WOUND CARE: 101

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WARNING TO AUDIENCE

Disclaimer : Please be aware this presentation contains graphic photos that may not be suitable to those enjoying breakfast

Disclosures:

No relevant financial relationships with commercial interests to disclose.

GOALS

Understand terminology

Identify wound types

Understand algorithms of management

Learn about prevention & treatment modalities

WHY DO YOU NEED TO KNOW ABOUT IT?

Significant patient population

Significant morbidity/mortality

Quality of life

Economic burden

Waste reduction

Chronic Wounds…A Silent Epidemic

Rarely seen in individuals who are otherwise healthy

Frequently suffer from “highly branded” diseases such as diabetes and obesity (comorbid conditions)

This seems to have overshadowed the significance of wounds per se as a major health problem

Represent a silent epidemic that affects a large fraction of the world population and poses major and gathering threat to the public health and economy of the United States

A 2018 retrospective analysis of Medicare beneficiaries identified

8.2 million people had wounds with or without infections

$28.1 billion to $96.8 billion

Highest expenses were for surgical wounds followed by diabetic foot ulcers

The annual wound care products market is expected to reach $15–22 billion by 2024.

The National Institutes of Health’s (NIH) Research Portfolio Online Reporting Tool (RePORT) now

Cost is growing rapidly due to:

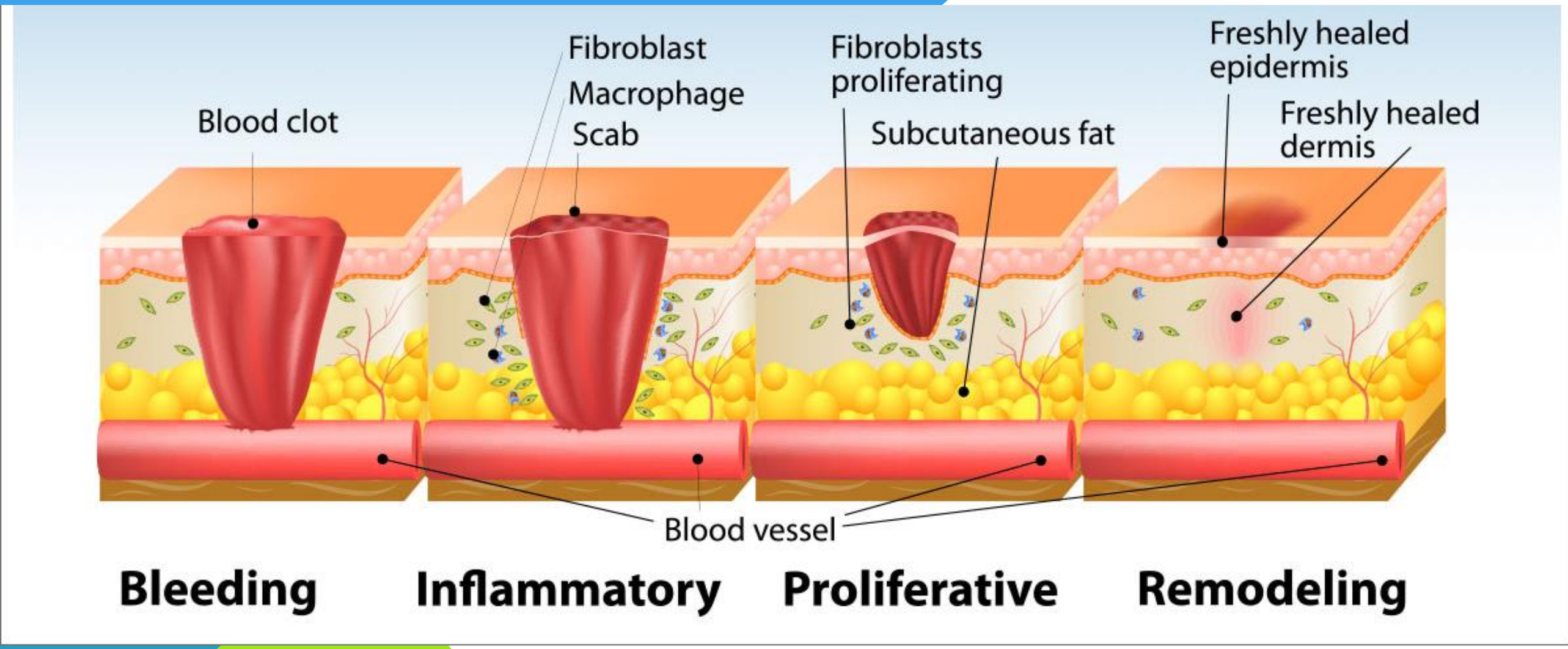
increasing health care costs

aging population

sharp rise in the incidence of diabetes and obesity

Skin disorders account for 39 billion dollars of lost wages and medical care involving 65 million Am

4 PARTS OF NORMAL WOUND HEALING



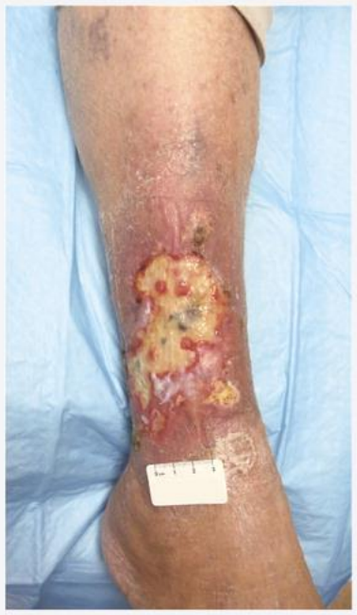
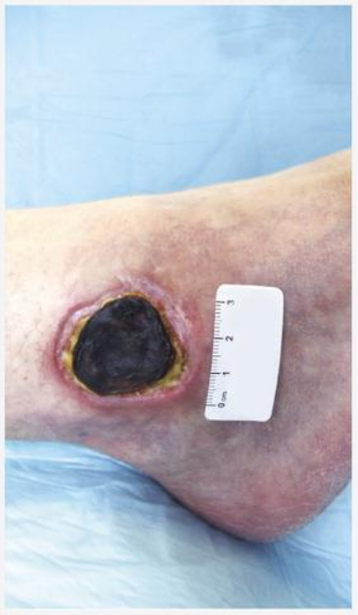
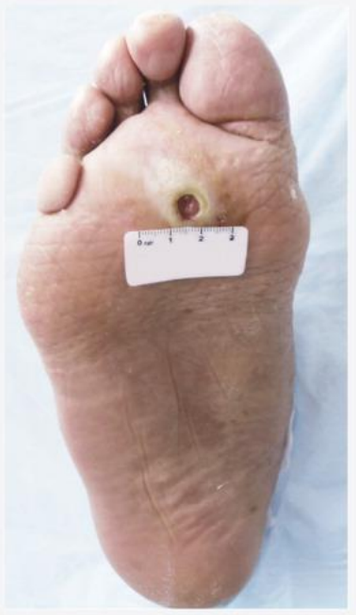
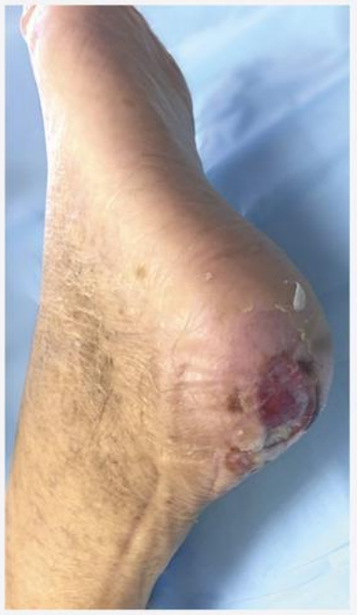
CHRONIC WOUNDS

A wound that has failed to proceed through an orderly and timely reparative process to produce anatomic and functional integrity of the injured site.

A wound becomes an ulcer after 3 weeks.

What type of wound is this?

venous arterial diabetic pressure

Feature	Ulcer Type			
	Venous	Arterial	Neuropathic Diabetic	Pressure
Underlying condition	Varicose veins, previous deep-vein thrombosis, obesity, pregnancy, recurrent phlebitis	Diabetes, hypertension, smoking, previous vascular disease	Diabetes, trauma, prolonged pressure	Limited mobility
Ulcer location	Area between the lower calf and the medial malleolus	Pressure points, toes and feet, lateral malleolus and tibial areas	Plantar aspect of foot, tip of the toe, lateral to fifth metatarsal	Bony prominences, heel
Ulcer characteristic	Shallow and flat margins, moderate-to-heavy exudate, slough at base with granulation tissue	Punched out and deep, irregular shape, unhealthy wound bed, presence of necrotic tissue, minimal exudate unless infected	Deep, surrounded by callus, insensate	Deep, often macerated
				
Condition of leg or foot	Hemosiderin staining, thickening and fibrosis, eczematous and itchy skin, limb edema, normal capillary refill	Thin shiny skin, reduced hair growth, cool skin, pallor on leg elevation, absent or weak pulses, delayed capillary refill, gangrene	Dry, cracked, insensate, calluses	Atrophic skin, loss of muscle mass
Treatment	Compression therapy, leg elevation, surgical management	Revascularization, antiplatelet medications, management of risk factors	Off-loading of pressure, topical growth factors	Off-loading of pressure; reduction of excessive moisture, shear, and friction; adequate nutrition

https://resident360.nejm.org/clinical-pearls/evaluation-and-management-of-lower-extremity-ulcers

VENOUS ULCERS

70-90% of all Lower Extremity Wounds.

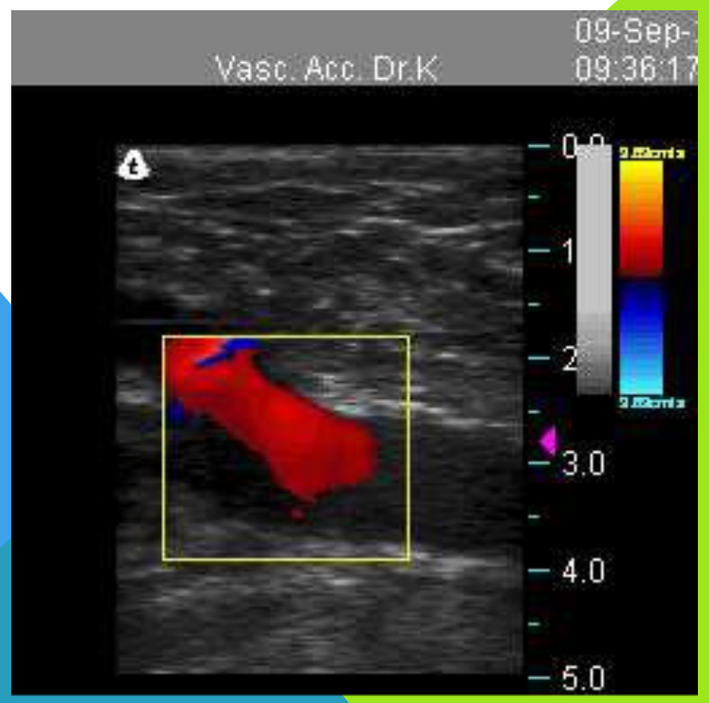
Up to one-third of treated patients experience four or more episodes of recurrence (60-70 % within 5 years).

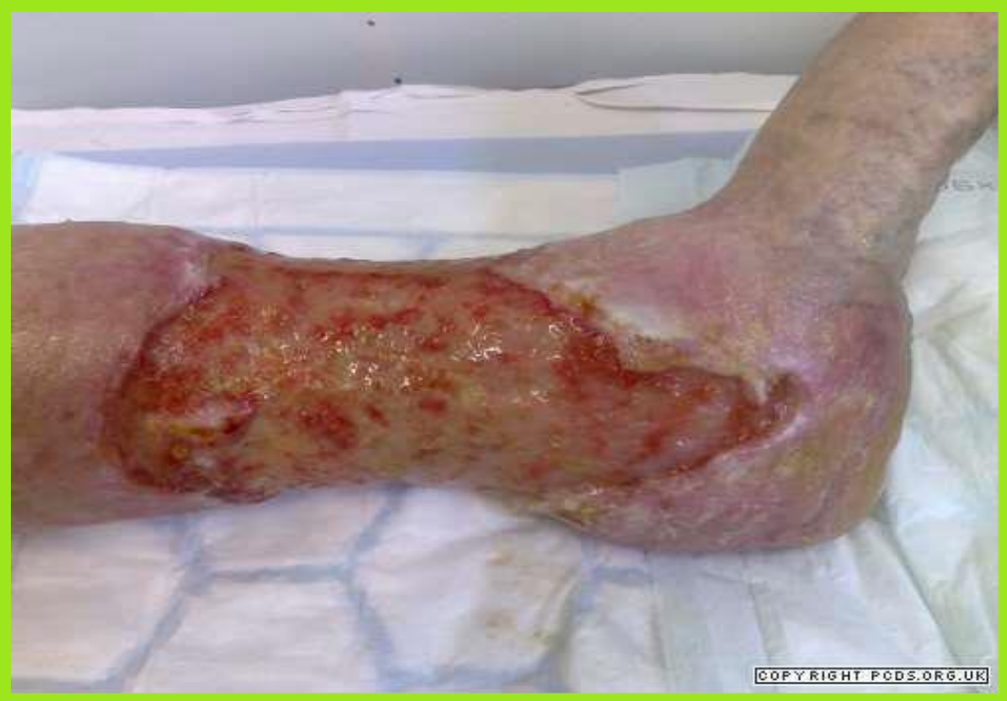
Prevalence in USA - 600,000 annually.

Annual cost of treating VLU in the US - $2.5-3.5 billion.

Compliance is difficult.

>50% take over 6 mo to heal.





VENOUS ULCERS

81% believe that their mobility is adversely affected

73% disturbed sleep

68% reported that the ulcer had a negative emotional impact on their lives, including feelings of fear, social isolation, anger, depression, and negative self-image

58% found caring for the ulcer burdensome

50% mood affected.

Younger working patients, leg ulceration correlated with time lost from work, job loss, and adverse effects on finances

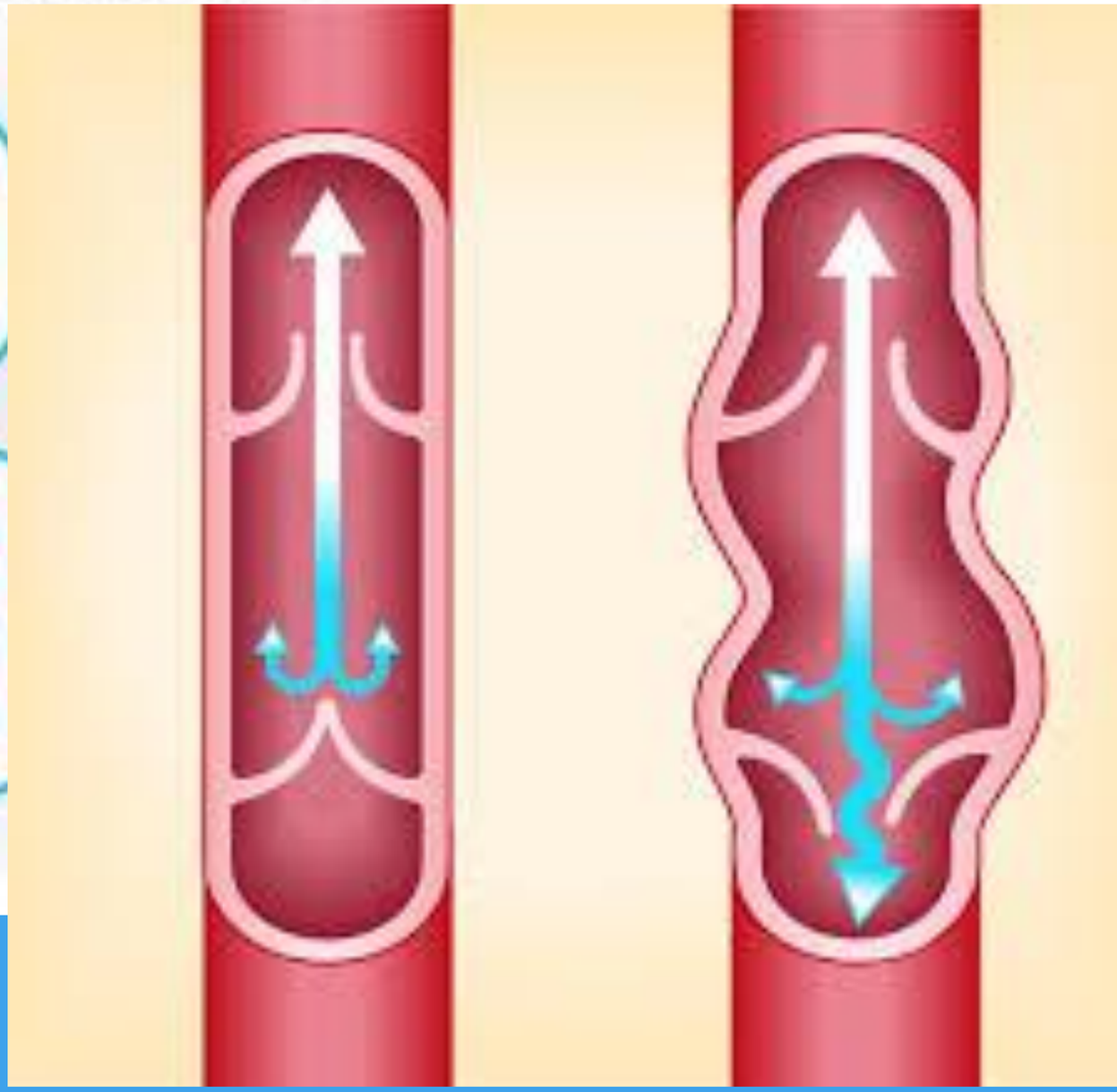
Persistent pain and pain at dressing changes results in lower activity, depression, irritation and reduced social activity

Early retirement is found in up to 12.5% of workers with venous ulcers

Cost 2 billion dollars annually to lost wages an work days

Chronic venous insufficiency

A condition where the walls and valves of the vein do not function effectively



https://texasheartandvein.com/varicode-veins-venous-insufficiency/

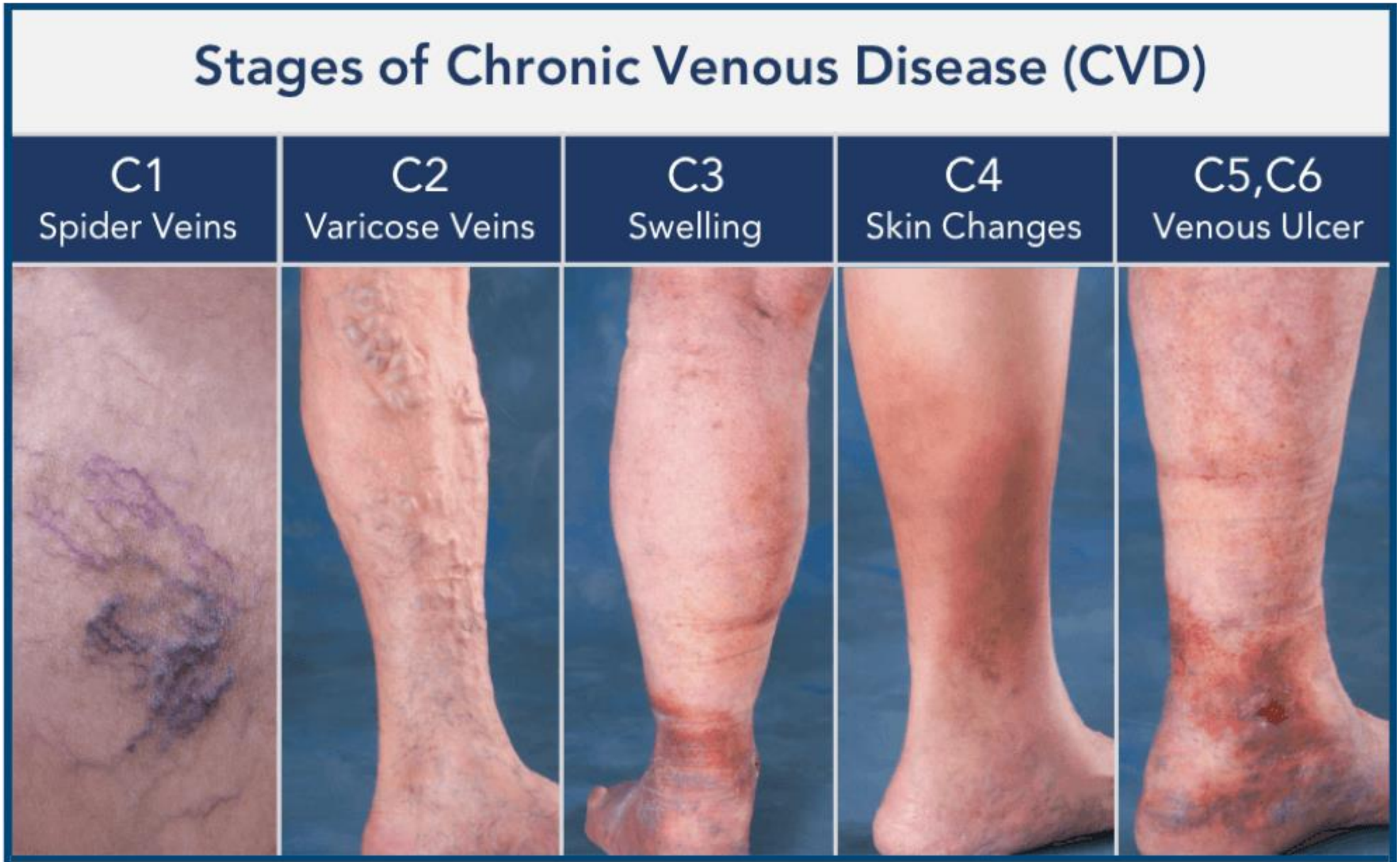

Stages of Chronic Venous Disease (CVD)
C1
Spider Veins
C2
Varicose Veins
C3
Swelling
C4
Skin Changes
C5,C6
Venous Ulcer
Stages of
Chronic Venous Disease

Table 1. CEAP classification of chronic venous disease

Clinical Classification (C)	
C_0	No visible/palpable signs of venous disease
C_1	Telangiectasias or reticular veins
C_2	Varicose veins
C_3	Edema
C_{4a}	Pigmentation and/or eczema
C_{4b}	Lipodermatosclerosis and/or atrophy
C_5	Healed venous ulcer
C_6	Open venous ulcer
	Subscript
A	Asymptomatic
S	Symptomatic

Etiologic Classification (E)	
E_c	Congenital
E_p	Primary
E_s	Secondary (postthrombotic)
E_n	No venous etiology identified
Anatomic Classification (A)	
A_s	Superficial veins
A_p	Perforator veins
A_d	Deep veins
A_n	No venous location identified
Pathophysiologic Classification (P)	
P_r	Reflux
P_o	Obstruction
$P_{r,o}$	Reflux and obstruction
P_n	No venous pathophysiology identifiable

Source: Adapted from the 2011 Clinical Guidelines of the Society for Vascular Surgery and American Venous Forum (J Vasc Surg. 2011;53:2S-48S)

VENOUS ULCERS

Clinical assessment of individuals with venous insufficiency of the lower limbs, despite being important, does not independently identify affected systems or anatomic levels.[9,10] Ulcers caused by CVI can result from obstruction or reflux in the DVS, reflux in superficial system veins and in perforating veins, or from a combination of both.

Duplex scan is considered the gold standard among noninvasive diagnostic methods for venous diseases of the lower limbs, is currently the most widely indicated because it allows qualitative and quantitative assessment. It provides both anatomic and functional information, thus allowing a more complete and detailed assessment of the venous system.

- Saliba Jr., Orlando Adas, Giannini, Mariangela, & Rollo, Hamilton Almeida. (2007). Noninvasive diagnostic methods to evaluate venous insufficiency of the lower limbs. *Jornal Vascular Brasileiro*, *6*(3), 266-275. https://dx.doi.org/10.1590/S1677-54492007000300010

Order venous reflux study or specify “evaluate for venous reflux” in the order.

Otherwise you will get a r/o DVT study!

DFU

The most common cause of non-traumatic lower extremity amputations in the US and Europe

Responsible for 20% of the nearly 3 million hospitalizations every year related to diabetes

Estimated up to 25% of all diabetics will develop a diabetic foot ulcer

DFU… A FEW FACTS

Low survival prognosis

DFU 3-year cumulative mortality rate of 28

Amputated patients approaching 50% mortality

67%percent of all lower extremity amputation patients have diabetes

Recurrence rate is 66%

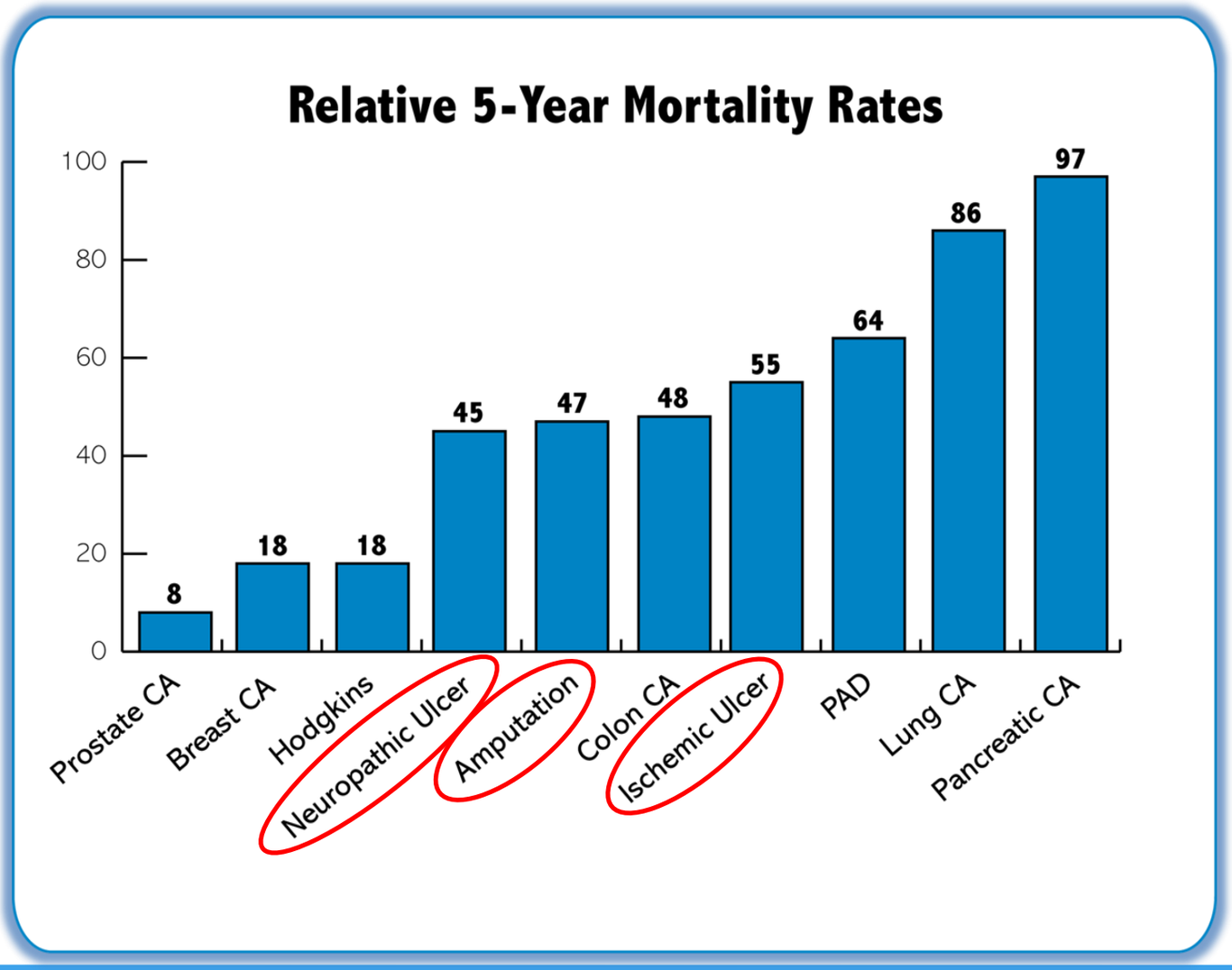
Lower-extremity amputation rate 28 X that of people without diabetes

ARE DM RELATED WOUNDS AND AMPUTATIONS WORSE THAN CANCER?

5-year survival rate after one major lower extremity amputation is about 50%

Once amputation occurs, 50% of patients will develop an ulcer in the contralateral limb within 5 years

International Wound Journal Vol 4 No 4; 2007



ARTERIAL ULCERS

Between toes or tips of toes

Over phalangeal heads

Around lateral malleolus

Areas subjected to trauma/rubbing footwear

Even wound margins

Punched out appearance

Pale, deep wound bed

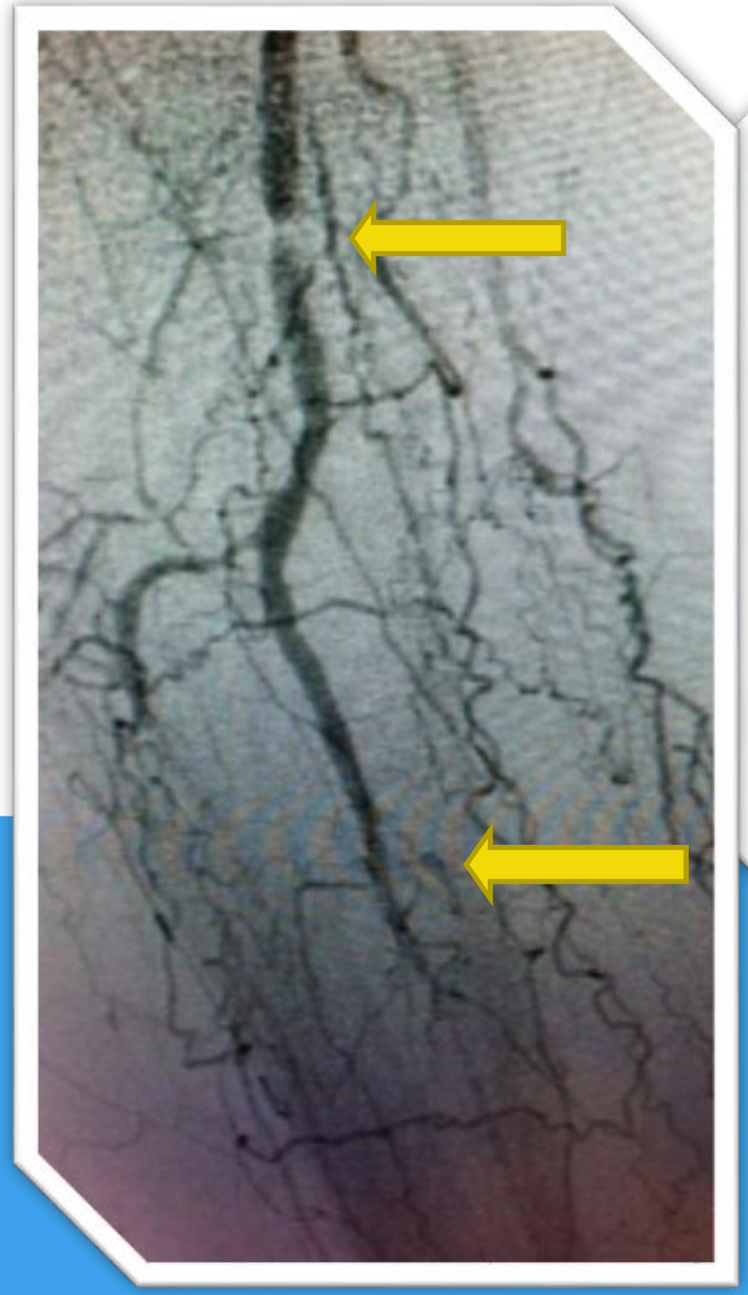
Blanched peri-wound tissue

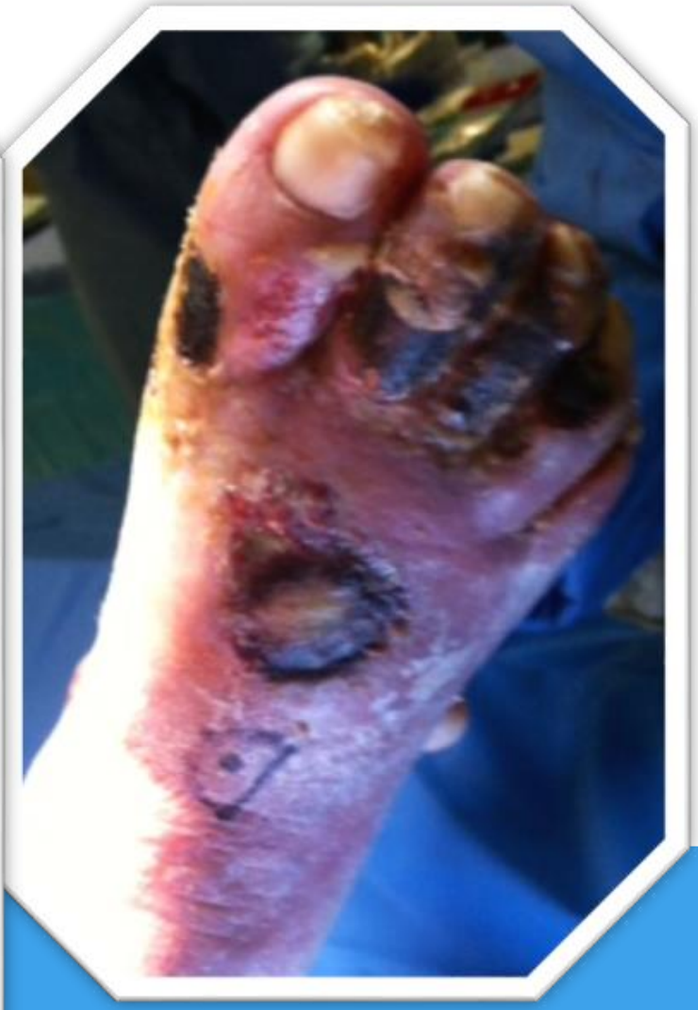
Extreme pain

Cellulitis

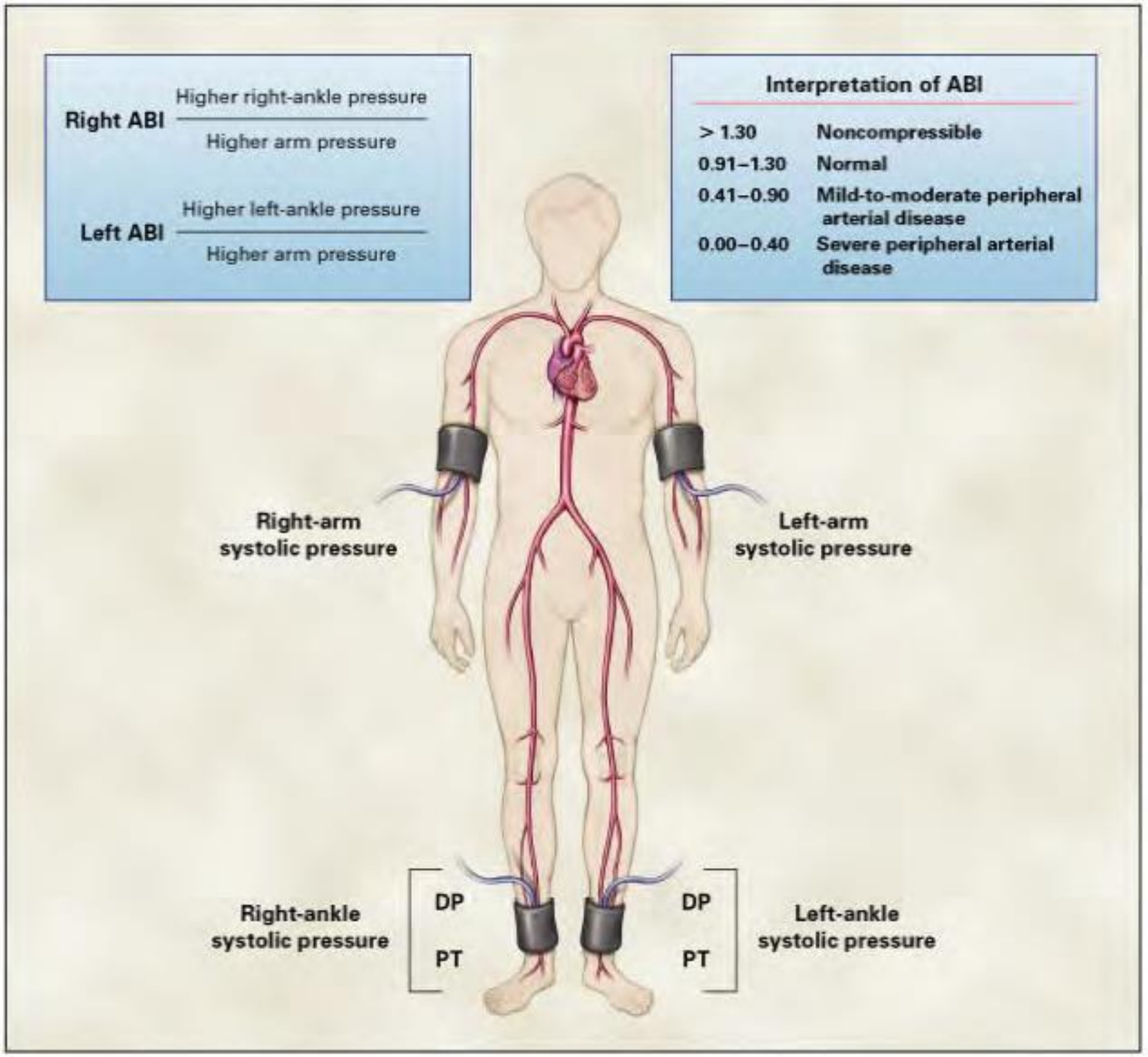
Minimal exudate

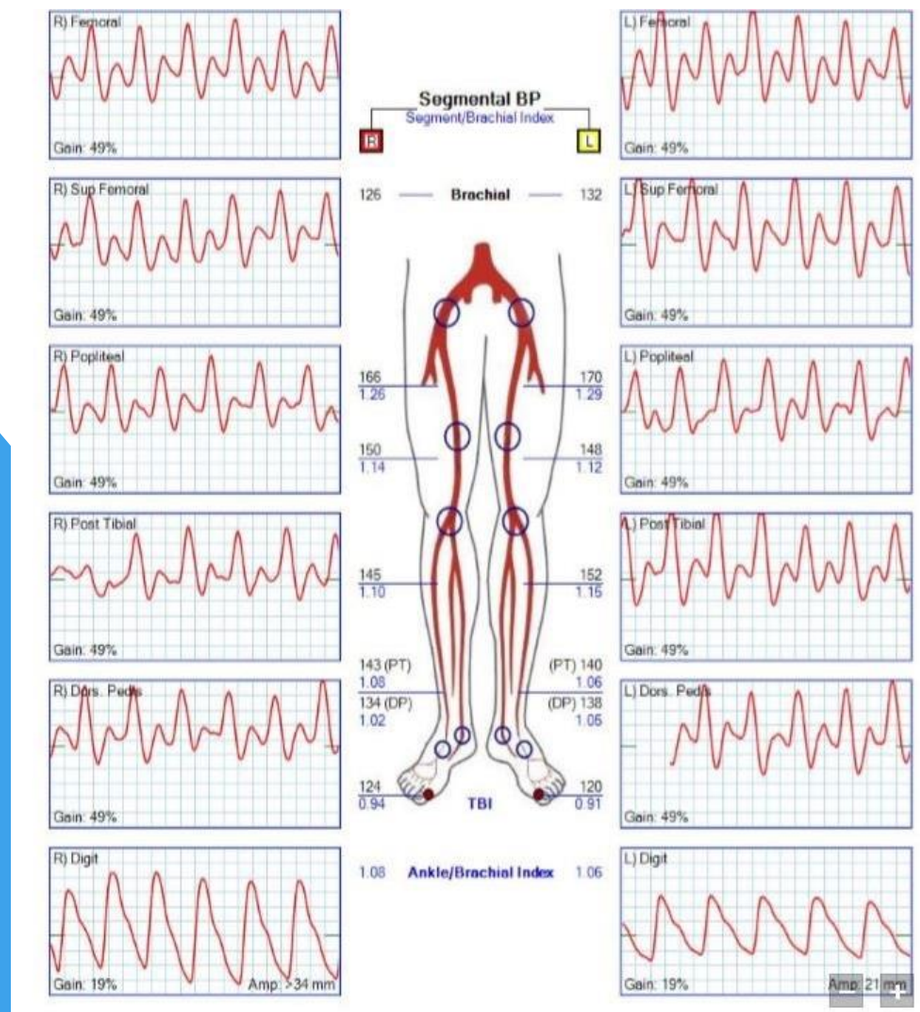
Gangrene/necrosis

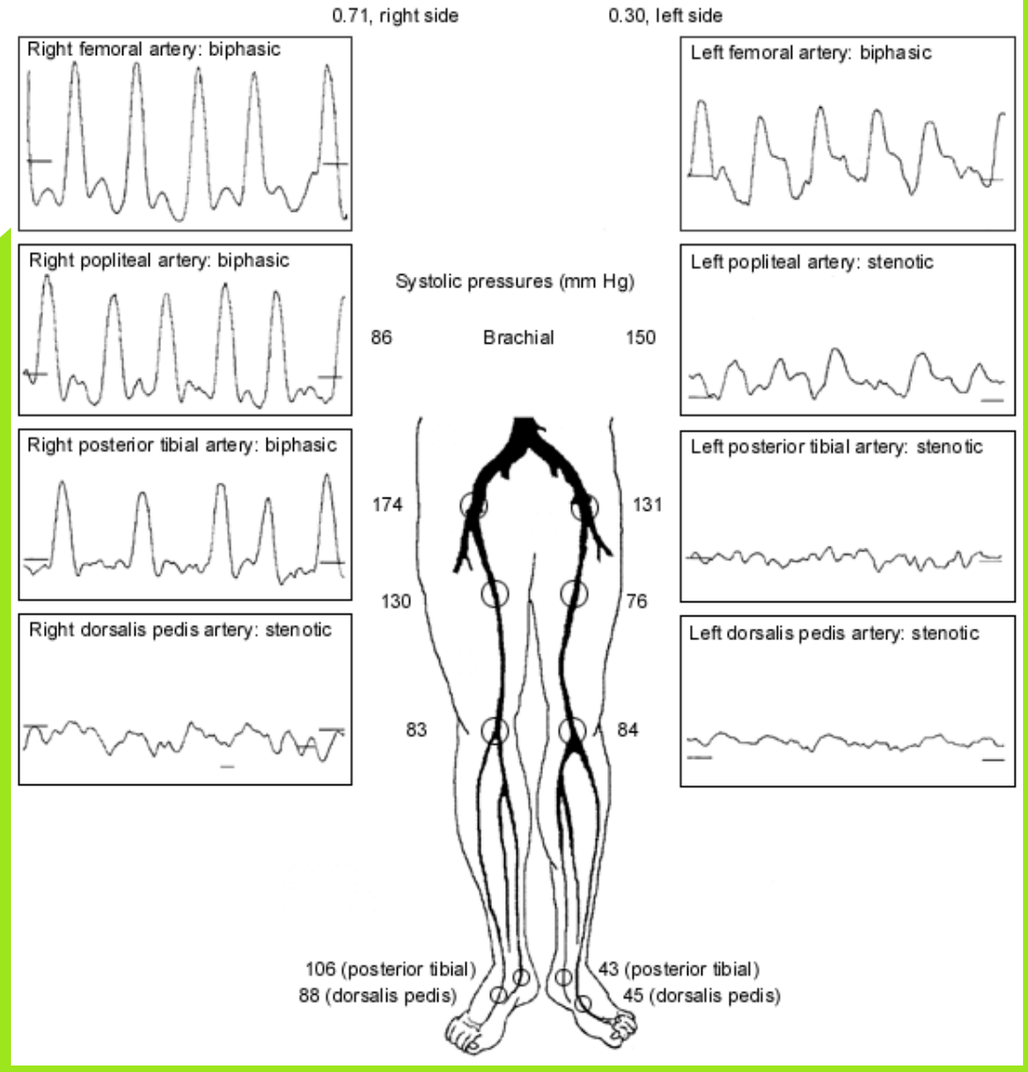




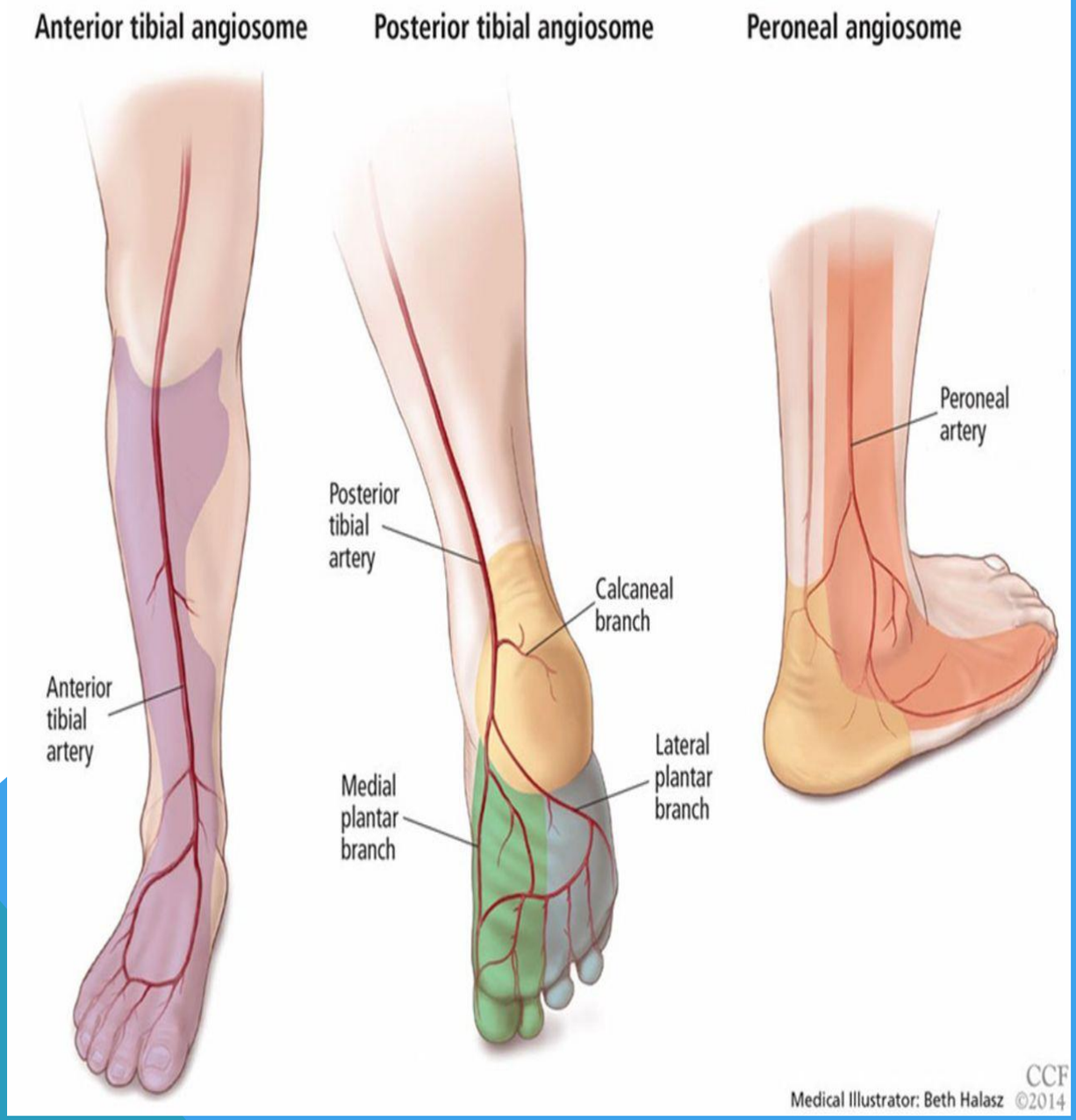
Perfusion Assessment

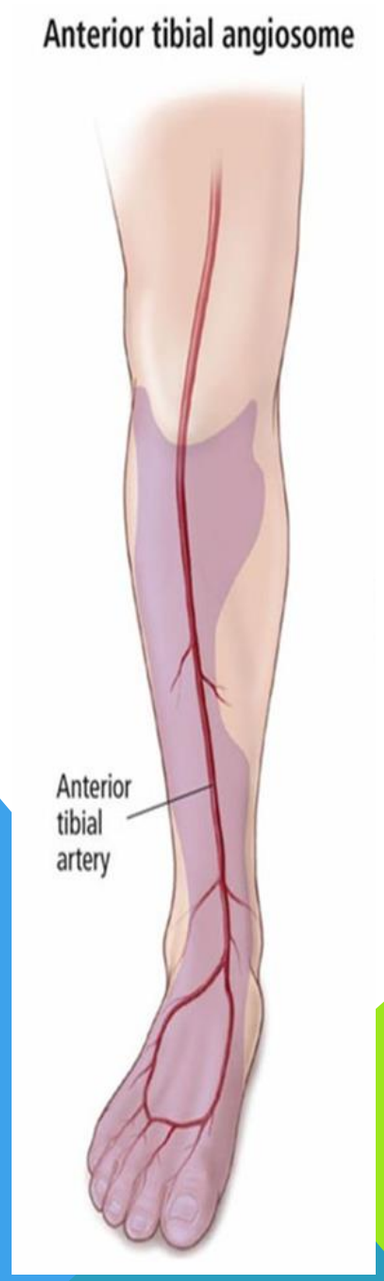


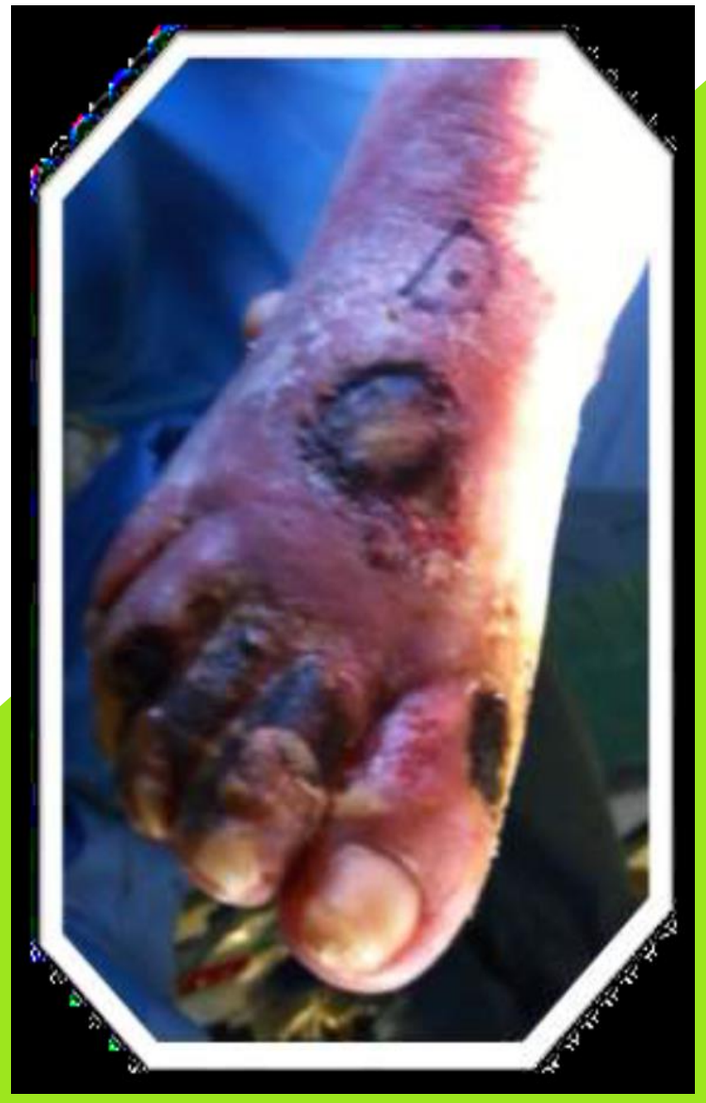

R) Femoral
Gain: 49%
Segmental BP
Segment/Brachial Index
R
L
L) Femoral
Gain: 49%
R) Sup Femoral
Gain: 49%
126
Brachial
132
L) Sup Femoral
Gain: 49%
R) Popliteal
Gain: 49%
166
1.26
150
1.14
L) Popliteal
Gain: 49%
170
1.29
148
1.12
R) Post Tibial
Gain: 49%
145
1.10
L) Post Tibial
Gain: 49%
152
1.15
R) Dors. Pedis
Gain: 49%
143 (PT)
1.08
134 (DP)
1.02
(PT) 140
1.06
(DP) 138
1.05
L) Dors. Pedis
Gain: 49%
124
0.94
TBI
120
0.91
R) Digit
Gain: 19%
Amp: >34 mm
1.08
Ankle/Brachial Index
1.06
L) Digit
Gain: 19%
Amp: 21 mm

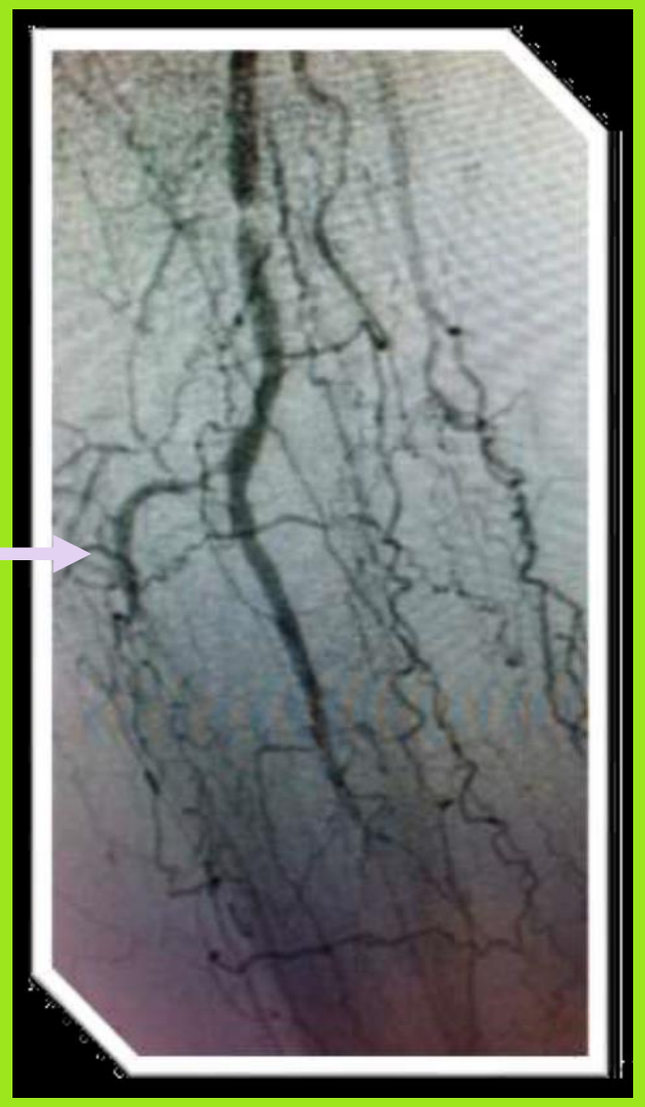

0.71, right side
0.30, left side
Right femoral artery: biphasic
Left femoral artery: biphasic
Right popliteal artery: biphasic
Left popliteal artery: stenotic
Systolic pressures (mm Hg)
86
Brachial
150
Right posterior tibial artery: biphasic
Left posterior tibial artery: stenotic
174
131
130
76
Right dorsalis pedis artery: stenotic
Left dorsalis pedis artery: stenotic
83
84
106 (posterior tibial)
88 (dorsalis pedis)
43 (posterior tibial)
45 (dorsalis pedis)

Angiosomes of the lower extremity

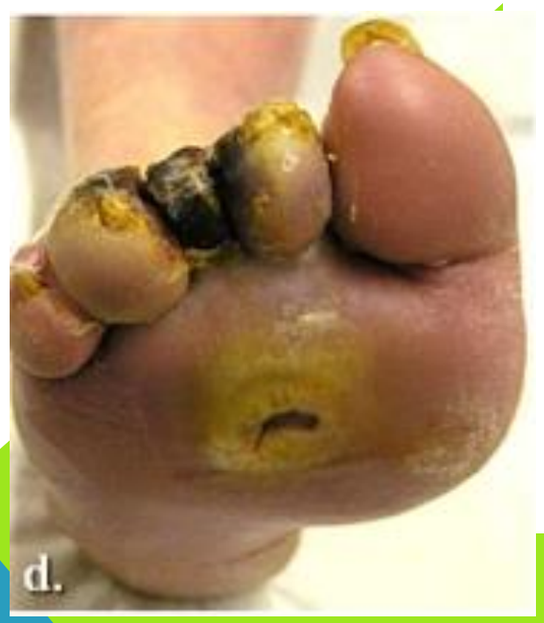


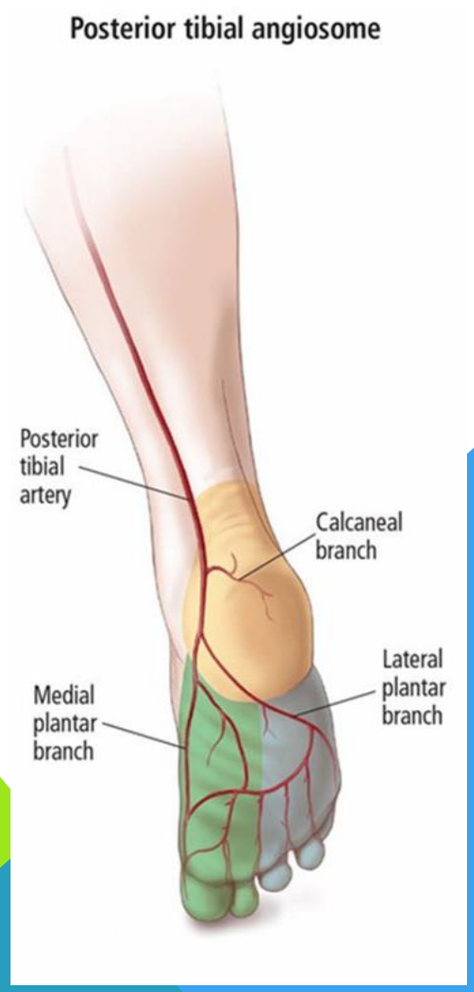

Anterior tibial angiosome
Anterior
tibial
artery





AND…
Why is it still here?




Posterior tibial angiosome
Posterior tibial artery
Calcaneal branch
Lateral plantar branch
Medial plantar branch


a.
b.
d.
e.


WHEN YOU WAKE UP
EXPECTATION
REALITY

Pressure Injury

The most important cause is pressure exerted for an excessive period of time.

Further damaging factors:

-friction at the skin surface

-shearing forces

-moisture

Why do you need to know about pressure injuries?

Preventable

Documentation in the patient chart to trigger appropriate care if admitted!!!

Tremendous implications for your organization.

Pressure Injury

In the state of Washington, mandatory reporting to the Department of Health of Stages III and IV hospital-acquired pressure ulcers (HAPUs) began in 2006.

CMS NEVER EVENT

https://www.cms.gov/newsroom/fact-sheets/eliminating-serious-preventable-and-costly-medical-errors-never-events

, S., Whitney, J. D., Lowe, J. R., Taylor, S., O'Donnell, F., & Minton-Foltz, P. (2010). Development and implementation of a hospital-acquired pressure ulcer incidence tracking syste and algorithm. *Journal for healthcare quality : official publication of the National Association for Healthcare Quality*, *32*(6), 44–51. doi:10.1111/j.1945-1474.2010.00076.x

Hospital-Acquired Conditions

- Foreign Object Retained After Surgery
- Air Embolism
- Blood Incompatibility
- Stage III and IV Pressure Ulcers
- Falls and Trauma
  - Fractures
  - Dislocations
  - Intracranial Injuries
  - Crushing Injuries
  - Burn
  - Other Injuries
- Manifestations of Poor Glycemic Control
  - Diabetic Ketoacidosis
  - Nonketotic Hyperosmolar Coma
  - Hypoglycemic Coma
  - Secondary Diabetes with Ketoacidosis
  - Secondary Diabetes with Hyperosmolarity
- Catheter-Associated Urinary Tract Infection (UTI)
- Vascular Catheter-Associated Infection
- Surgical Site Infection, Mediastinitis, Following Coronary Artery Bypass Graft (CABG):
- Surgical Site Infection Following Bariatric Surgery for Obesity
  - Laparoscopic Gastric Bypass
  - Gastroenterostomy
  - Laparoscopic Gastric Restrictive Surgery
- Surgical Site Infection Following Certain Orthopedic Procedures
  - Spine
  - Neck
  - Shoulder
  - Elbow
- Surgical Site Infection Following Cardiac Implantable Electronic Device (CIED)
- Deep Vein Thrombosis (DVT)/Pulmonary Embolism (PE) Following Certain Orthopedic Procedures:
  - Total Knee Replacement
  - Hip Replacement
- Iatrogenic Pneumothorax with Venous Catheterization

https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/HospitalAcqCond/Hospital-Acquired_Conditions.html

Pressure Injury:

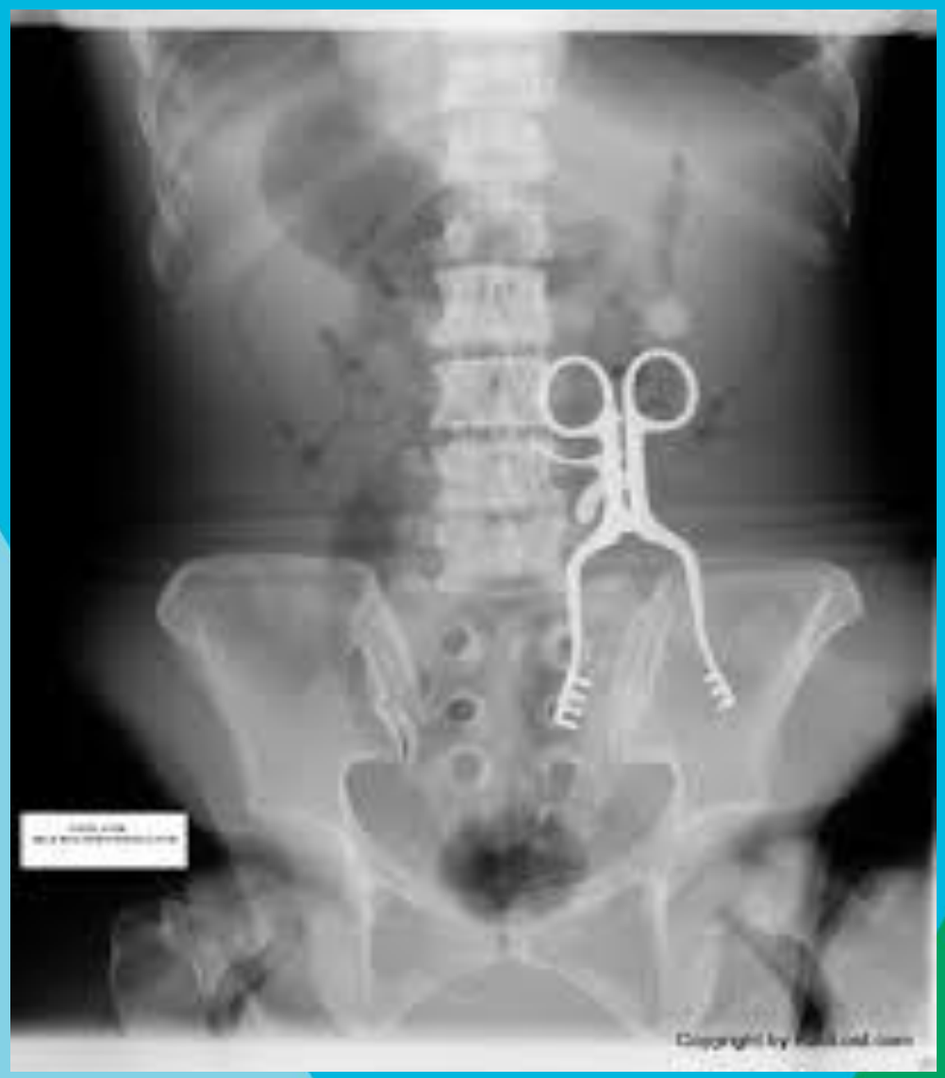
Localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical or other device.

Can present as intact skin or an open ulcer and may be painful.

Injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear.

The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, co-morbidities and condition of the soft tissue.

https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/npuap_pressure_injury_stages.pdf



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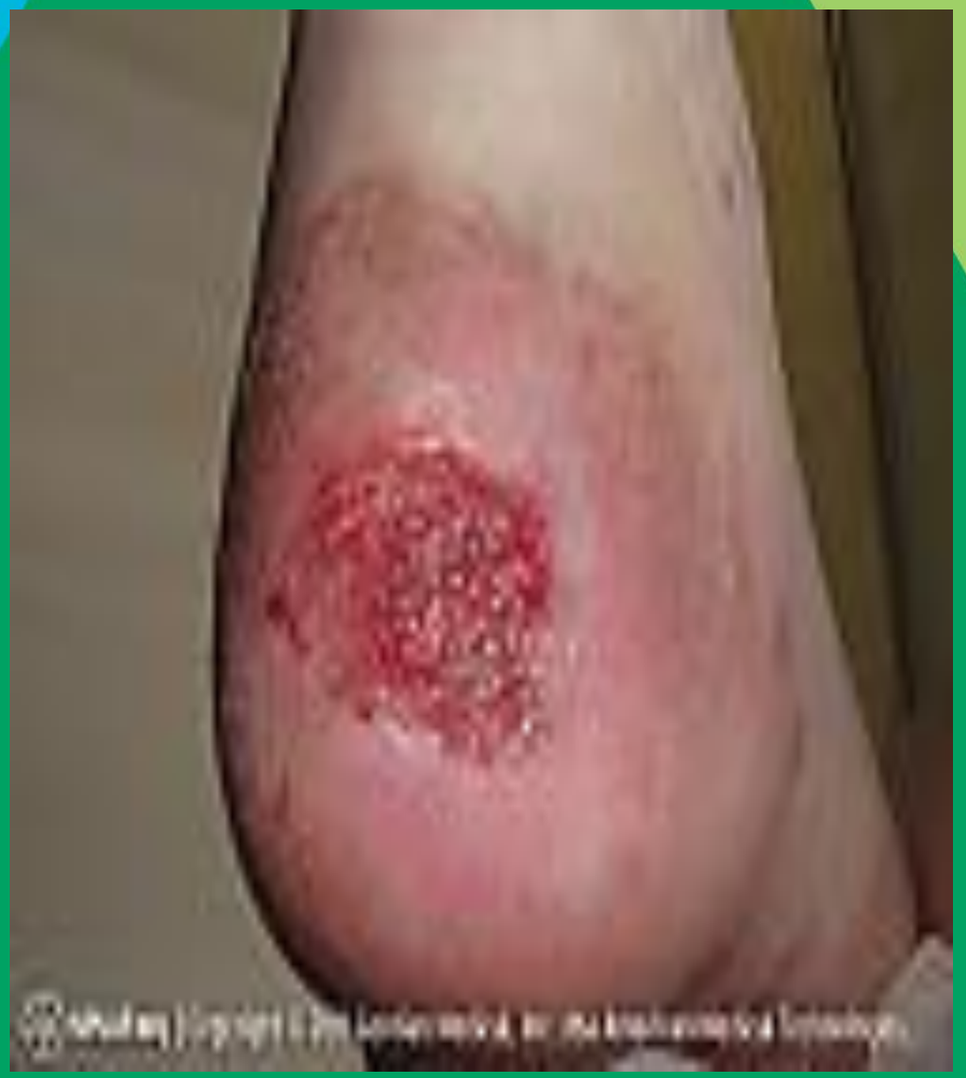


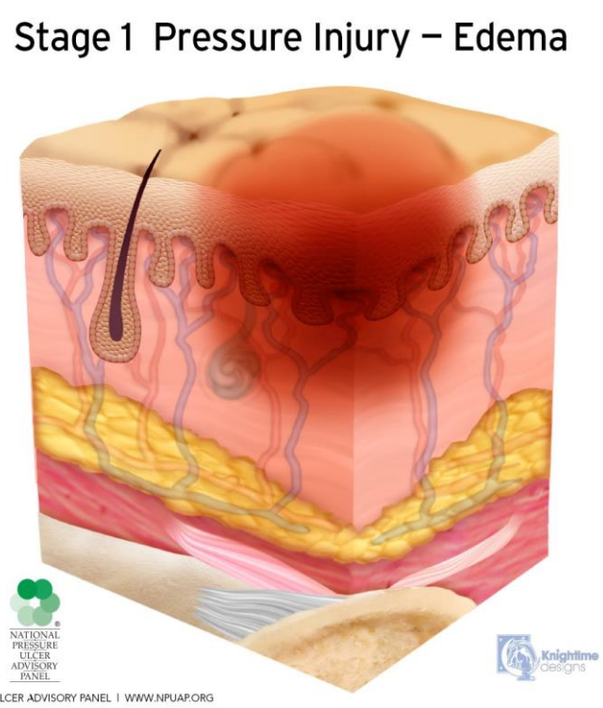
NPUAP Pressure Injury: Stage 1

-Non-blanchable erythema of intact skin.

-Can appear differently in darkly pigmented skin.

-Blanchable erythema +/- changes in sensation, temperature, or firmness may precede visual changes.

-Color changes do not include purple or maroon discoloration; these may indicate deep tissue pressure injury.



https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/npuap_pressure_injury_stages.pdf

NPUAP Pressure Injury: Stage 2

-Partial-thickness skin loss with exposed dermis

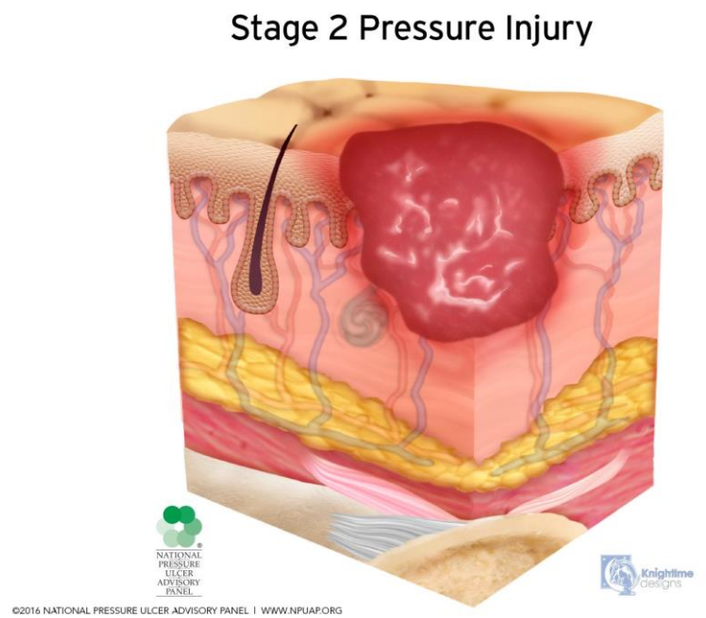
-The wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister.

-Adipose (fat) is not visible and deeper tissues are not visible.

-Granulation tissue, slough and eschar are not present.

-These injuries commonly result from adverse microclimate and shear in the skin over the pelvis and shear in the heel.

-This stage should not be used to describe moisture associated skin damage (MASD) including incontinence associated dermatitis (IAD), intertriginous dermatitis (ITD), medical adhesive related skin injury (MARSI), or traumatic wounds (skin tears, burns, abrasions).



https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/npuap_pressure_injury_stages.pdf

NPUAP Pressure Injury: Stage 3

-Full-thickness skin loss

-Adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present.

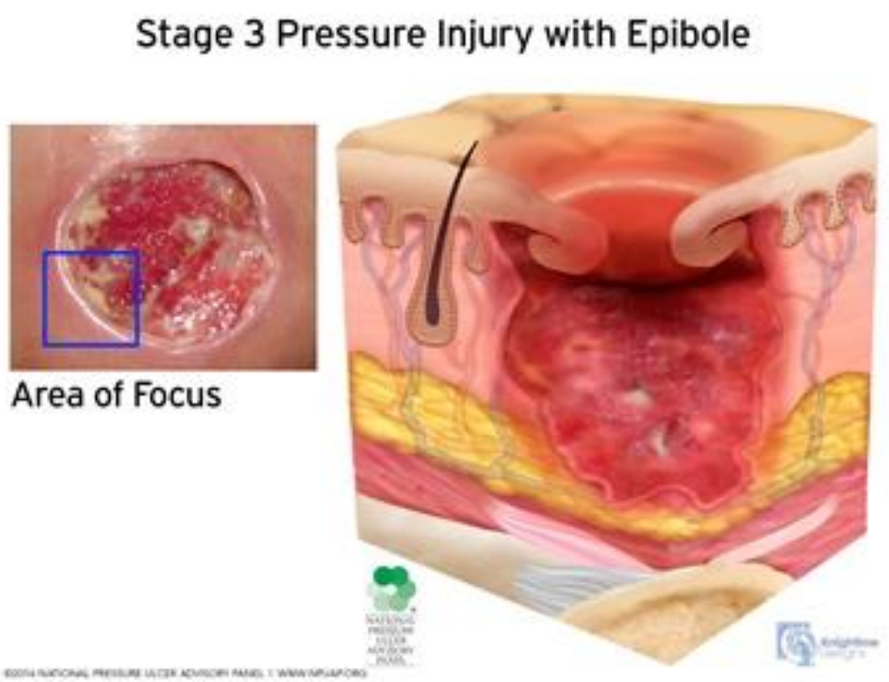
-Slough and/or eschar may be visible.

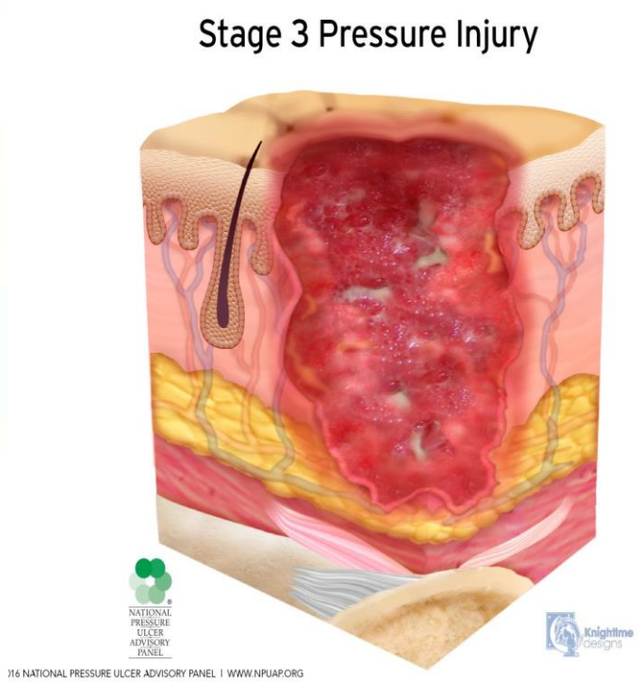
-The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds.

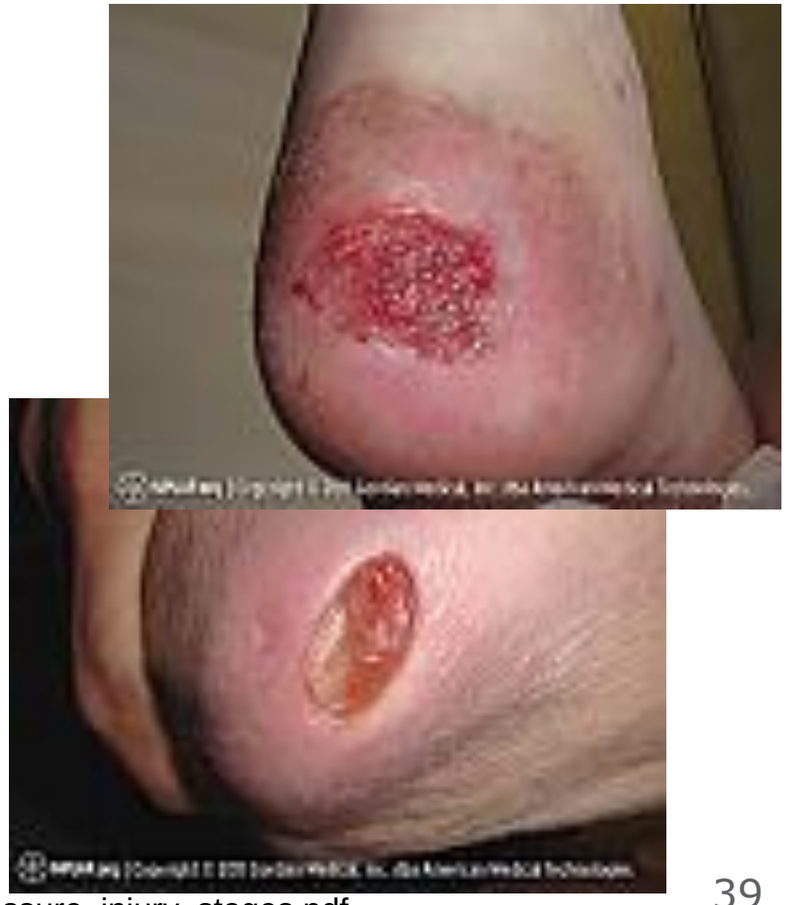
-Undermining and tunneling may occur.

-Fascia, muscle, tendon, ligament, cartilage and/or bone are not exposed.

-If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury.







https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/npuap_pressure_injury_stages.pdf

NPUAP Pressure Injury: Stage 4

-Full-thickness skin and tissue loss

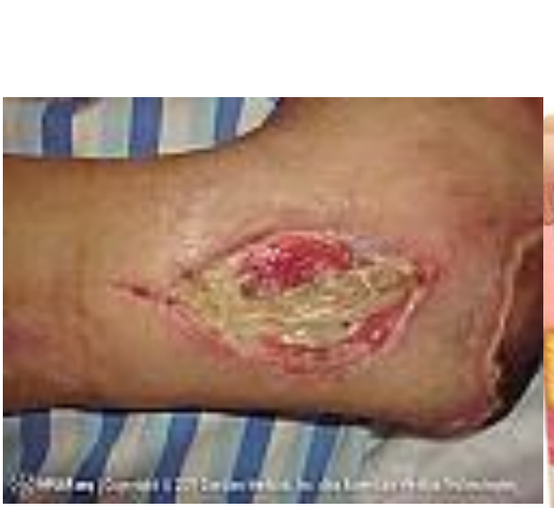
-Exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer.

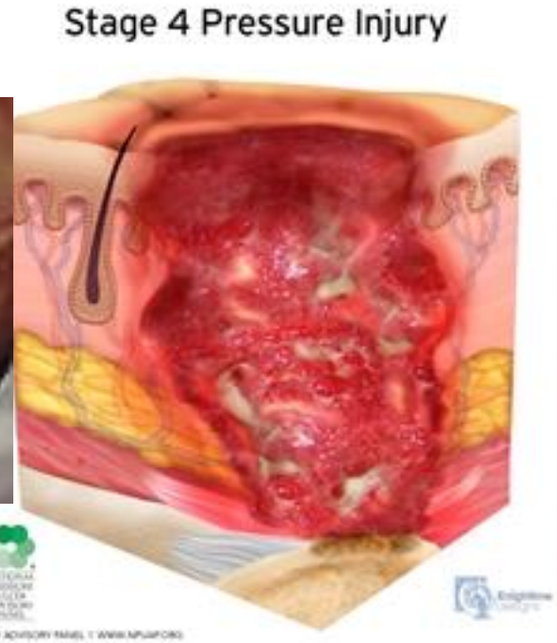
-Slough and/or eschar may be visible.

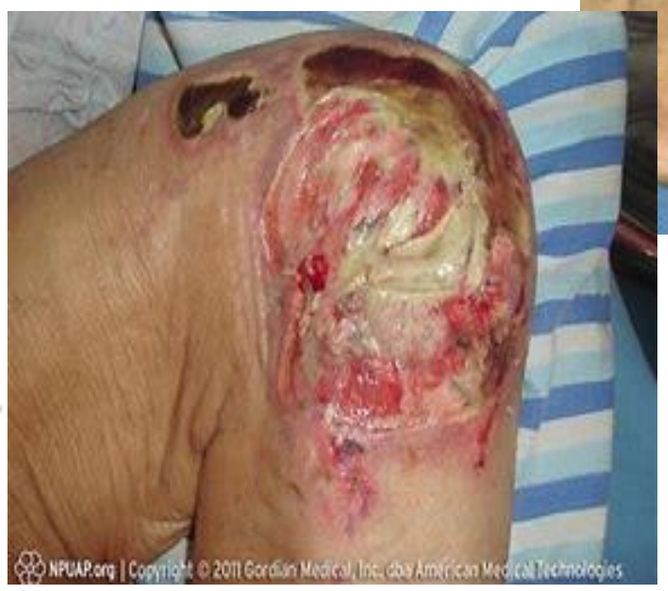
-Epibole (rolled edges), undermining and/or tunneling often occur.

-Depth varies by anatomical location.

-If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury



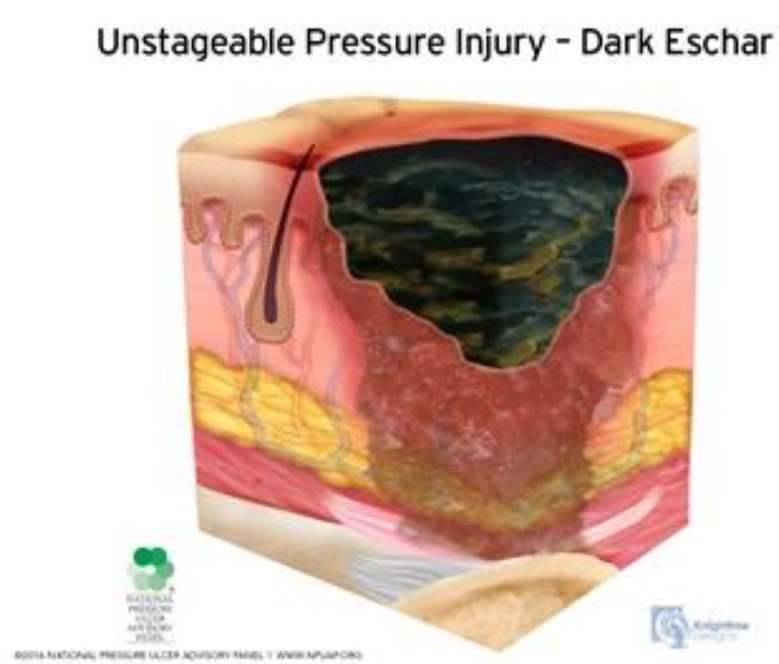


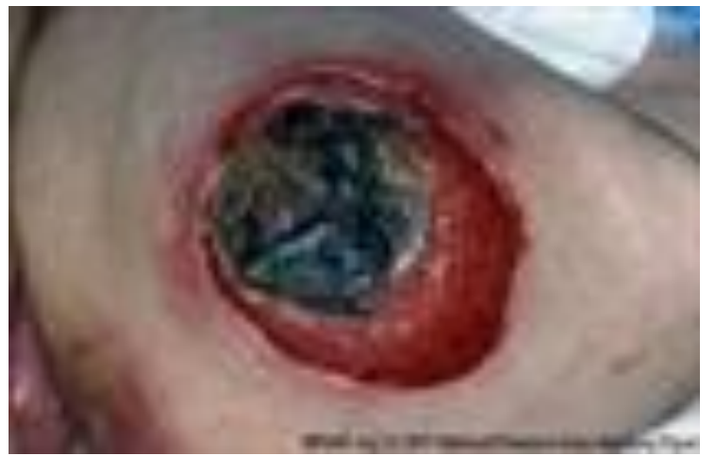


https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/npuap_pressure_injury_stages.pdf

NPUAP Pressure Injury: Unstageable

-Obscured full-thickness skin and tissue loss.
-Extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar.
-If slough or eschar is removed, a Stage 3 or Stage 4 pressure injury will be revealed.
-Stable eschar (i.e. dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed.





https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/npuap_pressure_injury_stages.pdf

NPUAP Pressure Injury:
Deep Tissue Pressure Injury

-Persistent non-blanchable deep red, maroon or purple discoloration.

-Intact or non-intact skin with localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood filled blister.

-Pain and temperature change often precede skin color changes.

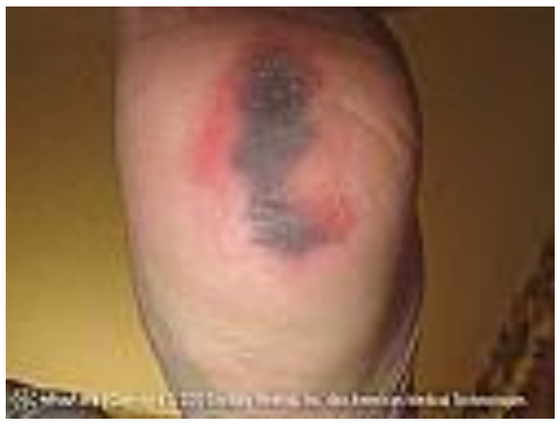
-Discoloration may appear differently in darkly pigmented skin.

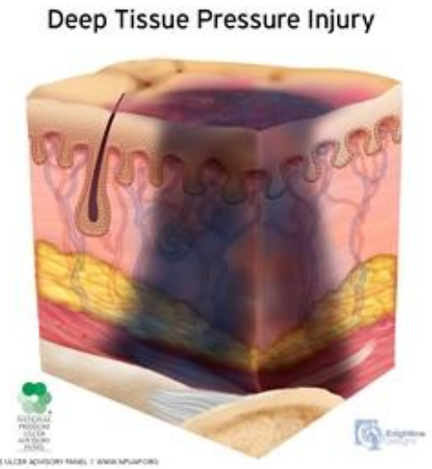
-This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface.

-The wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss.

-If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle or other underlying structures are visible, this indicates a full thickness pressure injury (Unstageable, Stage 3 or Stage 4).

-Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatologic conditions.





https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/npuap_pressure_injury_stages.pdf

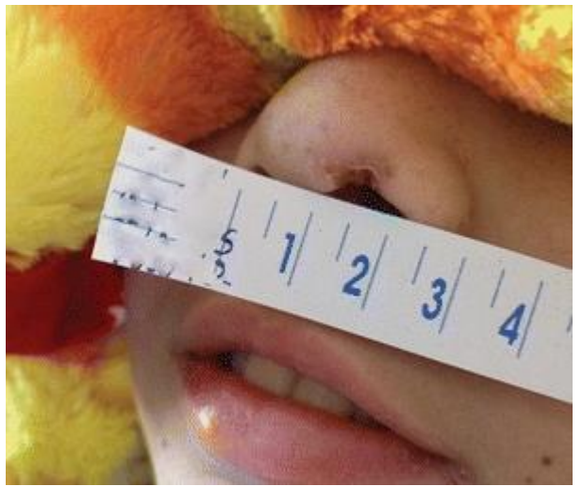
NPUAP Pressure Injury: Medical Device Related

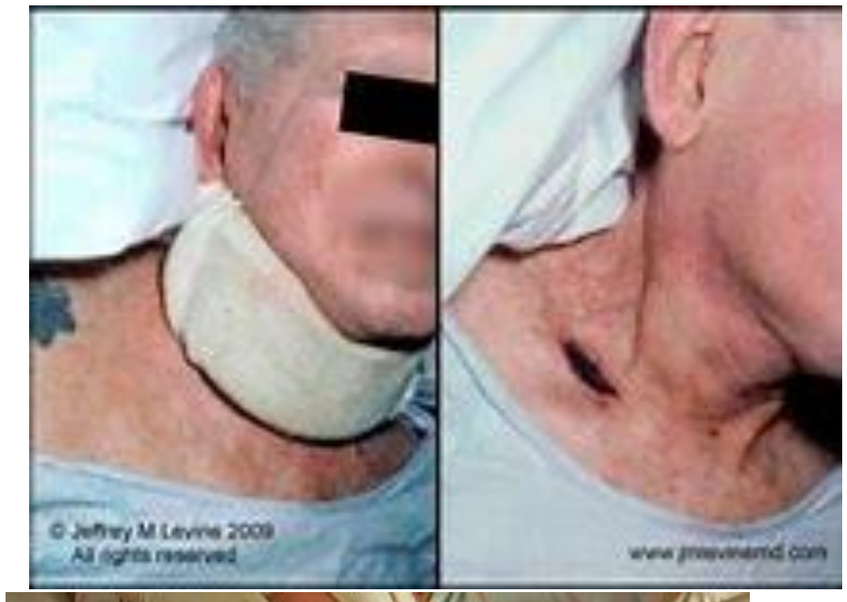
-This describes an etiology.

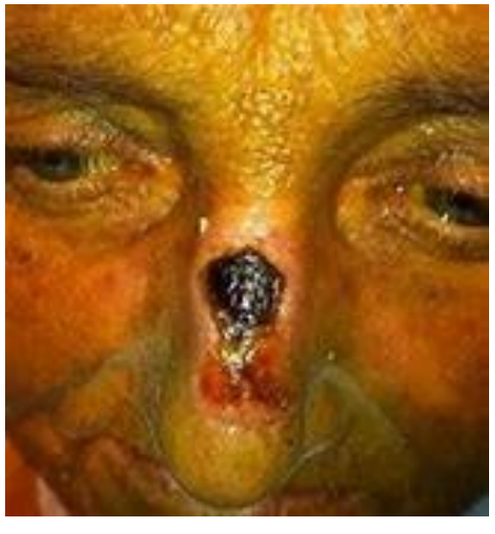
-Result from the use of devices designed and applied for diagnostic or therapeutic purposes.

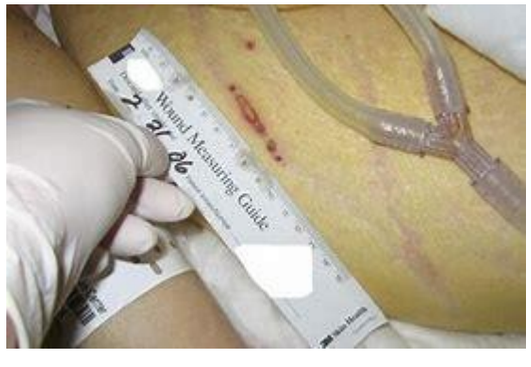
-The resultant pressure injury generally conforms to the pattern or shape of the device.

-The injury should be staged using the staging system.







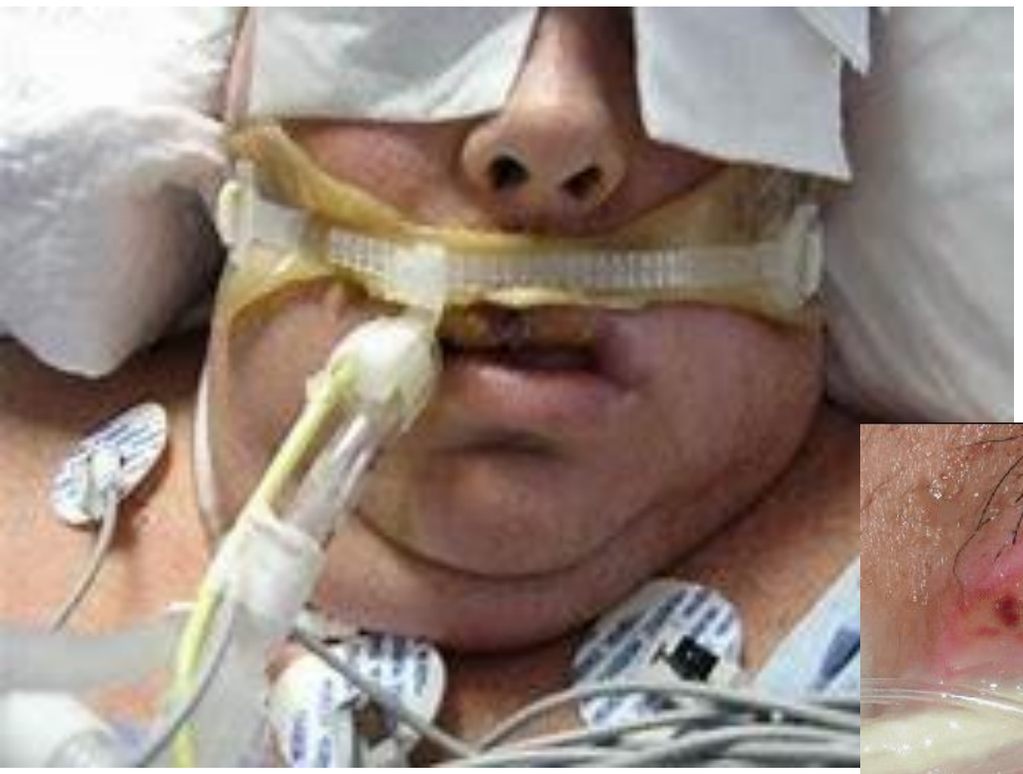


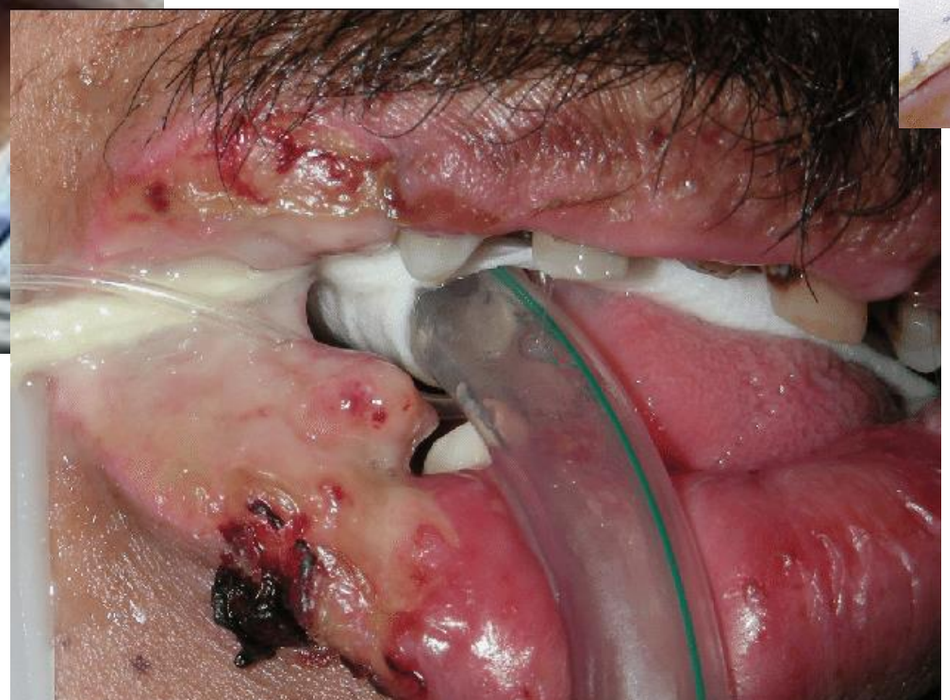
https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/npuap_pressure_injury_stages.pdf

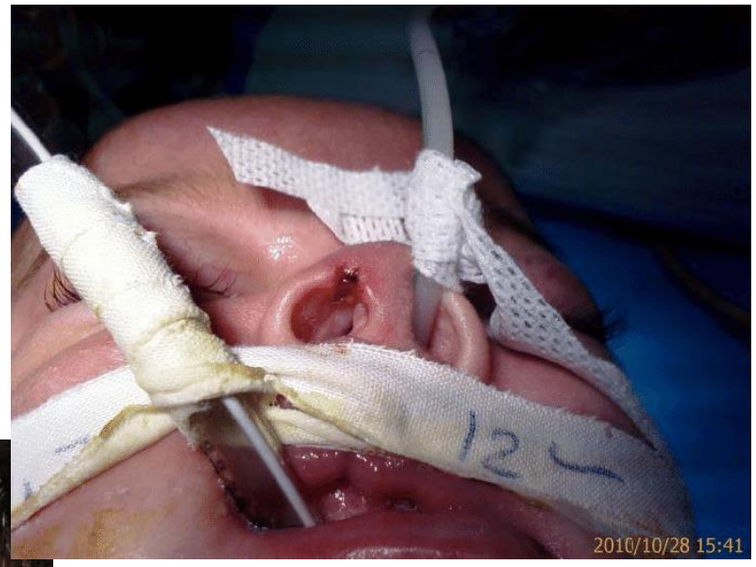
NPUAP Pressure Injury: Mucosal Membrane

-Found on mucous membranes with a history of a medical device in use at the location of the injury.

-Due to the anatomy of the tissue these ulcers cannot be staged.







Why pressure causes injury:

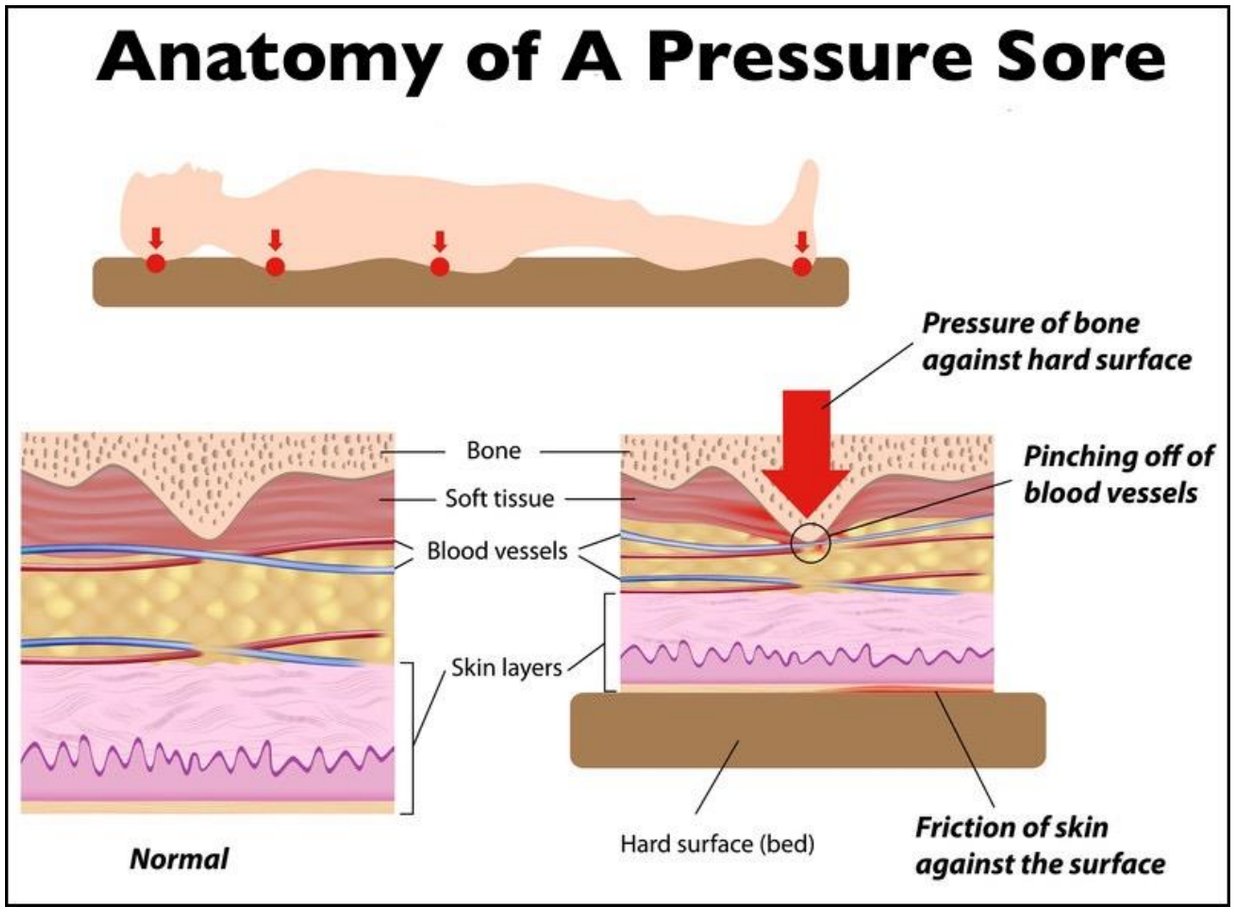
Non-uniform pressure and pressure gradients between adjacent areas of tissue causes the blood vessels in that tissue to be compressed, angulated or stretched out of their usual shape and distortion occurs.

Blood can't pass through the distorted vessels.

Tissues supplied by those blood vessels become ischemic.

Persistent ischemia causes necrosis.

This is the start of a pressure injury.



“bed of springs and nails”

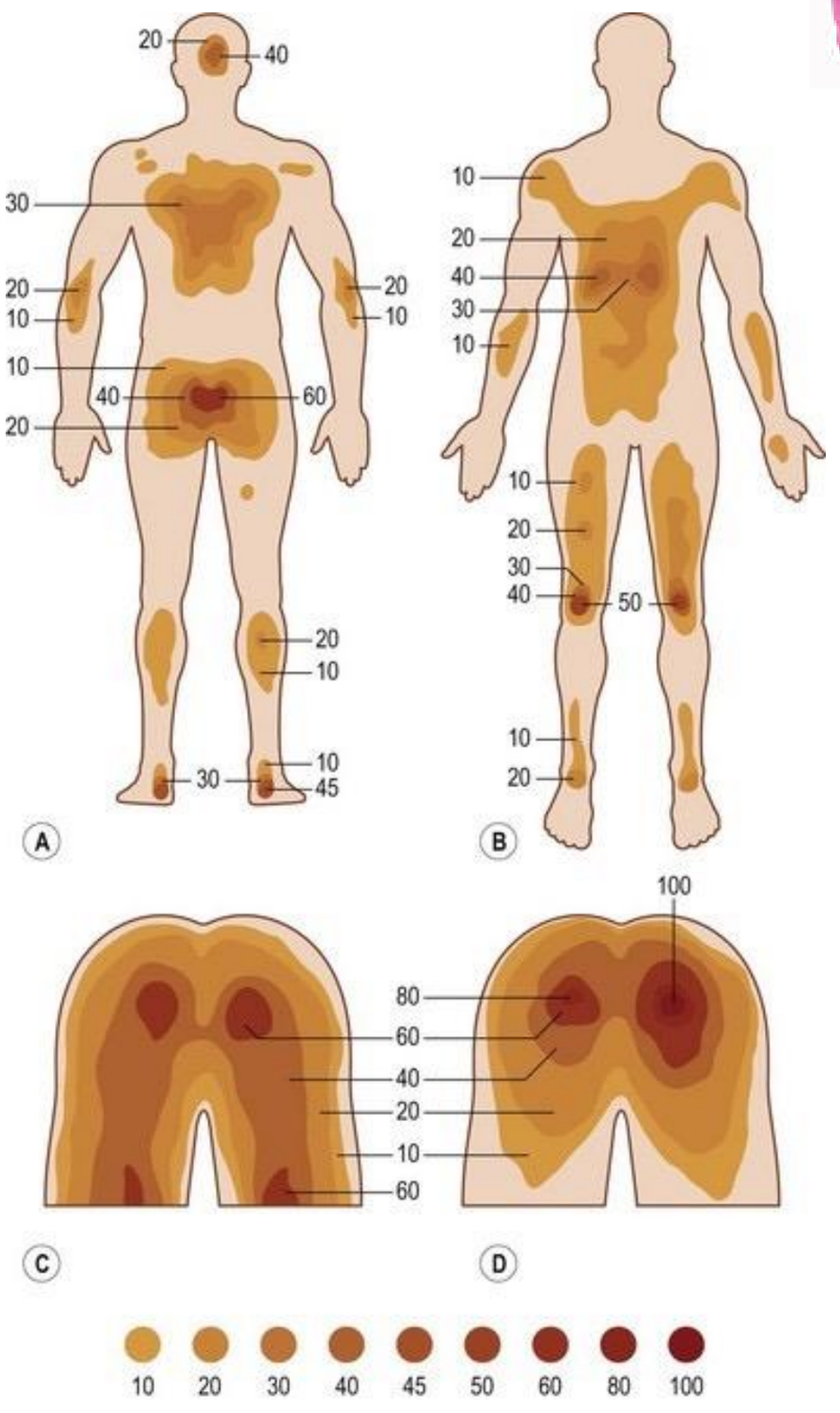
1965 Lindan studied pressure distribution on the surface of the human body by evaluating pressure in lying and sitting positions using a “bed of springs and nails.”

All major weight-bearing areas sustained pressures in excess of end-capillary pressures!!!!!!!!!!

Points of highest pressure:

Supine:
- sacrum
- buttock
- heel
- occiput

Sitting:
- ischial tuberosities



Pressure distribution of a healthy adult male **A)** supine, **(B)** prone, **(C)** sitting with feet hanging freely, and **(D)** sitting with feet supported. Values expressed in mmHg.

Adapted with permission from Lindan O, Greenway RM, Piazza JM. Pressure distribution on the surface of the human body I. Evaluation in lying and sitting positions using a “bed of springs and nails.” Arch Phys Med Rehabil 1965;46:378.)

How much and for how long?

Dinsdale

-Pressures roughly double capillary closing pressure applied for 2 hours -> irreversible ischemic damage to tissue.

-Pressures below this threshold were unlikely to cause tissue necrosis.

-Increased pressures correlated with increased likelihood of ulceration.

Kosiak et al.

- Similar findings in dog tissues.

-BUT… he found that if the pressure was released every 5 minutes few changes occurred.

Groth

-Relationship between applied pressure and the onset of tissue damage in a rabbit model.

-Inverse relationship wherein higher pressures caused damage in less time.

Husain

-Similar results in a rat model.

-Pressure applied over a large area was less injurious than when applied over a smaller one.

Nola and Vistnes

-Pressure on skin over a bone is more injurious than pressure on skin over muscle.

Daniel et al.

-Muscle was more susceptible to injury than skin & requires less pressure for a shorter duration.

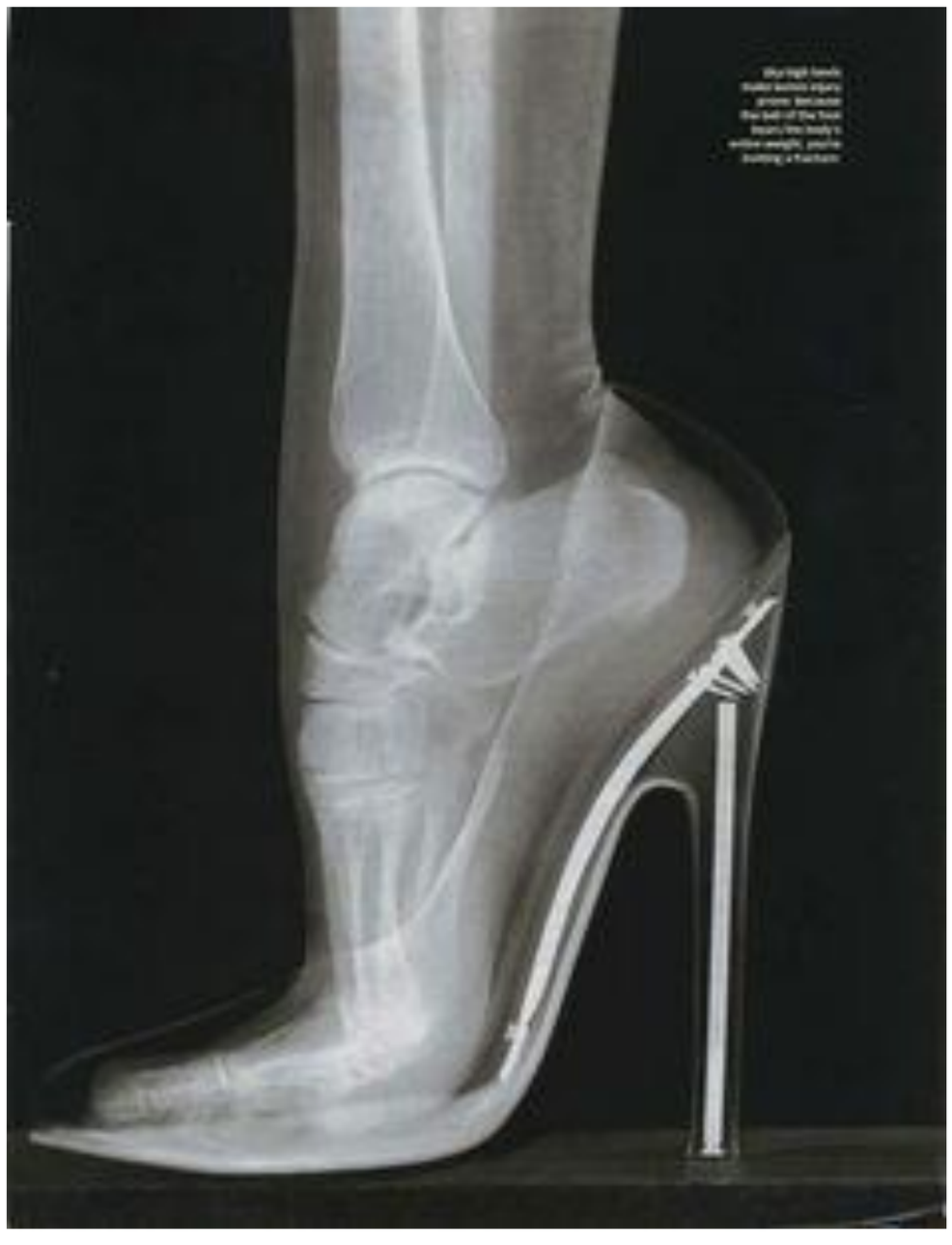
Example…

Feet

Thin covering of soft tissue

BUT…

Vasculature very well-adapted to withstand considerable distorting forces.



recap…

-Bony prominences which have a thin covering of soft tissue (heels, ankles, hips and elbows) are prone to injury.

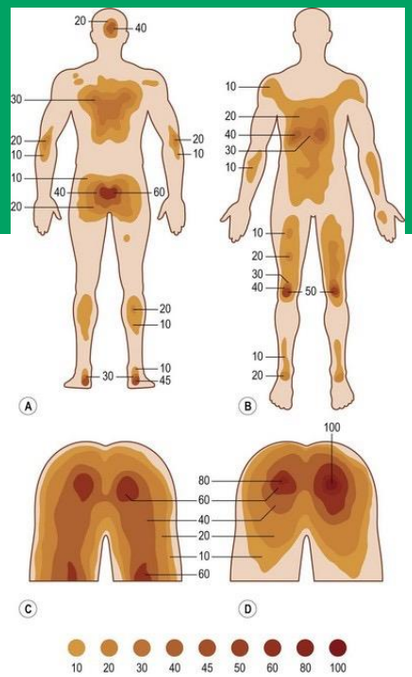
-Large compressive forces are concentrated when the body is supported on these pressure points due to the small surface area & little padding to dissipate those forces.

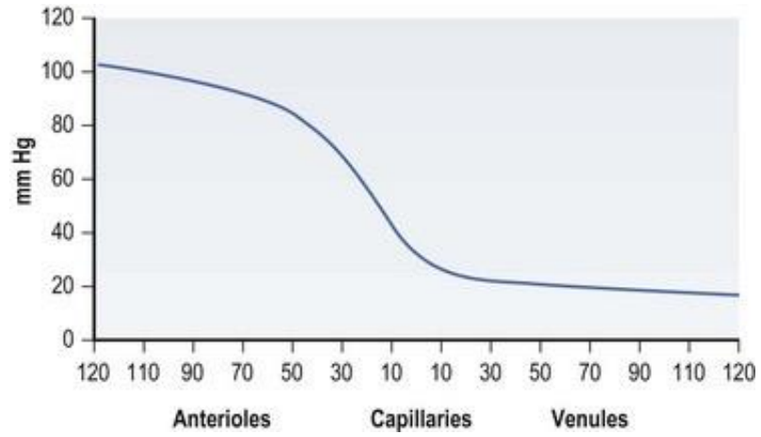
-Depth of tissue is not necessarily the determining factor.

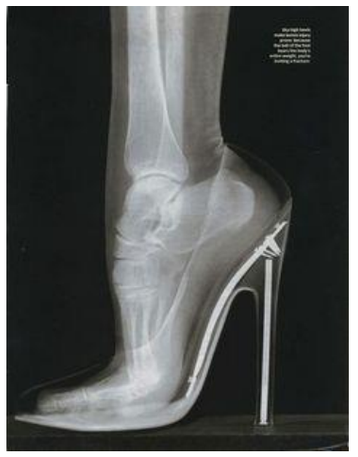
-Application of pressure in excess of end capillary pressure does not necessarily result in tissue ischemia.

-Much of the pressure applied to tissues is carried by the connective tissues surrounding the blood vessels.

-Various tissues have different susceptibility to pressure.







Pressure

Friction

Sheer

Moisture

Friction

Friction injury will be visible

Force resisting relative motion between two surfaces

Precursor to shear.

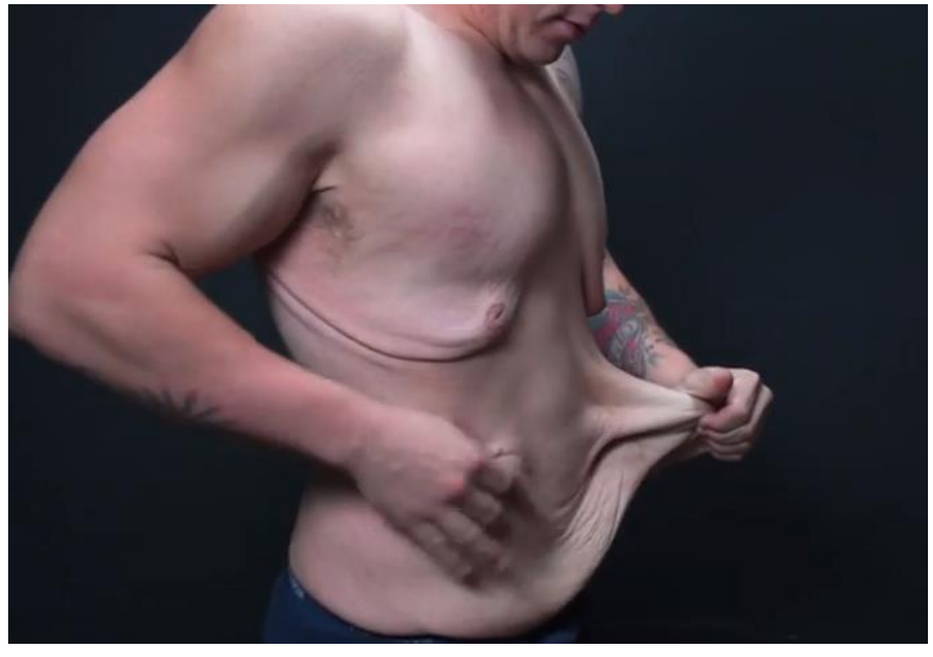
Excess friction may result in superficial skin injur

Insult by stripping the epidermal layer of the skin creates an environment conducive to further insult b altering the coefficient of friction.

This will increase the skin's adherence to the outside surface (sheets/bed…)

Friction then combines with shearing forces and the ultimate outcome may be a pressure injury

Tissues subjected to friction are more susceptible to pressure ulcer damage.



- [x] Pressure
- [x] Friction
- [ ] Sheer
- [ ] Moisture

Sheer

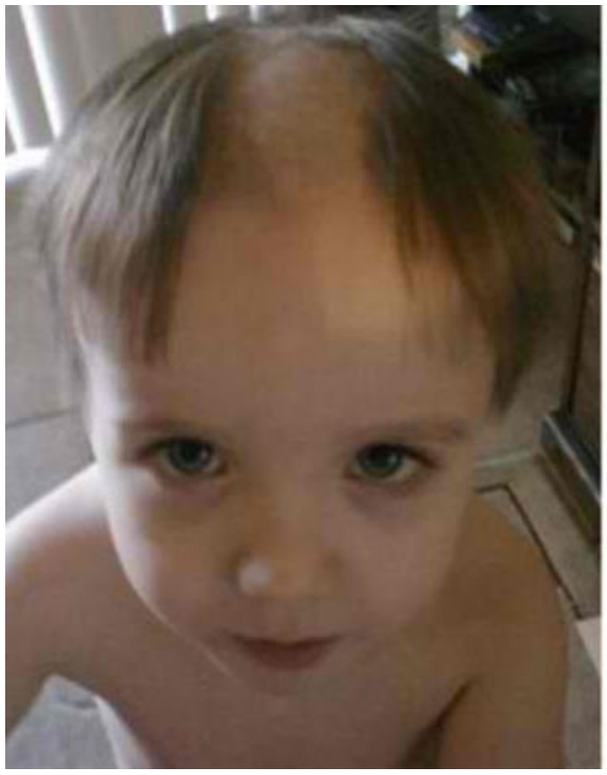
Shear injury will not be seen at the skin level because it happens beneath the skin.

Force caused when adjacent surfaces slide across each other.

Results in twisting and tearing of the underlying blood vessels.

Leads to tissue ischemia and localized tissue death.

Subcutaneous tissue in particular lacks tensile strength and is particularly susceptible to shear stress.



Dinsdale noted that addition of shear forces greatly *decreased the amount of pressure needed to cause ulceration* in a pig model, concluding that “a shear force is more disastrous than a vertical force.”

Goossens *et al.* noted similar results in human subjects, finding that the *addition of a small shear component drastically reduces the level of pressure needed to cause critical ischemia* over the sacrum.

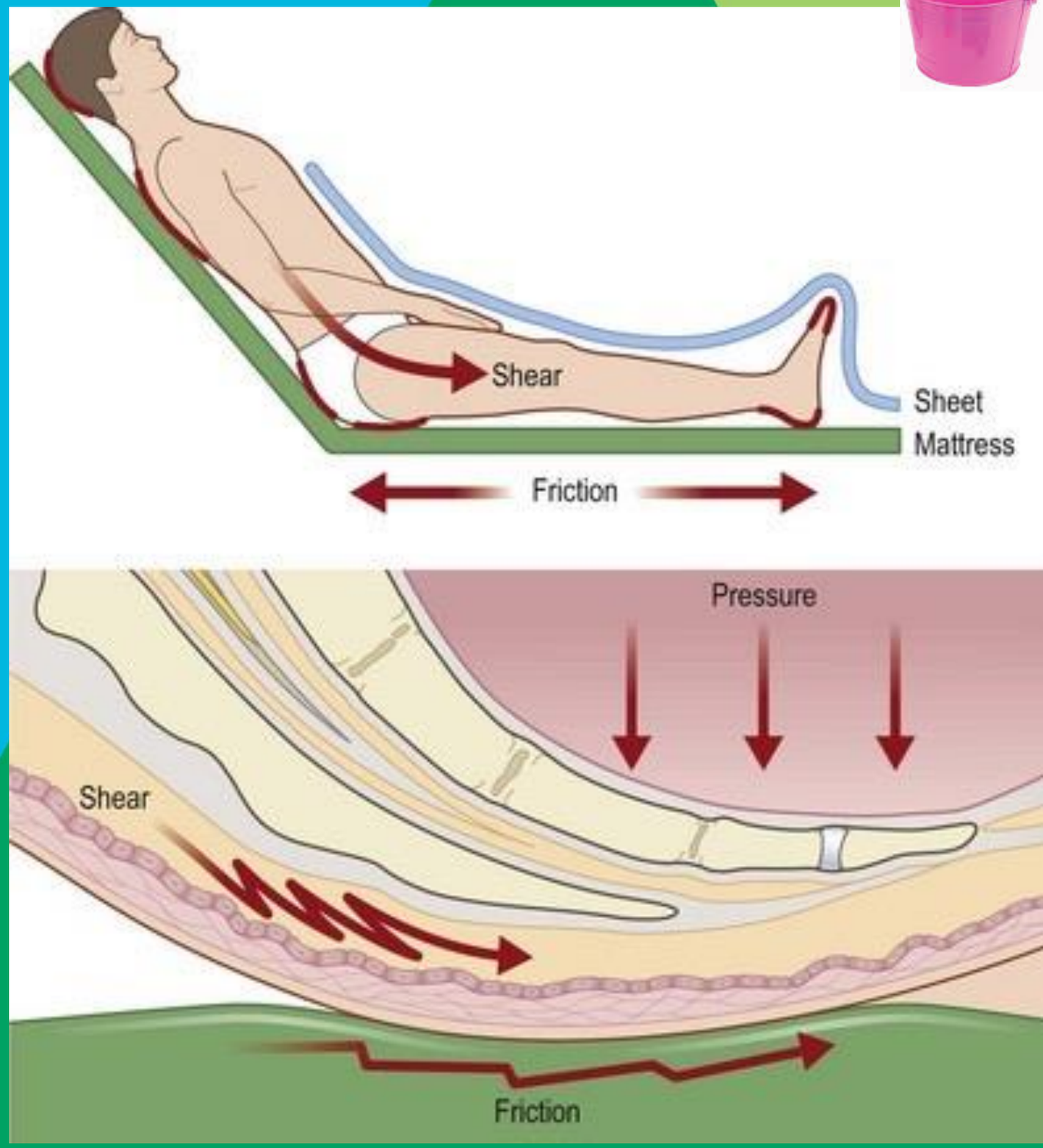
Flanagan, Fletcher. Tissue viability: managing chronic wounds. In: Brooker C, Nicol M, eds. Nursing Adults: The Practice of Caring

Sheer commonly occurs when patients are in bed with the HOB elevated.

The body begins to slide down the bed and friction keeps the skin in place…

There is a stretching or tearing of the blood vessels.

The bones and deep fascia of the pelvis move in one direction and the superficial fascia & skin don't or move in the opposite direction.



Patient transfers

Sliding/dragging/“boosting” patients up in bed

Patients that elevate themselves in bed by pushing with elbows and heels

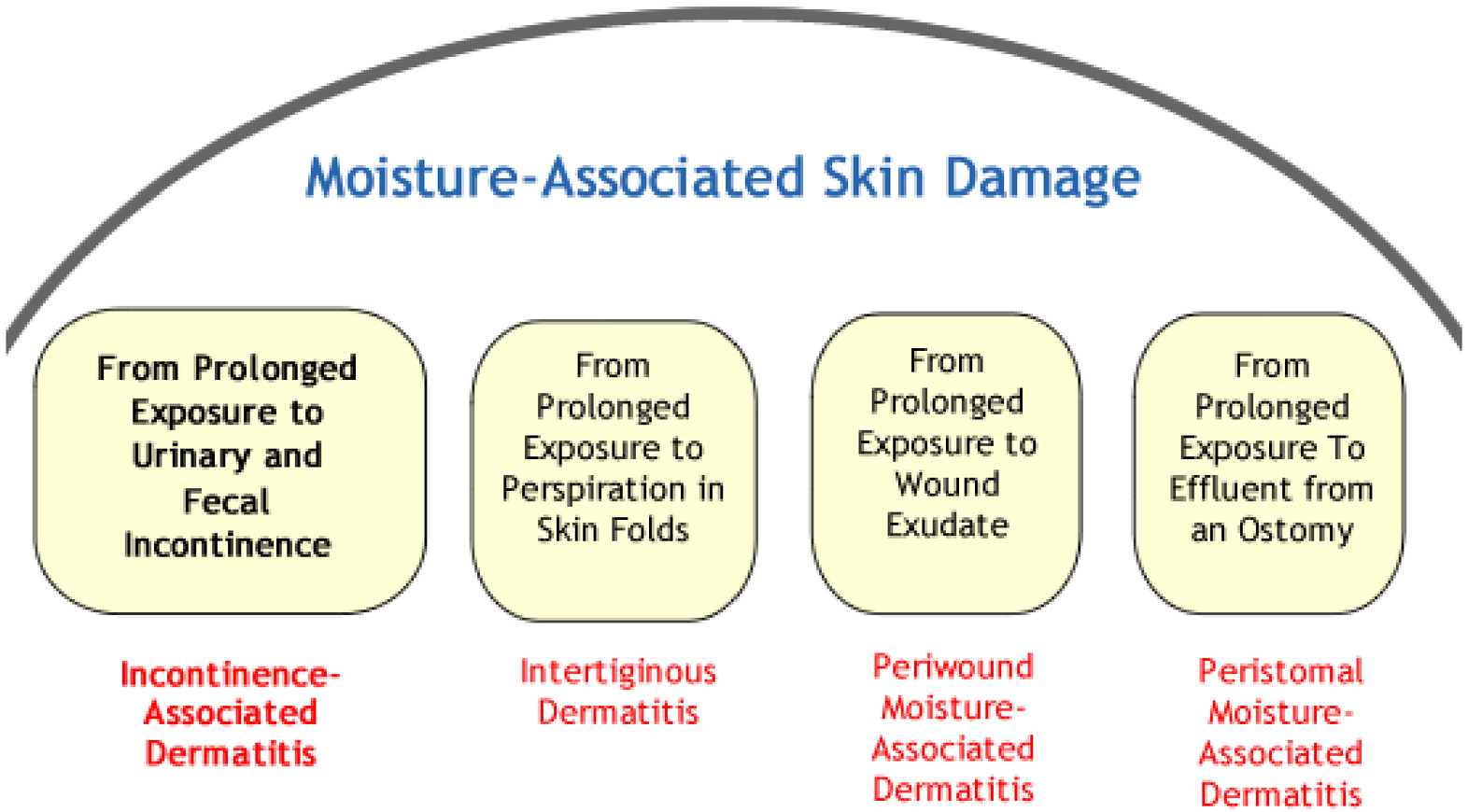
- Pressure
- Friction
- Sheer
- Moisture

Moisture

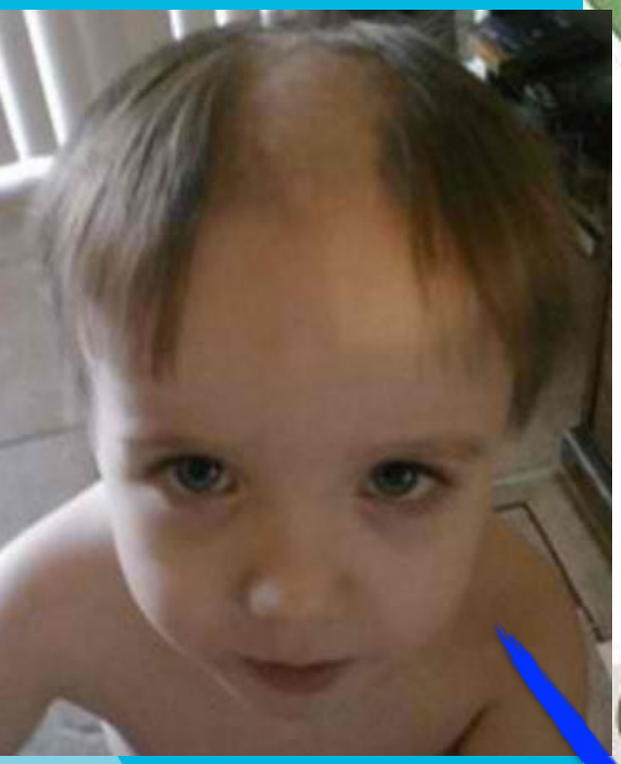
Moisture causes maceration & predisposes the skin to injury.

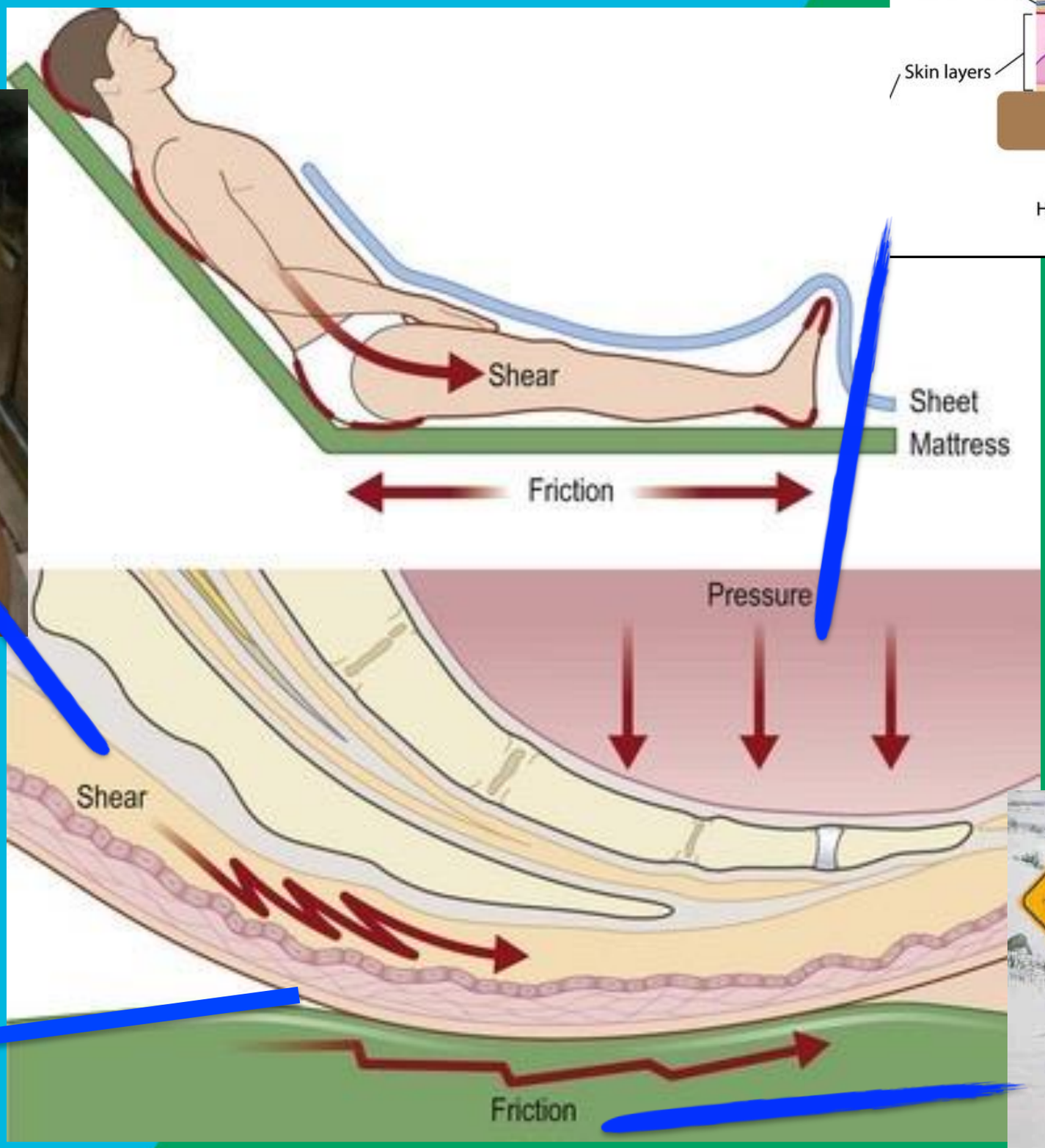
De-epithelialisation caused by trauma leads to transdermal water loss that creates maceration and adherence of the skin to clothing and any other supports in contact,.

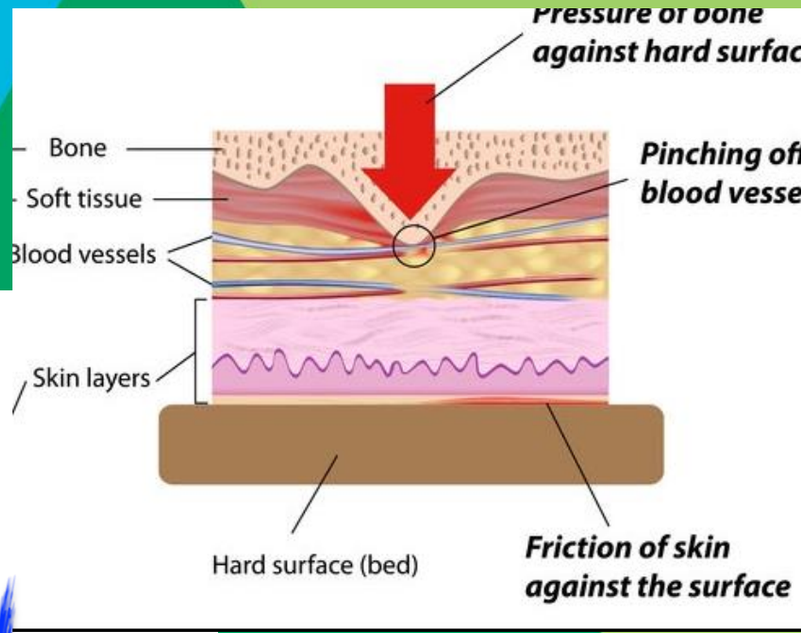
FRICTION!!!



Gray, M., et al. (2011). Moisture-associated skin damage: overview and pathophysiology. Journal of Wound, Ostomy and Continence Nursing, 38(3), 233-241.




Shear
Sheet
Mattress
Friction
Pressure
Shear
Friction


Pressure of bone against hard surfac
Bone
Soft tissue
Blood vessels
Pinching of blood vesse
Skin layers
Hard surface (bed)
Friction of skin against the surface

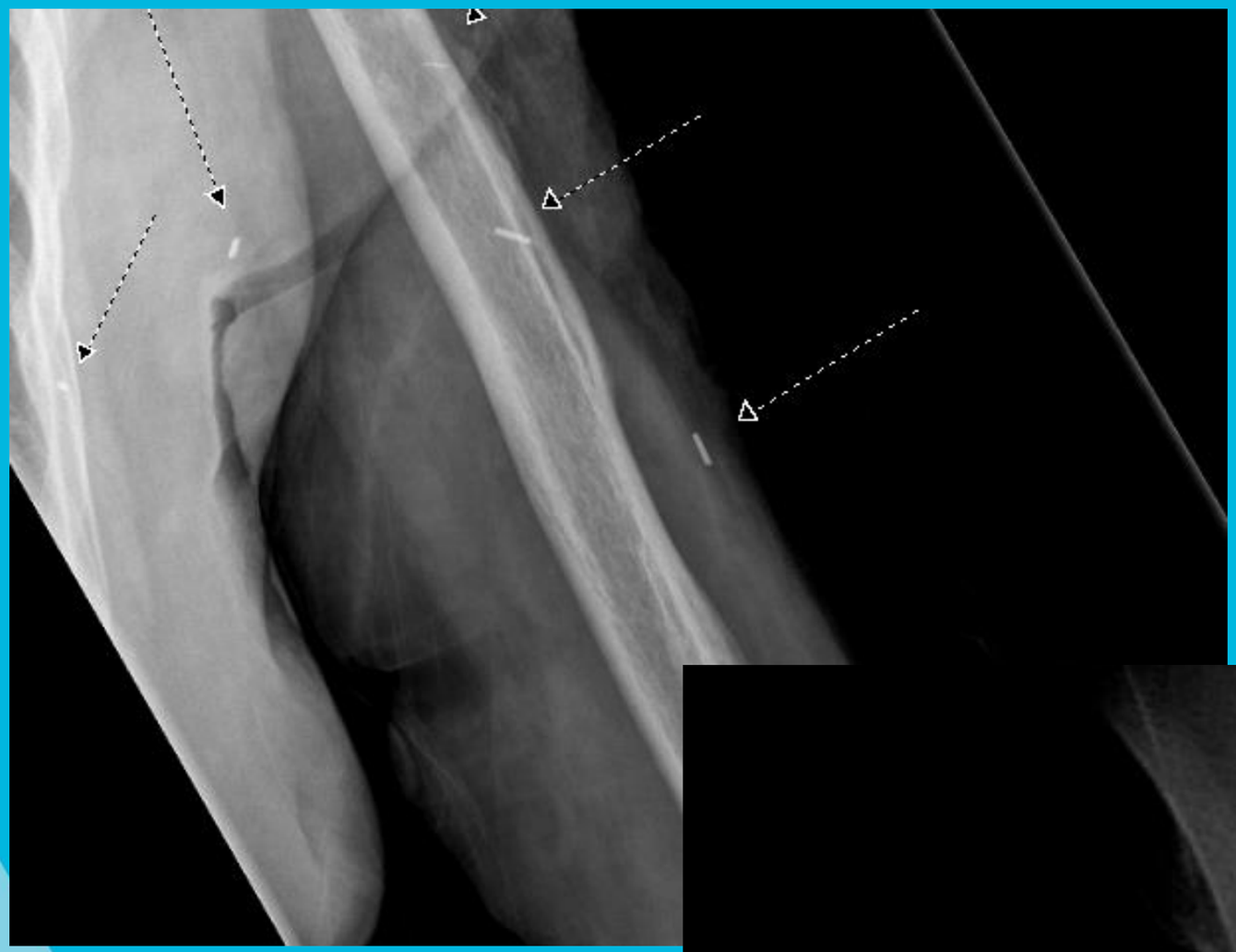

WATCH
FOR
ICE

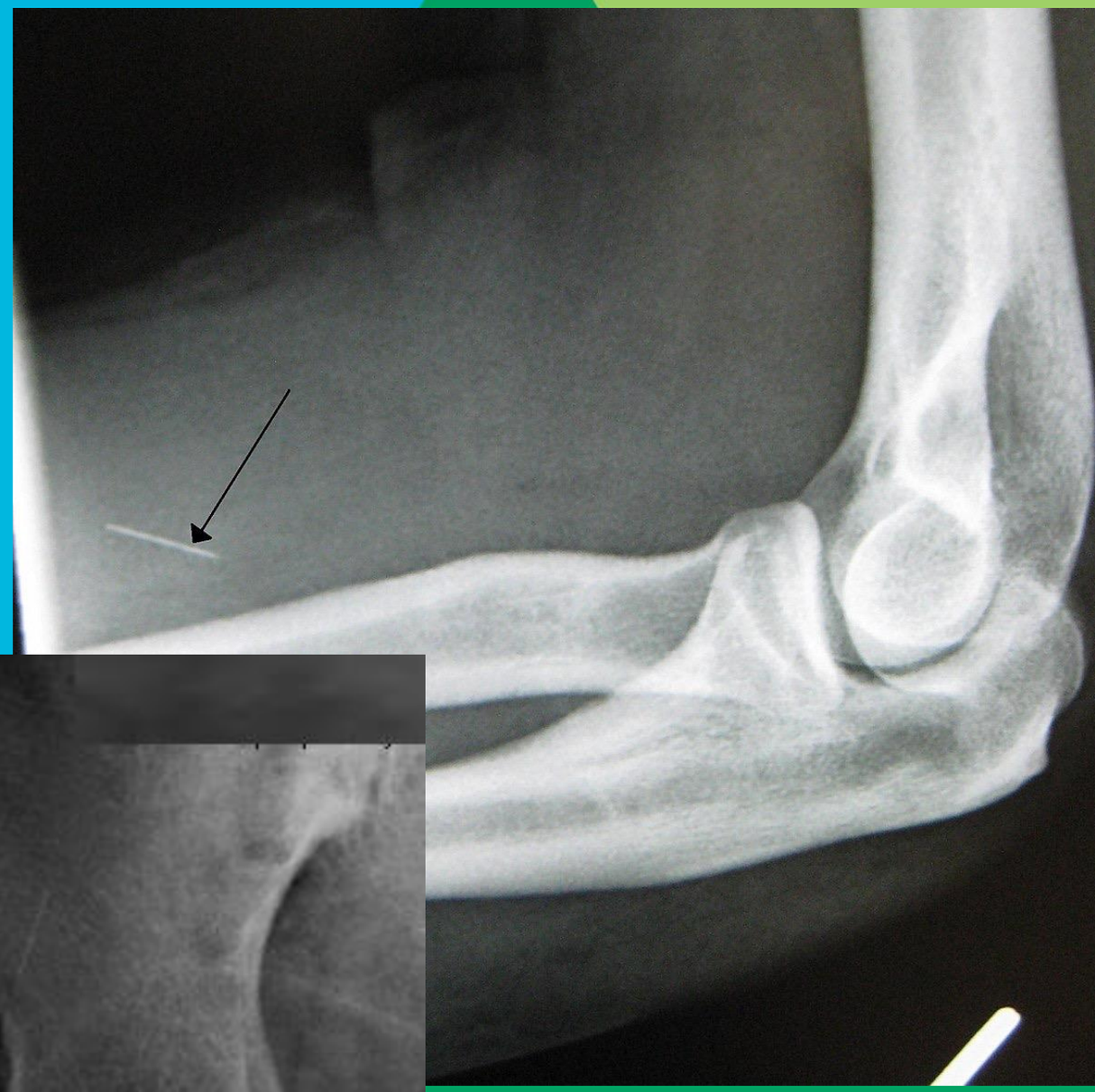
- [x] Pressure
- [x] Friction
- [x] Sheer
- [x] Moisture

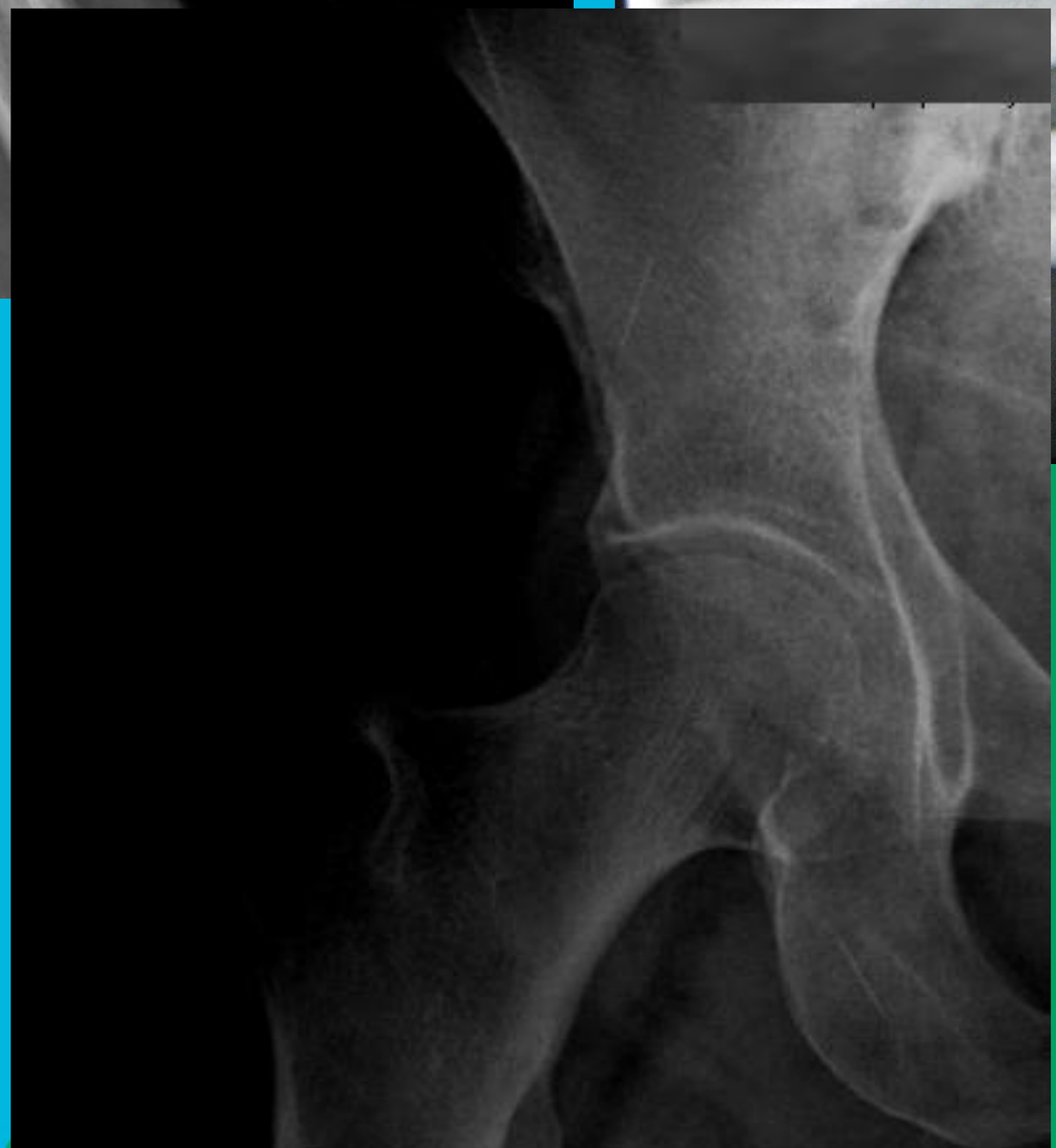
DID YOU KNOW…

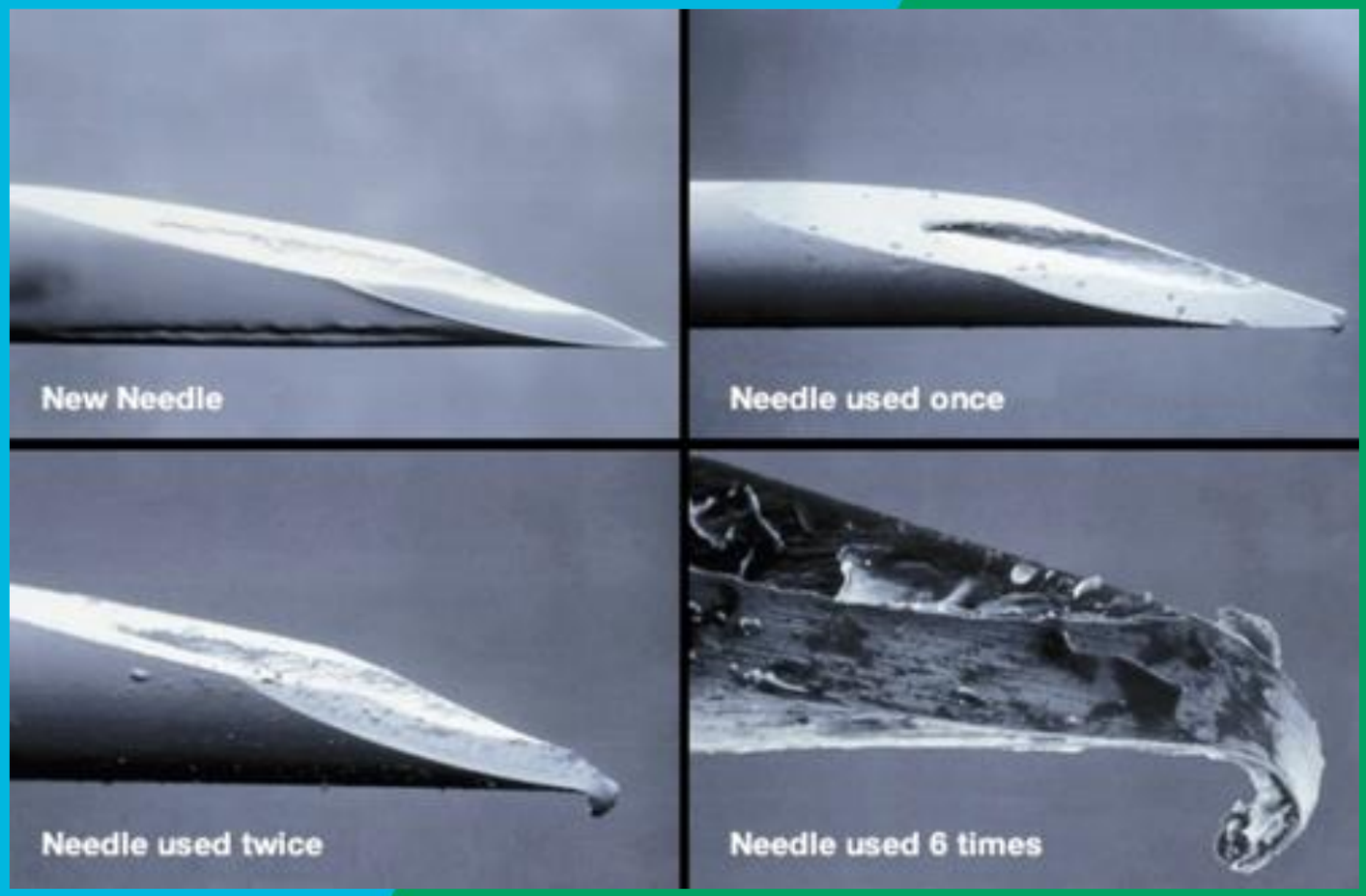
We ask our patients to:

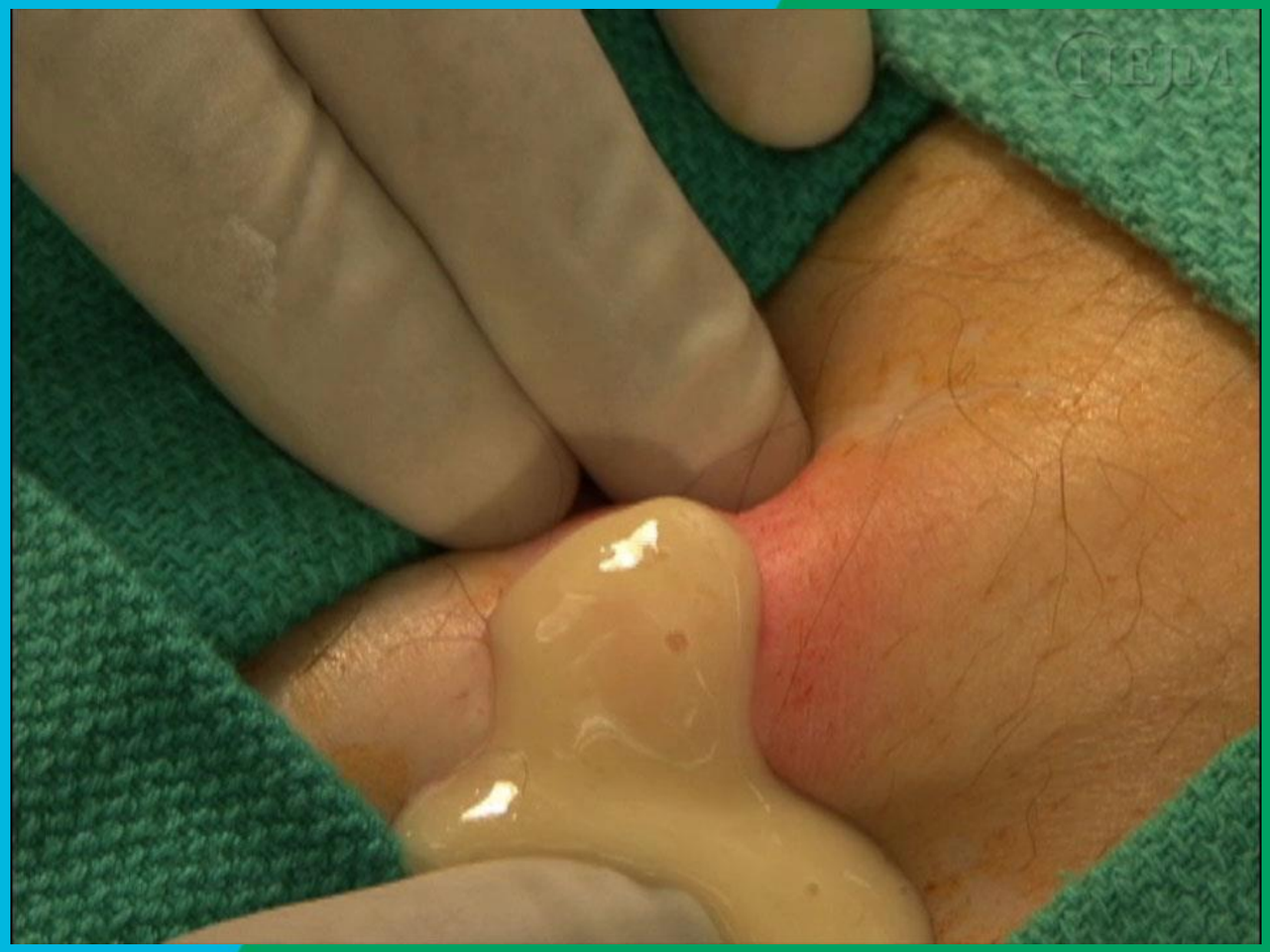
1) Cut their nails short and use surgical scrub brushes to clean under nails.
2) Use “pump soap” in the shower as well as when washing their wound followed by paper towels to blot it dry.
3) Wipe down cell phones, keyboards, doorknobs, light switches, keys…

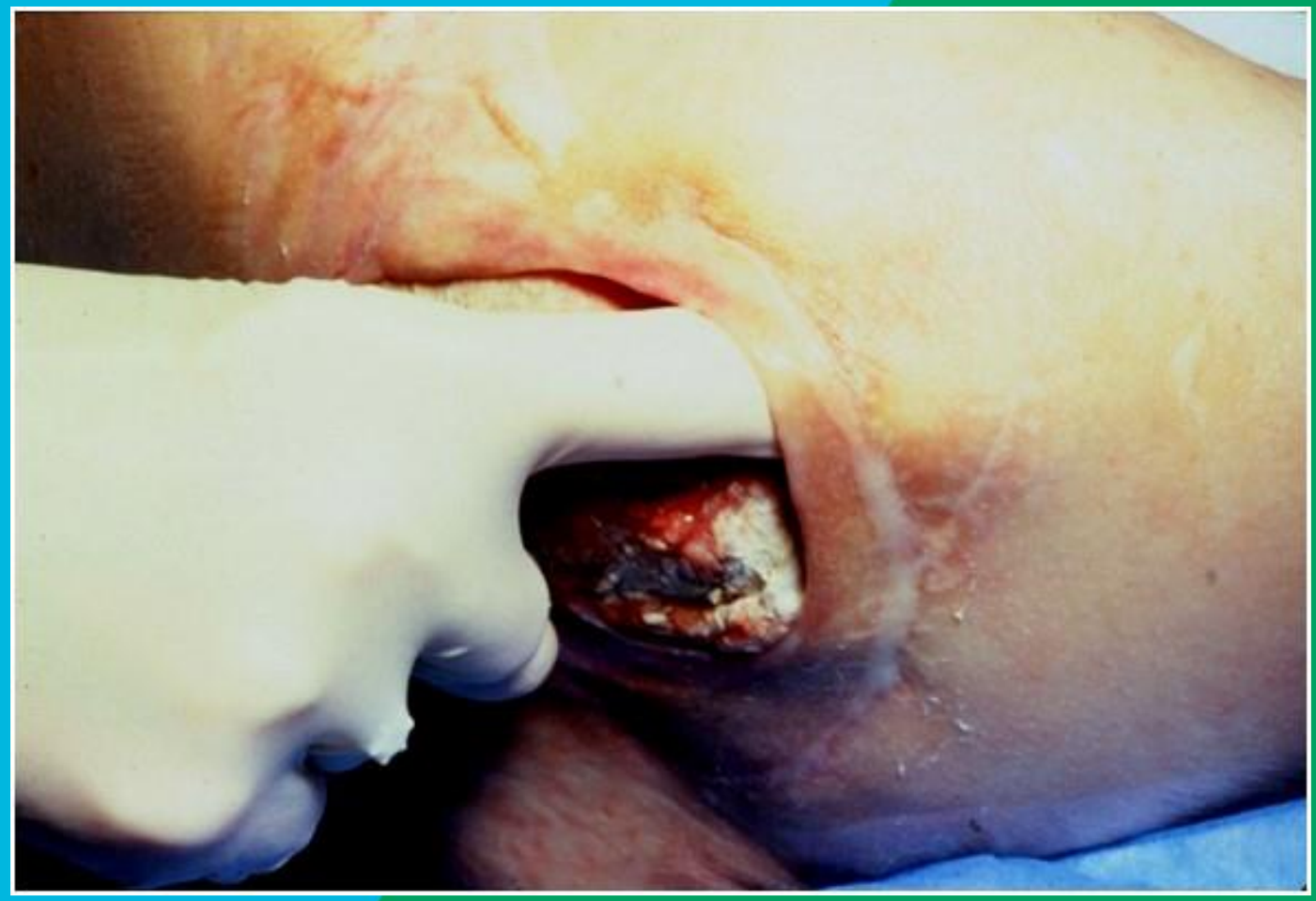







New Needle
Needle used once
Needle used twice
Needle used 6 times





THANK YOU

REFERENCES

Beck, F. et al, Prealbumin: A Marker for Nutritional Evaluation et al , Am Fam Physician. 2002 Apr 15;65(8):1575-1579

Enoch, S etal, CWound Bed Preparation: The Science Behind the Removal of Barriers to Healing Wounds. 2003;15(7)

Georgina Gethin Seamus Cowman Dinanda N Kolbach, Debridement for venous leg ulcers

The Cochrane Library, DOI: 10.1002/14651858.CD008599

Gardner SE, Frantz RA, Doebbeling BN. The validity of the clinical signs and symptoms used to identify localized wound infection. Wound Repair Regen 2001;9(3):178-86.Krizek T,

Fivenson D, Scherschun L. Clinical and economic impact of Apligraf for the treatment of nonhealing venous leg ulcers. Int J Dermatol. 2003;42:960–5

Fowler E, van Rijswijk L. Using wound debridement to help achieve the goals of care. Ost Wound Manag 1995;41(7A Suppl):23S-35S.

Joris JL, Dubner R, Hargreaves KM. Opioid Analgesia at Peripheral Sites: A Target for Opioids Released During Stress and Inflammation. Anesth Analgesia. 1987;66(12):1277-1281

Kapitzke et al, Endogenous opioid analgesia in peripheral tissues and the clinical implications for pain control, Ther Clin Risk Manag. 2005 December; 1(4): 279–297. Published online 2005 December. PMCID: PMC1661636

O'Meara S, Cullum N, Nelson EA, Dumville JC. Compression for venous leg ulcers Cochrane Database Syst Rev. 2012 Nov 14

Robson MC, Barbul A. Guidelines for the best care of chronic wounds. Wound Repair Regen. 2006;14:647–8. [PubMed]

Rook, J et al, Temporal effects of topical morphine application on cutaneous wound healing Anesthesiology. 2008 July; 109(1): 130–136.

Stein C. The Control of Pain in Peripheral Tissue by Opioids. New Eng J Med. 1995;332(25):1685-1690

Steed DL, Attinger C, Colaizzi T, Crossland M, Franz M, Harkless L, Johnson A, Moosa H, Robson M, Serena T, Sheehan P, Veves A, Wiersma-Bryant L. Guidelines for the treatment of diabetic ulcers. Wound Repair Regen. 2006;14:680–92. [PubMed]

Human Skin Wounds: A Major and Snowballing Threat to Public Health and the Economy. Wound Repair Regen. Wound Repair Regen. 2009 Nov–Dec; 17(6): 763–771 PMCI PMC2810192

Tennant F [2010B]. Use and Benefits of Topical Opioids by Intractable Pain Patients. Poster presented at 21st Annual Clinical Meeting of the American Academy of Pain Management; September 21-24, 2010; Las Vegas, Nevada.

Tandon OP, Mehta AK, Halder S, et al. Peripheral Interaction of Opioids and NMDA Receptors in Inflammatory Pain in Rats. Indian J Physiol Pharmacol. 2010;54(1):21-31

White, R. et al (2005) Critical colonisation: clinical reality or myth? Wounds UK; 1: 1, 94–95.
White, R. (2003) The wound infection continuum. In: White, R. (ed)