
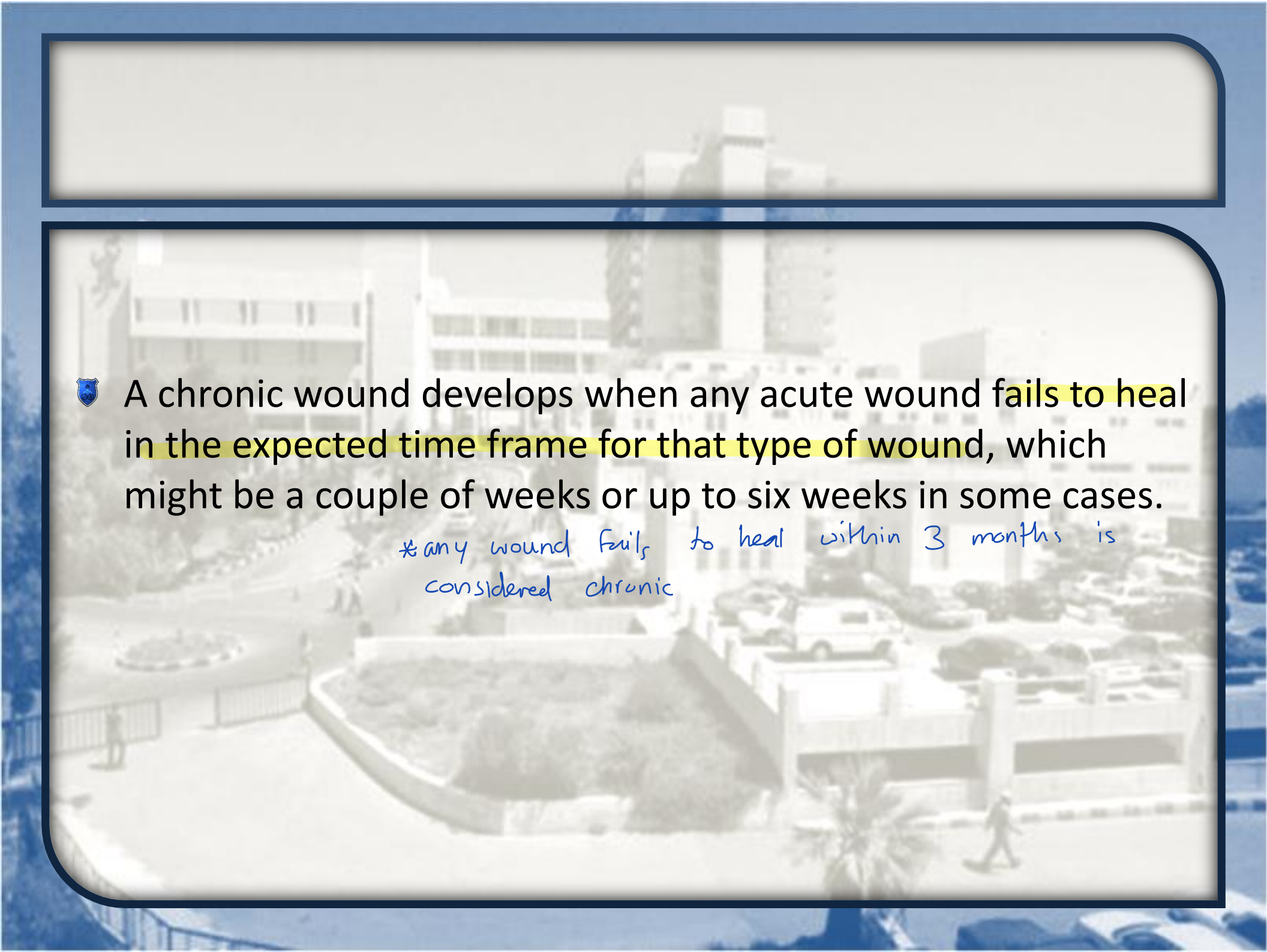




Chronic Wounds

 *Chronic wounds are defined as wounds that have failed to proceed through the orderly process that produces satisfactory anatomic and functional integrity or that have proceeded through the repair process without producing an adequate anatomic and functional result.*



A chronic wound develops when any acute wound fails to heal in the expected time frame for that type of wound, which might be a couple of weeks or up to six weeks in some cases.

*any wound fails to heal within 3 months is considered chronic

🛡️ The vast majority of chronic wounds can be classified into four categories:

- Ischaemic Arterial Ulcers.
- Venous Stasis Ulcers.
- Diabetic wounds. → combination of ischemic and neuropathic but the most common cause for this type of wounds is neuropathic (trophic)
- Pressure ulcers.

🛡️ A small number of wounds that do not fall into these categories may be due to causes such as radiation poisoning, ischemia, or malignancy.

Scope of the problem

 Incidence 2.7% - 29.5%

 High risk patients:

- Quadriplegics
- Neurosurgery
- Orthopedic..post-op hips..up to 66%
- Critical care MICU/CCU/SICU...33% - 41%
- Prolonged anaesthesia time
- Debilitated and elderly(age > 70)

Ischemic ulcers

Ischemic arterial ulcers occur due to a lack of blood supply and are painful at presentation. → Ischemia one of the most powerful stimuli to pain

They usually are associated with other symptoms of peripheral vascular disease, such as intermittent claudication, rest pain, night pain, and colour changes.

it's worse than night pain

loss of gravity, lower limbs at the level of the heart + stenosed vessels → no enough blood to the limbs

Clamping pain usually induced by walking (especially for long distances) relieved by rest
→ Pathophysiology: no supply meeting the oxygen demand

shut down of Na^+/K^+ pump ← drop in ATP ← anaerobic metabolism ← (2 ATP not 36)

and accumulation of
lactic acid

* Ankle-brachial index in healthy people usually = 0.9-1

* ≤ 0.9 = in diabetic patients = 1-1.1 \rightarrow because sound waves transmit

faster in solids than
liquids

On examination, there may be diminished or absent pulses
with decreased ankle-brachial index and poor formation of
granulation tissue. Other signs of peripheral ischemia, such as
dryness of skin, hair loss, scaling, and pallor can be present.

different
between
diabetics
and
non-diabetics

as diabetics have more atherosclerosis

\rightarrow causes elevation of ankle-brachial index in
diabetics

The wound itself usually is shallow with smooth margins, with
pallor of base and surrounding skin might be present.



→ the base is white with minimal amount of granulation tissue

→ Amputation stump ulcer of a previously amputated big toe

→ Floor of granulation tissue, stuff and hard of metatarsal edges: punched out

this redness indicates cellulitis that needs to be managed



الموميّة
mummification of the tissue

this black discoloration is from from

→ 2nd and 3rd toes: gangrenous tissue

gross descriptive term

dry (when infected)
wet

Risk of ulcer formation with high metabolic rate → progression of ischemia

(1) Early management with amputation
(2) Expected management with autoamputation

Management of ischemic ulcers

* Wet gangrene: surgical emergency → بحطه على فوهة
بفتح الهمزة
بفتح الهمزة

↓
Pain is a powerful
stimuli for angiogenesis
So the ulcer
formed is
highly vascular

🛡️ The management of these wounds is too-pronged and includes **revascularization and wound care**.

– It depends on the severity of the underlying arterial insufficiency.

✓ 🛡️ The affected region can sometimes be revascularized via vascular bypass or angioplasty.

→ Revascularization is very important so if there's a stenosed area we should perform bypass

✓ 🛡️ * Sometime all parts of blood supply is stenosed and here bypass isn't beneficial. If infection is present, appropriate antibiotics are prescribed.

When proper blood flow is established, debridement is performed.

If the wound is plantar (on walking surface of foot), patient is advised to give rest to foot to avoid enlargement of the ulcer.

↳ offloading technique → offload pressure on the plantar aspect to give the ulcer a chance to heal

Proper glycemic control in diabetics is important.

(not only ischemic insult also a neuropathic one)

Smoking should be avoided to aid wound healing.

↳ a single cigarette can lead to vasoconstriction for 90 minutes

Venous stasis ulcer

- 🛡️ The clinically characteristic picture is that of an ulcer that fails to re-epithelialise despite the presence of adequate granulation tissue.
- 🛡️ Venous stasis occurs due to the incompetence of either the superficial or deep venous systems.
 - ***Chronic venous ulcers usually are due to the incompetence of the deep venous system and are commonly painless.***

*Usually these ulcers are in the gaiter area (medial aspect of the leg just above the medial malleolus)



- Venous circulation of the lower limb: — deep
— superficial

→ lesser and greater saphenous

* Usually the flow from the superficial circulation towards the deep circulation

→ main venous drainage of the lower limbs while the upper limbs is the superficial

🛡️ Stasis ulcers tend to occur at the sites of incompetent perforators, **the most common being above the medial malleolus, over Cockett's perforator.**

🛡️ The wound usually is shallow, with irregular margins and pigmented surrounding skin.



→ caused by hemosiderin deposition from RBC hemolysis

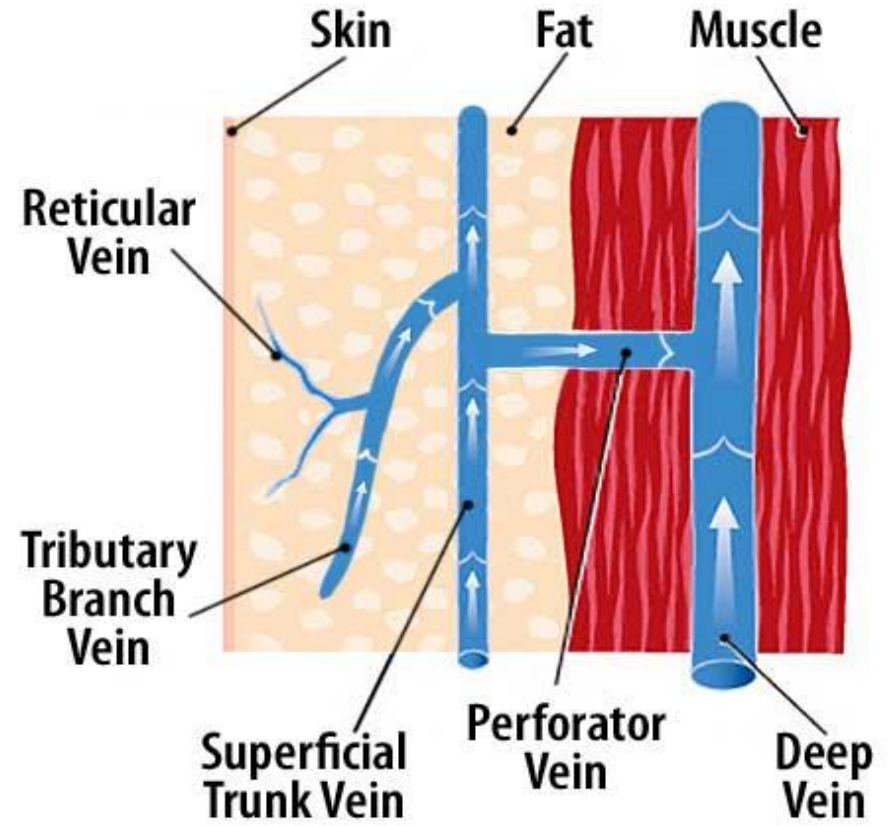




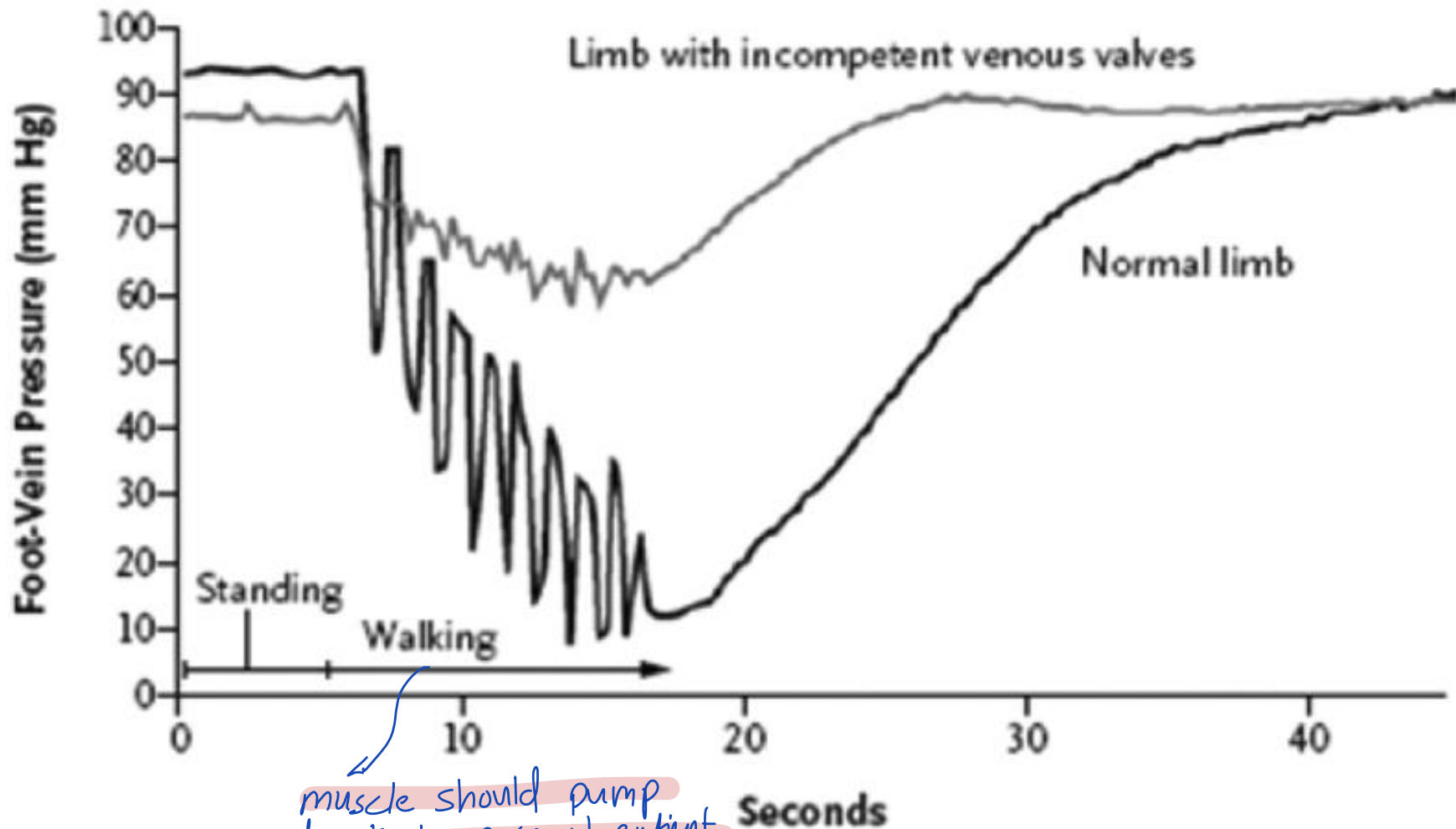
examined to check the site of incompetence especially in patients with varicose veins

- Hunterian perforator(s) in proximal thigh
- Dodd perforator(s) in distal thigh
- Boyd's perforator(s) below the knee
- Cockett's perforators of the posterior arch vein

Chronic venous insufficiency → incompetent venous valves
 - Peripheral heart & muscles and venous valves



*DVT & thrombus will form on the valve destroying it



muscle should pump
back in normal patient
but in chronic venous insufficiency
it's still high

(Reproduced from Coleridge Smith¹⁷ with the permission of the publisher)

*This pressure will more toward the superficial circulation that is not used to this form of pressure \Rightarrow vessels become dilated and tortuous and RBCs with time will escape from intravascular to interstitial fluid

-Any RBC outside the intravascular compartment is considered a foreign body



fibrosis in the lower third of the leg

Management

- 🛡️ The cornerstone of treatment of venous ulcers is compression therapy.
 - It can decrease blood vessel diameter and pressure, which increases their effectiveness, preventing blood from flowing backwards.
- 🛡️ Compression is also used to decrease release of inflammatory cytokines, lower the amount of fluid leaking from capillaries and therefore prevent swelling, and prevent clotting by decreasing activation of thrombin and increasing that of plasmin.

Most venous ulcers can be healed with perseverance and by addressing the venous hypertension.

Recurrences are frequent. Therefore, compression stockings are advised to prevent the formation of new ulcers in people with a history of the same.

* Most likely the pathophysiology of the disease is the metalloproteinases presence preventing it from complete healing

Diabetic Foot ulcer

One of the major complications of uncontrolled **Diabetes Mellitus**,

– Diabetic Foot Ulcers are a result of impedance of Wound Healing process due to a prolonged inflammatory phase.

Diabetes causes neuropathy, which inhibits nociception and the perception of pain. Thus patients may not initially notice small wounds to legs and feet, and may therefore fail to prevent infection or repeated injury.

*Wound healing has 3 phases: ① Inflammatory ② Proliferative ③ Remodeling

In Diabetic foot
Ulcer it's prolonged

Further, diabetes causes immune compromise and damage to small blood vessels, preventing adequate oxygenation of tissue, which can cause chronic wounds.

Pressure also plays a role in the formation of diabetic ulcers.

Diabetic patients are immunocompromised → both cellular and humoral immune systems are affected
↓
more affected especially neutrophils

The background of the slide is a faded, high-angle photograph of a large, multi-story hospital building with a central tower and several wings. The building is surrounded by a parking lot with cars and some greenery. The entire slide has a blue border with rounded corners.


🛡️ Once ulceration occurs, the chances of healing are poor.

🛡️ The treatment of diabetic wounds involves local and systemic measures.

- Achievement of adequate blood sugar levels is very important.
- Most diabetic wounds are infected.
 - Eradication of the infectious source is paramount to the success of healing.



Foot ulcers in diabetes require multidisciplinary assessment, usually by podiatrists, diabetes specialists and surgeons.



Treatment consists of appropriate bandages, antibiotics, debridement, arterial revascularisation and platelet-rich fibrin therapies.

base filled with granulation tissue and thickened edges (skin protecting itself)



↓
Called trophic because they used to think that neuropathology leads to ↓ nutrition so they become trophic

* Capillary Perfusion Pressure concept - 2

* If the pressure is above hydrostatic pressure of the capillary causing collapse of the vessel → when the tissue is compressed by both bony prominence and underlying pressure. → ischemic

Decubitus / Pressure Ulcer

→ immobilized patients

🛡️ A pressure ulcer is a localized area of tissue necrosis that develops when a soft tissue is compressed between a bony prominence and an external surface.


* Ischemia for tissues is different regarding the time due to their metabolic rate (↑ metabolic rate → ↓ ischemic time) → for example: brain 5-6 minutes
muscles 6 hours / skin up to 12 hours

🛡️ Pressure ulcer formation is accelerated in the presence of **friction, shear forces, and moisture.**

🛡️ Other contributory factors in the pathogenesis of pressure ulcers **include immobility, altered activity levels, altered mental status, chronic conditions, and altered nutritional status.**

* for example a patient with RTA, wheelchaired patients, ICU patients, stroke patients

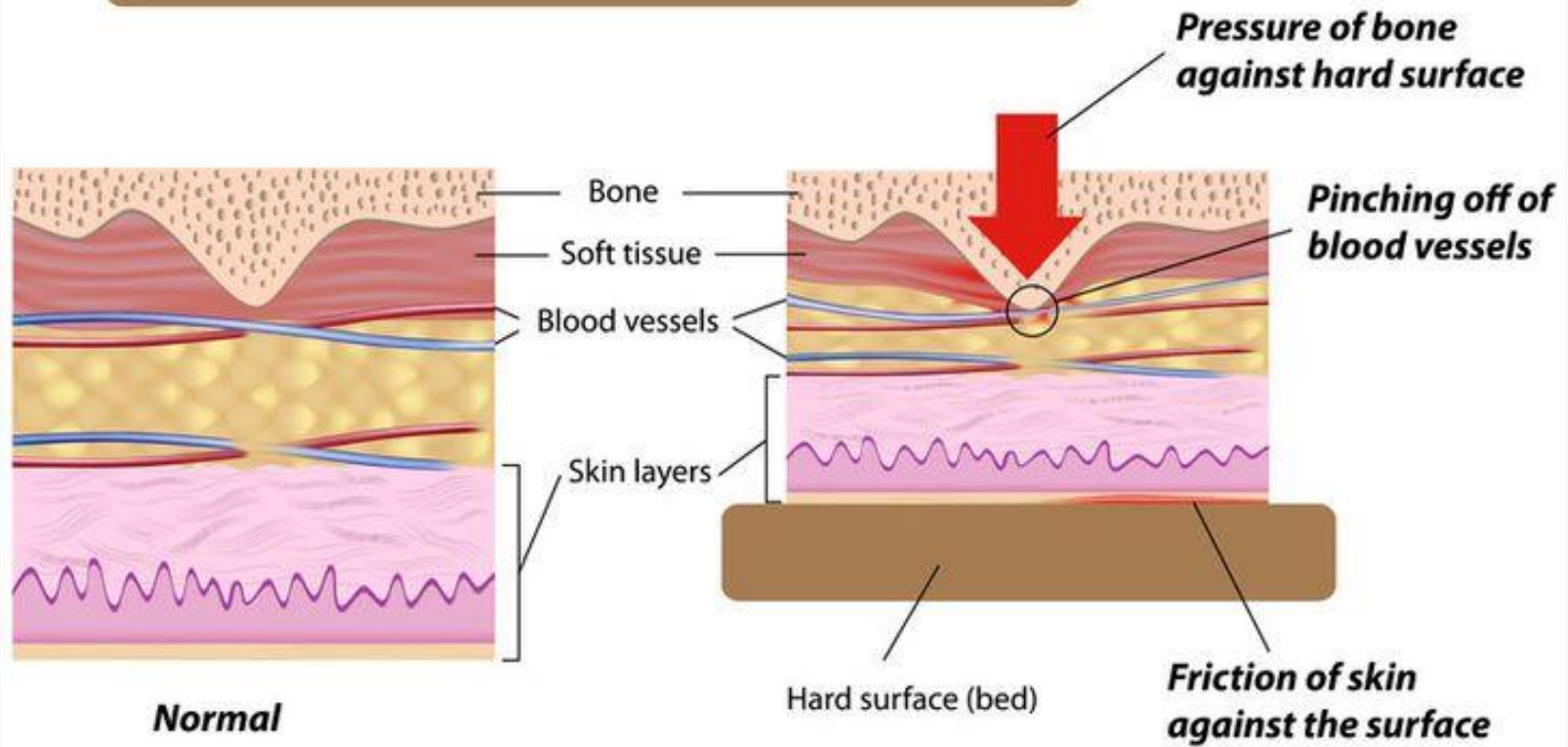
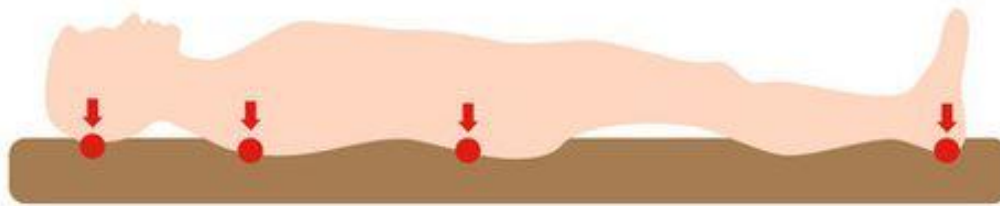
→ depending on the location of the patient

 The most common sites are the skin overlying the sacrum, coccyx, heels or the hips, but other sites such as the elbows, knees, ankles or the back of the cranium can be affected

* Factors that ↑ ischemia:

- ① friction
- ② shearing forces (kinking of the vessels of the skin)


Anatomy of A Pressure Sore



ICEBERG principle

- 🛡️ Pressure is distributed in a roughly upright cone, expanding outward and down through the subdermal tissues:
- 🛡️ Eschar indicates Stage 3 or higher
- 🛡️ Subcutaneous wound is larger than the visible area of eschar



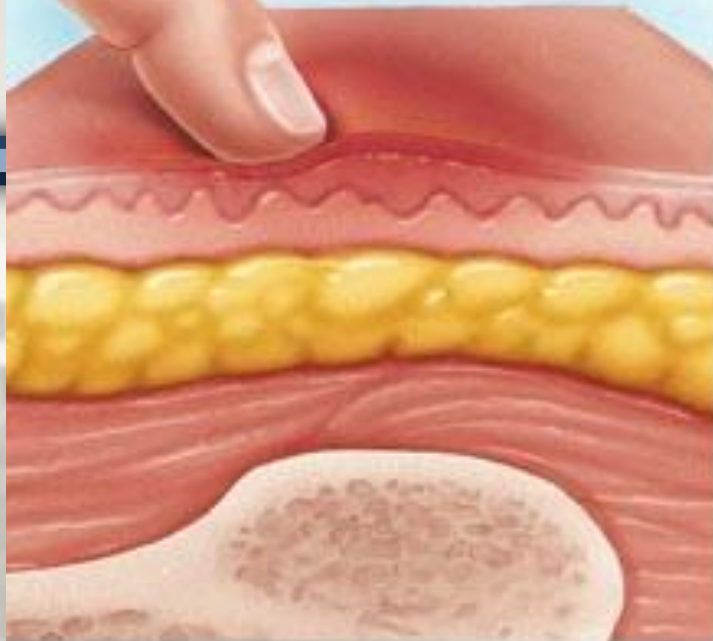


Pressure ulcers are divided into the following stages depending on severity:

- **Stage I: Intact skin with non-blanchable redness of a localized area usually over a bony prominence.**
- **Stage II: Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough.**
- **Stage III: Full thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed.**
- **Stage IV: Full thickness tissue loss with exposed bone, tendon or muscle.**

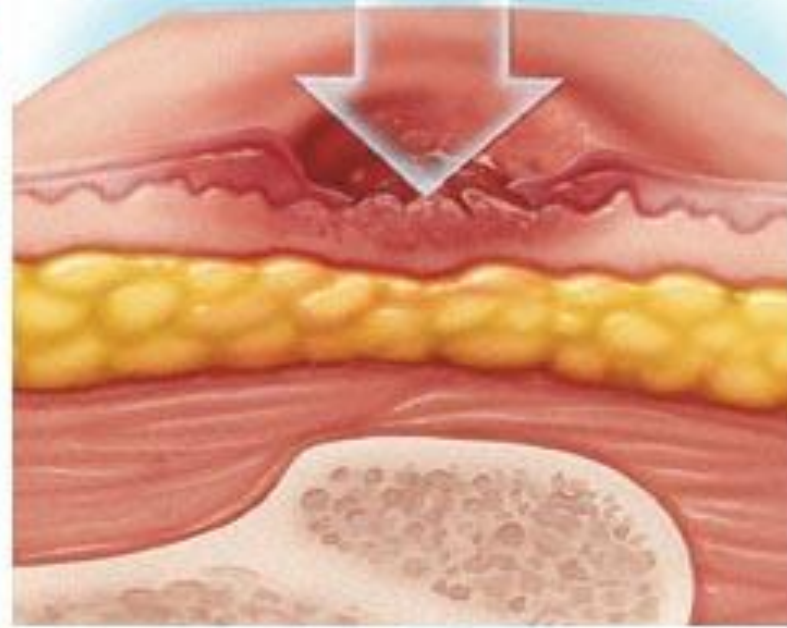
Stage 1

Skin is unbroken
but inflamed



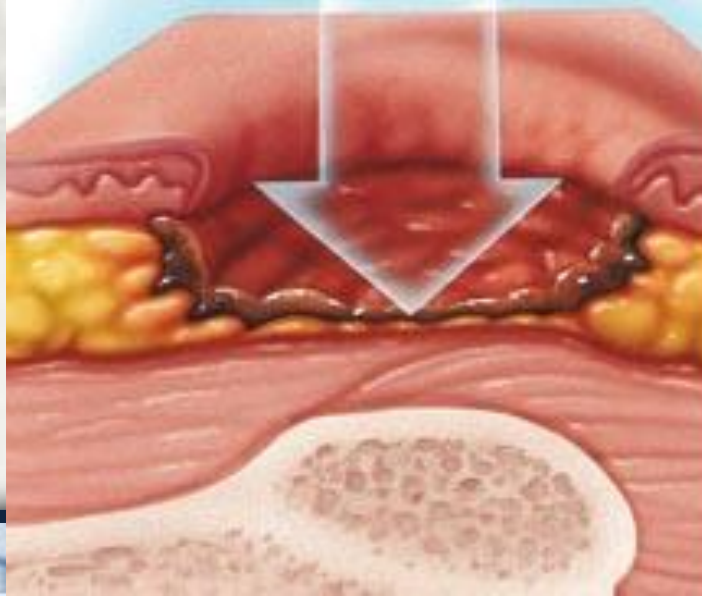
Stage 2

Skin is broken to
epidermis or dermis



Stage 3

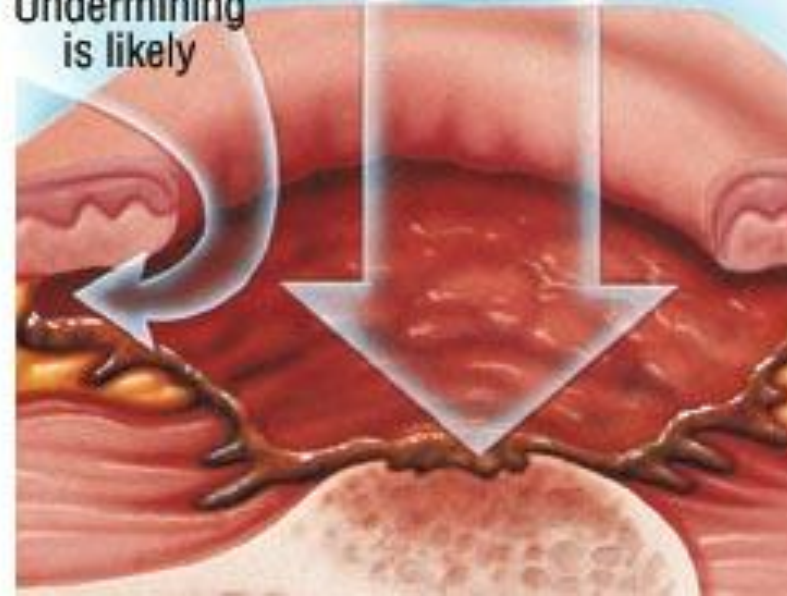
Ulcer extends to
subcutaneous fat layer

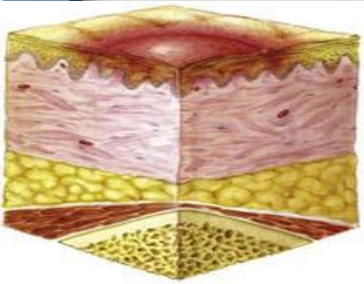


Stage 4

Ulcer extends to
muscle or bone

Undermining
is likely

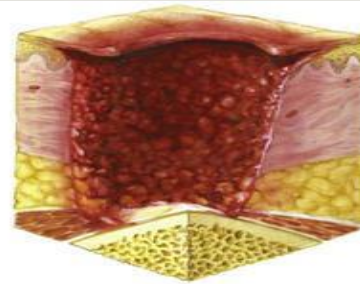




Stage 1



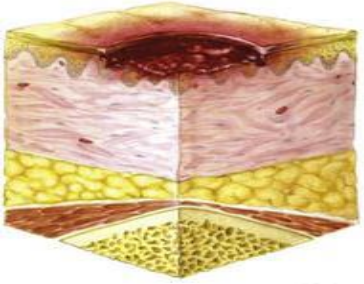
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Stage 4



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Stage 2



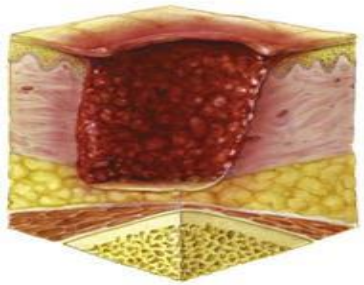
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Unstageable



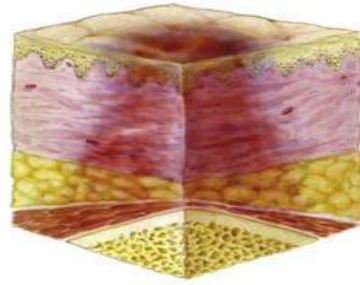
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Stage 3



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Suspected deep tissue injury



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PUPPS 3 – The National Pressure Ulcer Advisory Panel (NPUAP) Pressure Ulcer Staging System

Pressure ulcers are classified by the depth of tissue damage present.

The following staging of pressure ulcers are recommended for use by the Australian Wound Management Association, which is consistent with the recommendations of the National Pressure Ulcer Advisory Panel (NPUAP) U.S.A.

Stage 1

Observable pressure related alteration of intact skin whose indicators as compared to the adjacent or opposite area of the body may include changes in one or more of the following: skin temperature (warmth or coolness), tissue consistency (firm or boggy feel) and/or sensation (pain, itching). The ulcer appears as a defined area of persistent redness in lightly pigmented skin, whereas in darker skin tones, the ulcer may appear with persistent red, blue or purple hues.



STAGE 1



Please note: heel pressure ulcer covered with a film dressing

Stage 2

Partial thickness skin loss involving epidermis and/or dermis. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.



STAGE 2



Stage 3

Full thickness skin loss involving damage or necrosis of subcutaneous tissue that may extend down to but not through underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.



STAGE 3



Stage 4

Full thickness skin loss with extensive destruction, tissue necrosis or damage to muscle, bone, or supporting structures (for example, tendon or joint capsule). Undermining and sinus tracts may also be associated with Stage 4 pressure ulcers.



STAGE 4



→ most important part of management is to prevent

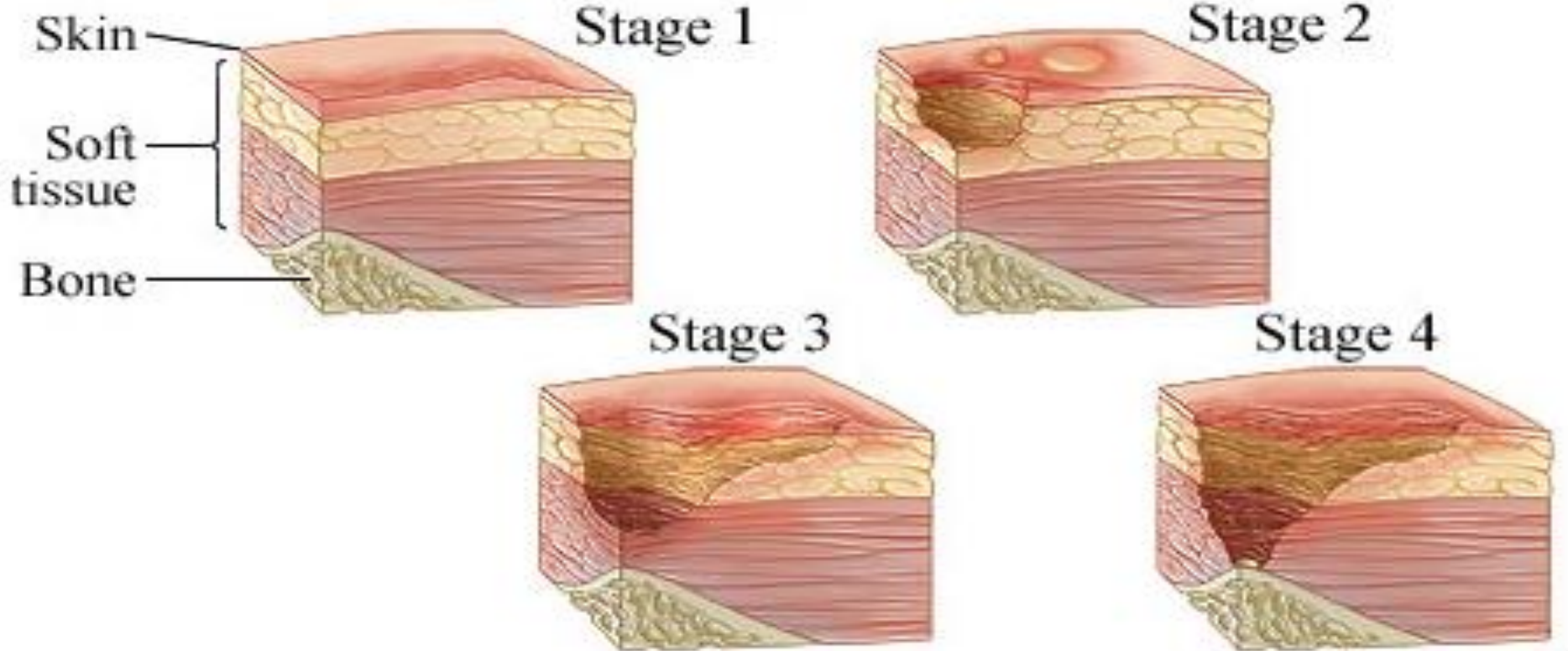


MANAGEMENT

- The most important care for a person at risk for pressure ulcers and those with bedsores is the redistribution of pressure so that no pressure is applied to the pressure ulcer.
- Debridement and Dressing is helpful in existing cases.
- Stage 1&2 → Day care settings only
- Stage 3&4 → need surgeons in case debridement and cleaning and planning for closure comes as clean as possible)

fecal and urine divergen by Foley catheter and colonostomy respectively

Stages of Pressure Sores





**Did you turn
me today?**

**People who can't move
themselves need your help.**

For more information on pressure ulcer prevention please visit
www.preventpressureulcers.ca



Canadian Association
of Wound Care  Association canadienne
des soins des plaies
www.cawc.ca

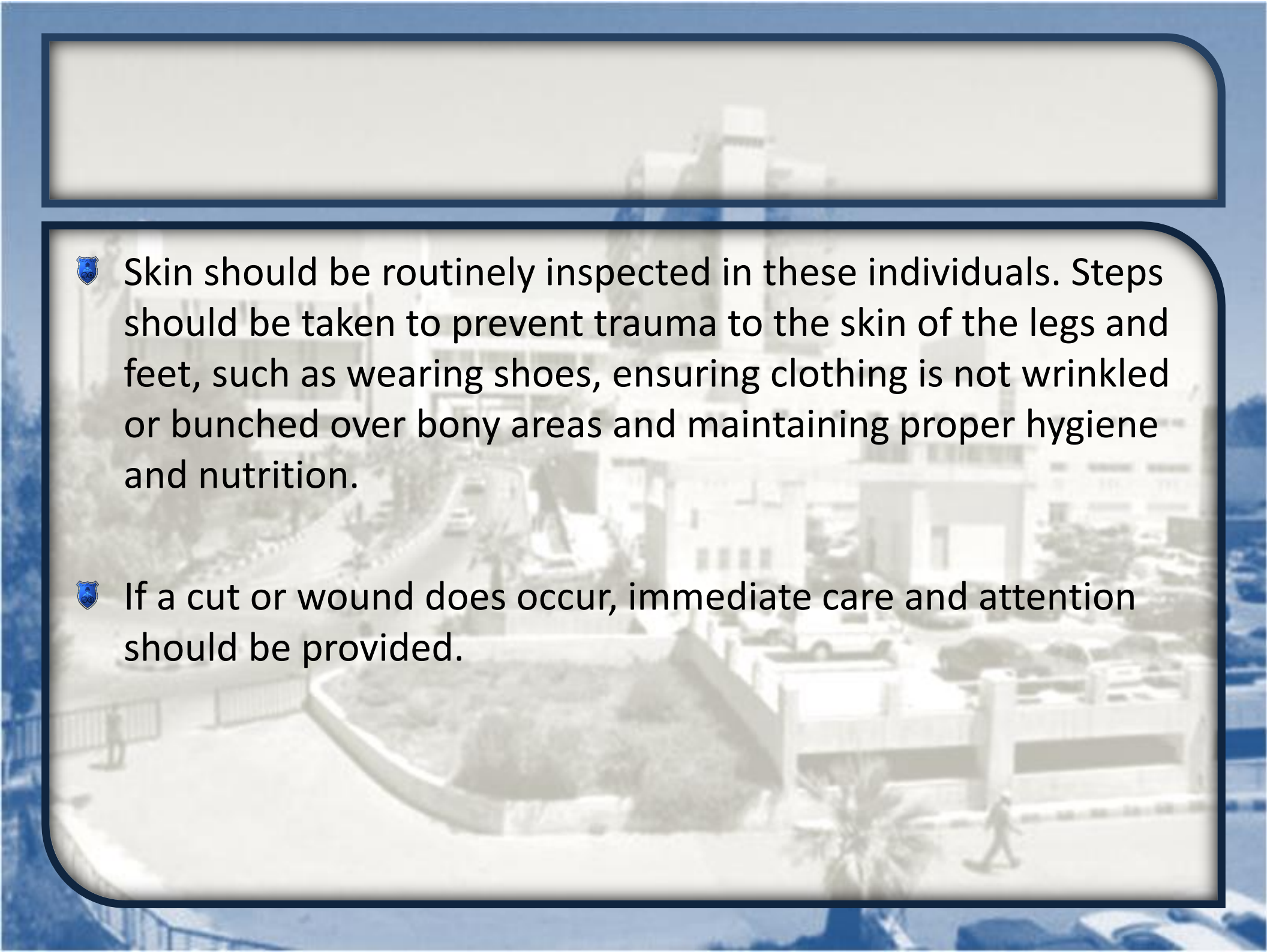
*→ Prevention
in term
of
management*

Malignant transformation of chronic wounds

- Any wound that does not heal for a prolonged period of time is prone to malignant transformation (Marjolin Ulcer)
- Malignant wounds are differentiated clinically from non-malignant wounds by the presence of overturned wound edges.
- In patients with suspected malignant transformations, biopsy of the wound edges must be performed to rule out malignancy.
- Cancers arising de novo in chronic wounds include both squamous and rarely basal cell carcinomas.

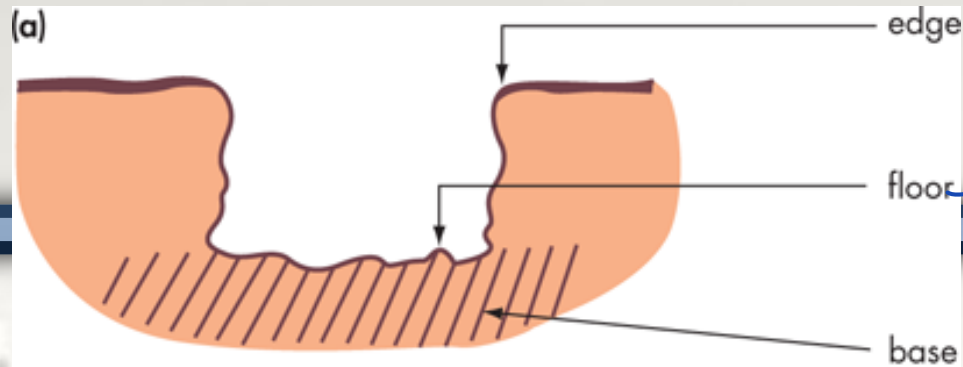
Chronic Wounds

- ❗ Chronic wounds are much easier to prevent than to treat.
- ❗ The best way to prevent a chronic wound is to actively and appropriately manage chronic medical conditions such as diabetes, high blood pressure, venous insufficiency and peripheral neuropathy.







🛡️ Skin should be routinely inspected in these individuals. Steps should be taken to prevent trauma to the skin of the legs and feet, such as wearing shoes, ensuring clothing is not wrinkled or bunched over bony areas and maintaining proper hygiene and nutrition.

🛡️ If a cut or wound does occur, immediate care and attention should be provided.



what we see

what we feel

Edge	Example
 <p>① punched out</p>	<p>trophic ulcer arterial ulcer syphilis</p>
 <p>② undermined</p>	<p>pressure injury e.g. bed sore</p>
 <p>③ everted</p>	<p>squamous cell carcinoma</p>
 <p>④ rolled</p>	<p>basal cell carcinoma</p>

tip of the iceberg because the injury is deep

Source: John Murtagh, Jill Rosenblatt: *John Murtagh's General Practice*, 6e: www.murtagh.mhmedical.com
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