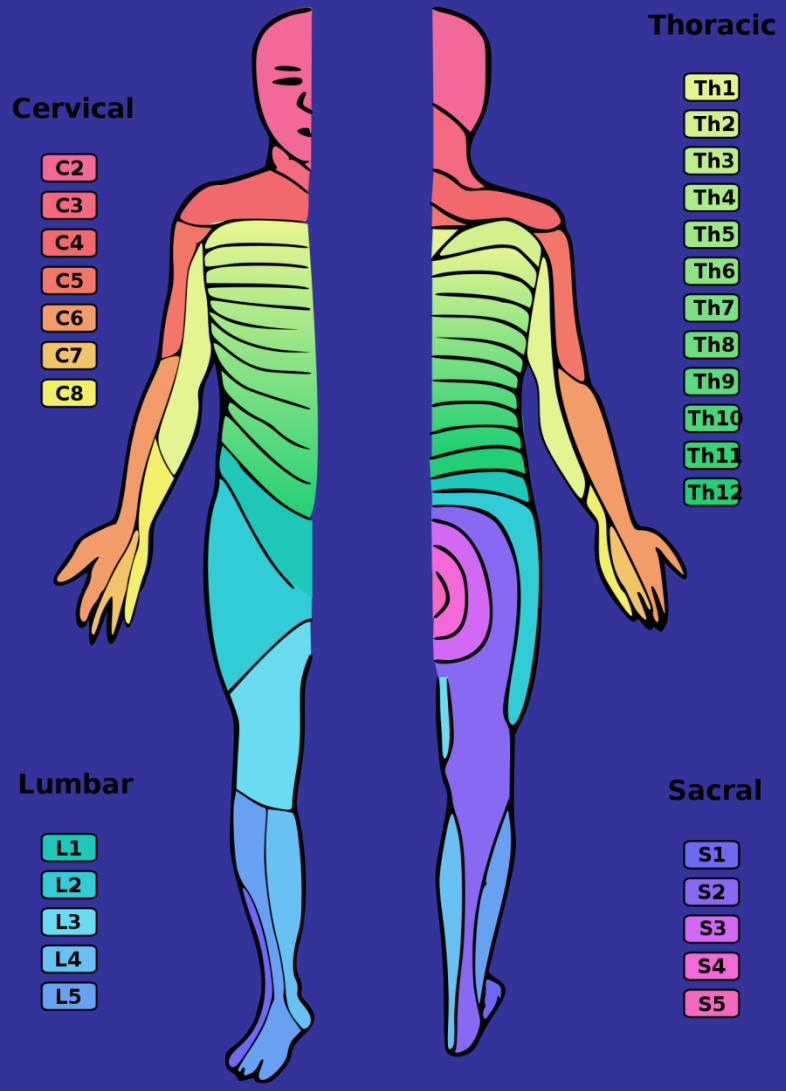
D. Critical Attributes of the Pain

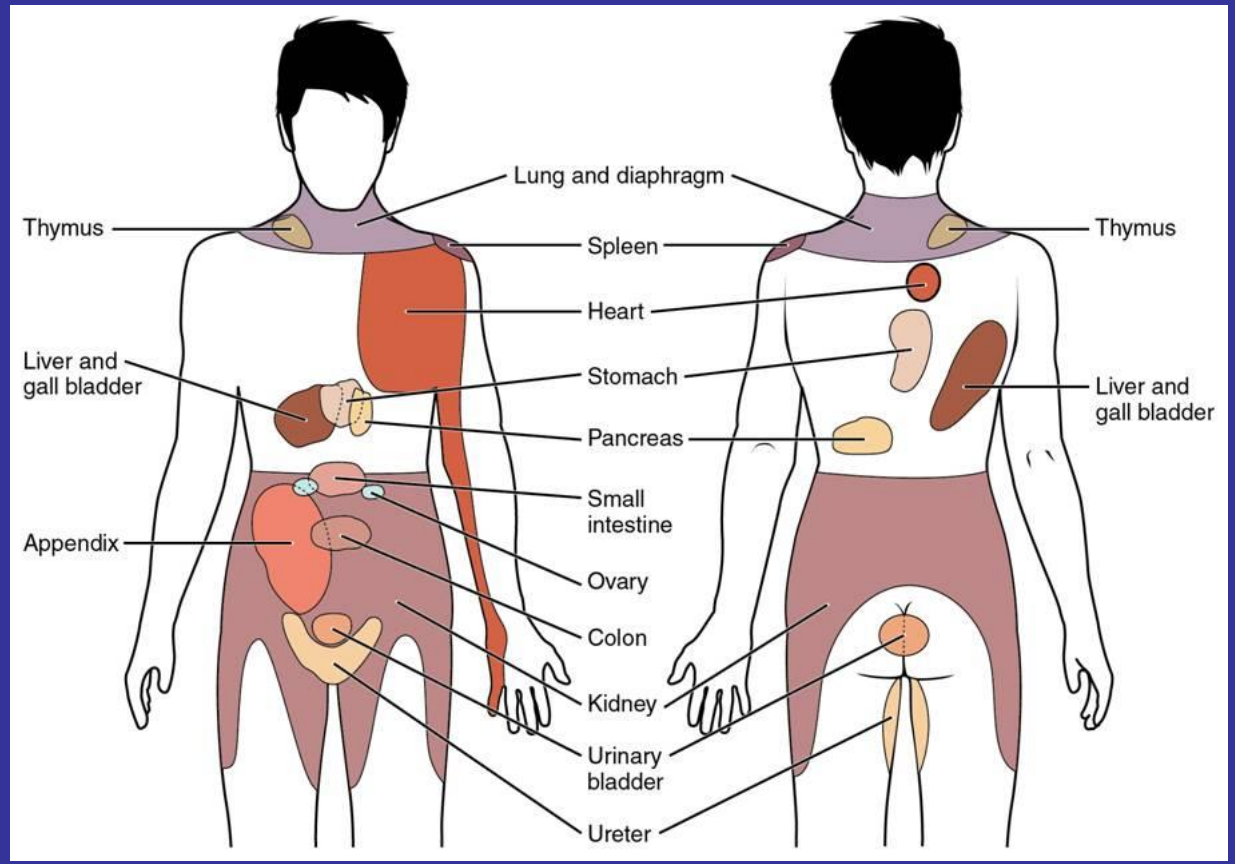
1. Location: document each area of pain individually
2. Quality or Character: free response (sharp, burning…)
3. Chronology: initial, worst, continuous or intermittent?
4. Precipitating and alleviating factors:
5. did intervention (medication) help?
6. Cultural/experiential aspects; depression…
   expectations, meaning of pain, consequences

5. Cultural Influencers of Pain Perception



E. Dermatomes, Referred Pain, & Radiating Pain





Left: Ralf Stephan © User:Mono / Wikimedia Commons, a sister project of Wikipedia https://commons.wikimedia.org/wiki/File:Dermatoms_(re-labeled).svg Above: Autonomic Reflexes and Homeostasis http://cnx.org/content/m46579/1.2/ OpenStax College https://commons.wikimedia.org/wiki/File:1506_Referred_Pain_Chart.jpg

Outline

Overview

I. Benefits and Disadvantages of Pain

II. How Injury → Inflammation & Pain

III. The Role of the Nociceptors

IV. Wound Pain Assessment

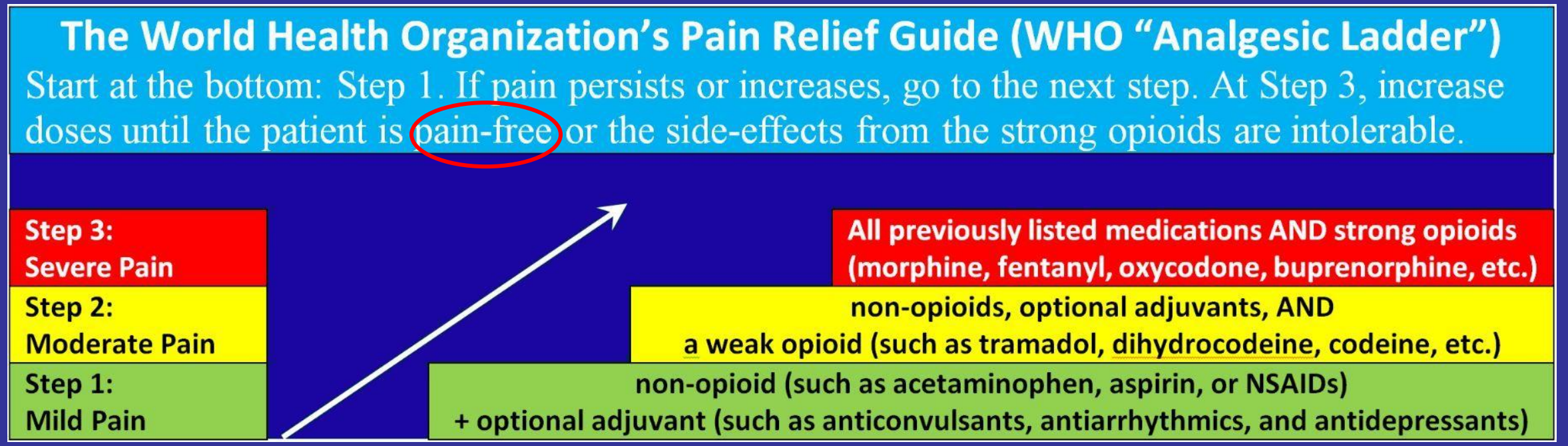
V. Interventions to Diminish Wound Pain

   A. Fundamentals

   B. Topical (Medications, Dressings)

   C. Non-Topical (Medications, Alternatives)

1986: Current “Standard of Care”?

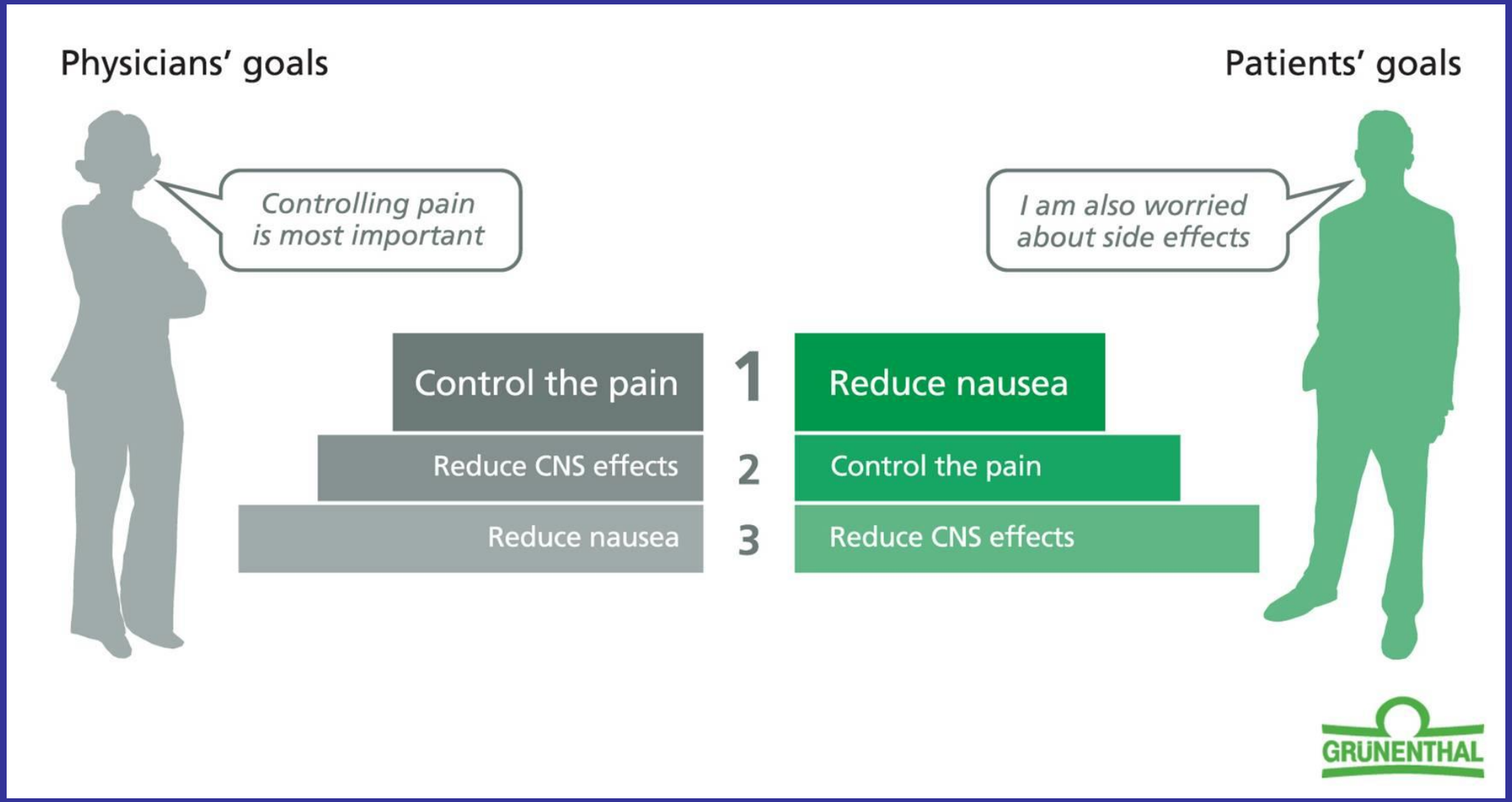


Goal is ZERO pain, not improved function

Non-pharma pain relievers excluded, & opioids are king

Repeated revision attempts… but no new guidelines yet

1. Patient-Centered GOALS



A. Goals, Principles, & Fundamentals

2. MOST research is on procedural pain
3. Nurses may feel powerless to address pain
4. Pain is made worse by

   Fear, anxiety, sleep deprivation, depression

   Trust decreases fear & anxiety

   Exercise helps with sleep & depression

5. Brief Motivational (Negotiated) Interviewing: BMI or BNI

   Facilitate behavior change (non-paternalistic): express empathy, develop discrepancy, avoid argumentation, roll with resistance, support self-efficacy

6. The Gate Control Theory of Pain

Light traffic: easy access and speedy travel

Heavy traffic: less access and slower travel

The brain can be "trained" to turn off pain that is not "useful"

“PAIN IS IN THE BRAIN” - Melsack (& Wall)

7. Before Pain Interventions: Address *Each* Cause

a. Inflammation
b. Hypoxia
c. Infection
d. Maceration
e. Neuropathy
f. Procedural pain

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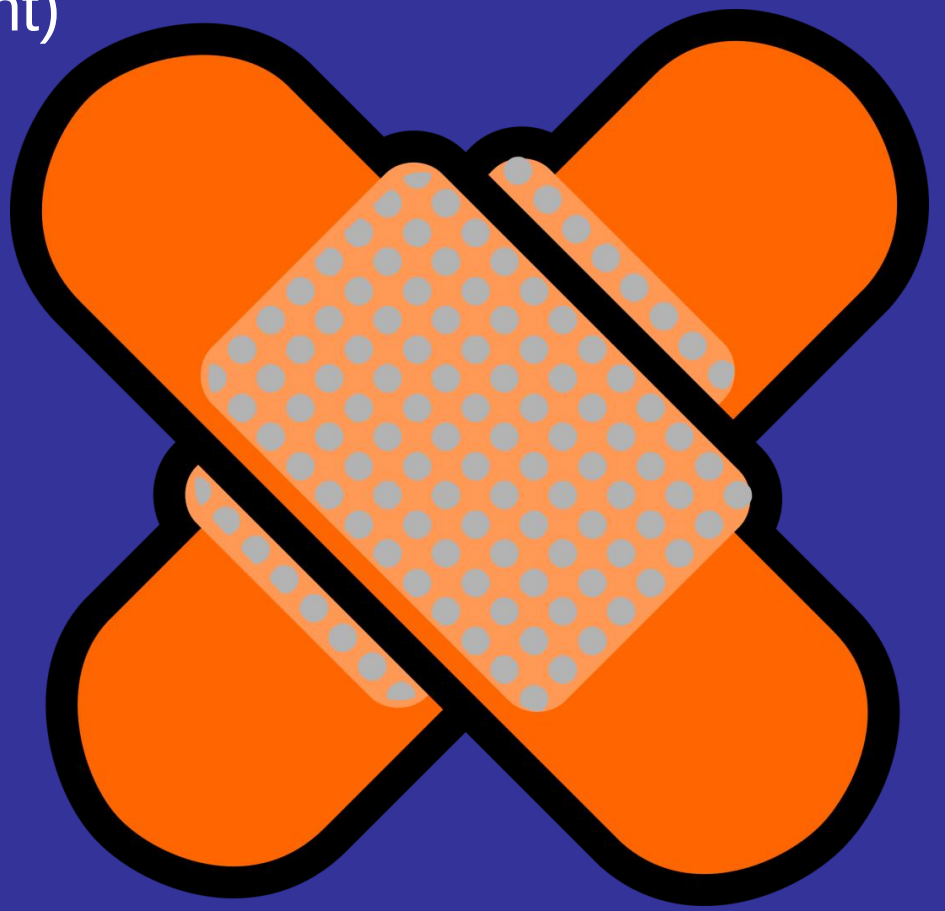
B.1. Topical Interventions: Medications

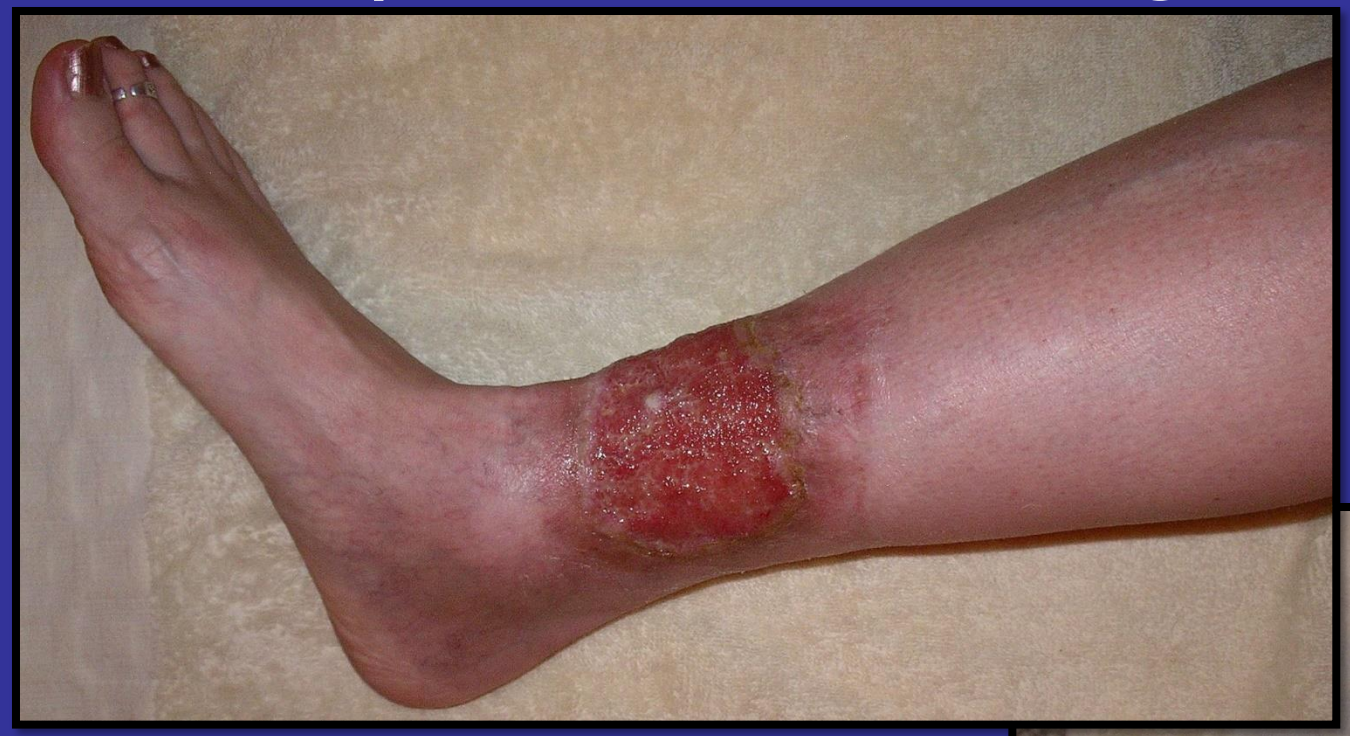
a. Lidocaine, etc.
b. NSAIDs
c. Morphine
d. Capsaicin
e. Herbals such as aloe vera, turmeric
f. Menthol: Near, NOT IN, open wound
g. Nitroglycerin ointment: Near, NOT IN, open wound
h. Laser acupuncture (red & infrared)

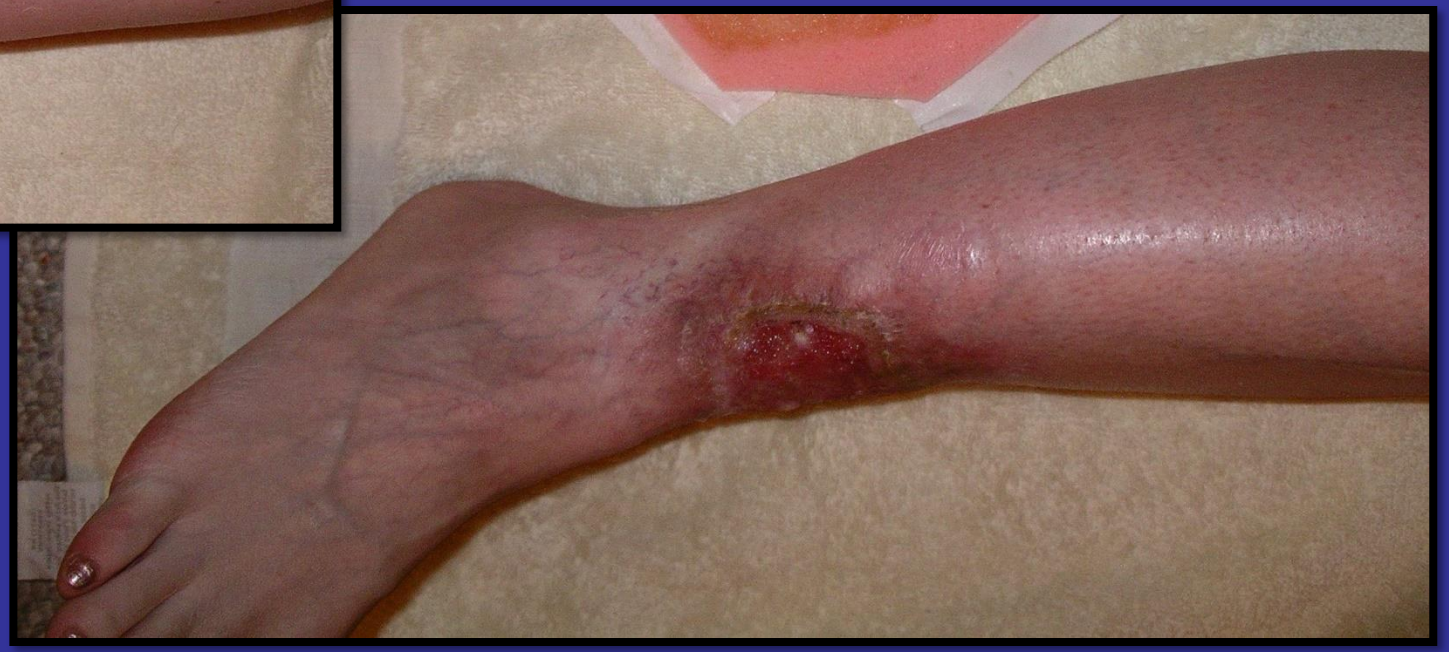
B. 2. Topical Interventions: Dressings

a. Occlusive or semi-occlusive (hydrogels, hydrocolloids, foams)
   (soothe raw nerve endings; moisture is important)
b. Polymeric membrane dressings (PMDs)
   (subdue & focus the nociceptor response)
c. Non-adherent
   (decreases procedural pain)
d. Compression
   (especially if pain is caused by edema, but…)
e. Antimicrobial
   (when infection is a cause of pain)



Topicals & Dressings Can Decrease Pain





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C. 1. Systemic Medications (all have side effects)

a. - Opioids: poor efficacy, may slow healing

b. - Prescription anti-inflammatories also slow healing

c. + Over-the-counter (try alternating) (Can replace 7p Ibuprofen with naproxen sodium and skip the 1a NSAID dose.)

d. +/- Specific Medications for Neuropathy

- Antidepressants (e.g., amitriptyline)
- Antiepileptics (e.g., gabapentin)

e. - CBD: pain worse or same in quality RCTs

C. 2 - 4.Alternative Interventions

2. Gate Control Implementation
   a. Distraction: music, TV, imagery, video games, sweets, scents, etc.
   b. Acupuncture, stroking or pinching lightly
   c. Electrical stimulation (TENS, others)
3. Increasing patient control

   Self care or time outs
4. Exercise!!! (CALF if unable to walk)

Avoid Trade-Offs

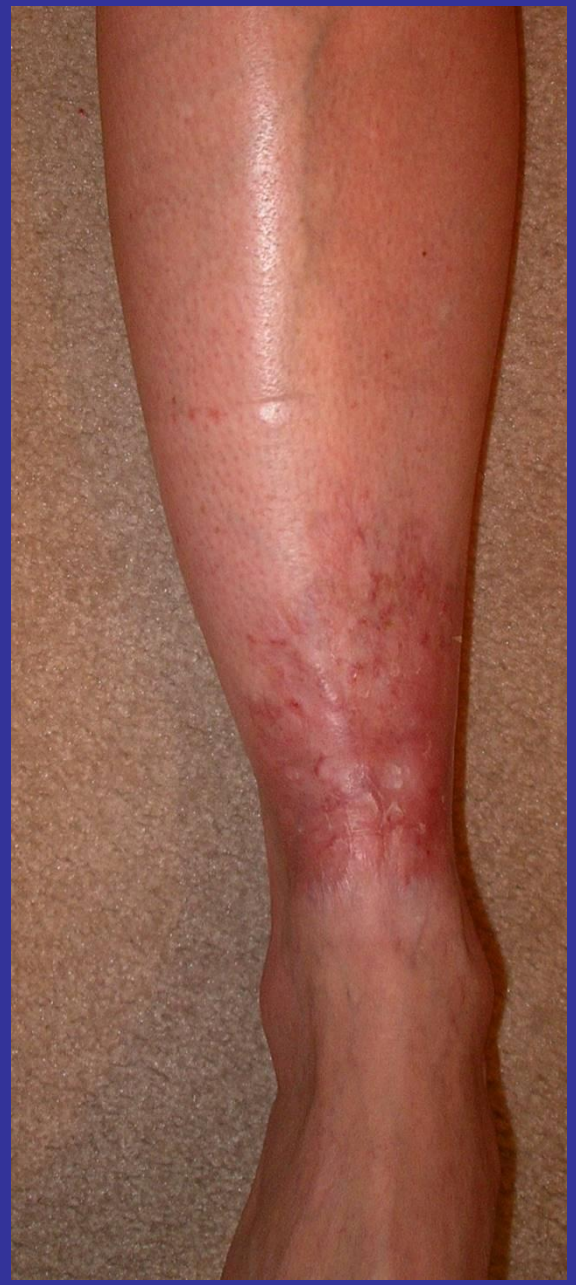
Silver cytotoxicity vs infection:
which will decrease healing more?

NSAIDs inhibit healing vs pain:
which will decrease healing more?

Try to choose solutions that are WIN – WIN!

- Irrigate wound well initially
- Use other pain-reducing solutions

Image from fotolia by Adobe



Final Look at Your ONE Patient

Thorough assessment?

What (all) is causing the pain?

Neurogenic inflammation?

Address causes!!!

Add direct interventions:

- Topical or systemic medications
- Gate theory methods
- Increased patient control
- Exercise

Summary

- The pain/inflammation cycle is usually adaptive
- Inappropriate pain/inflammation can slow or stall healing
- Health Care Professionals such as Nurses can intervene

Extensive references are in the handout

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