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Wound care 101 - Concurrent Breakout Session Repeated

Saturday, 11 June 2011	Start 8:30am	Duration: 50mins	Van Gogh
	Start 9:25am	Duration: 50mins	Van Gogh

Wound Management 2011

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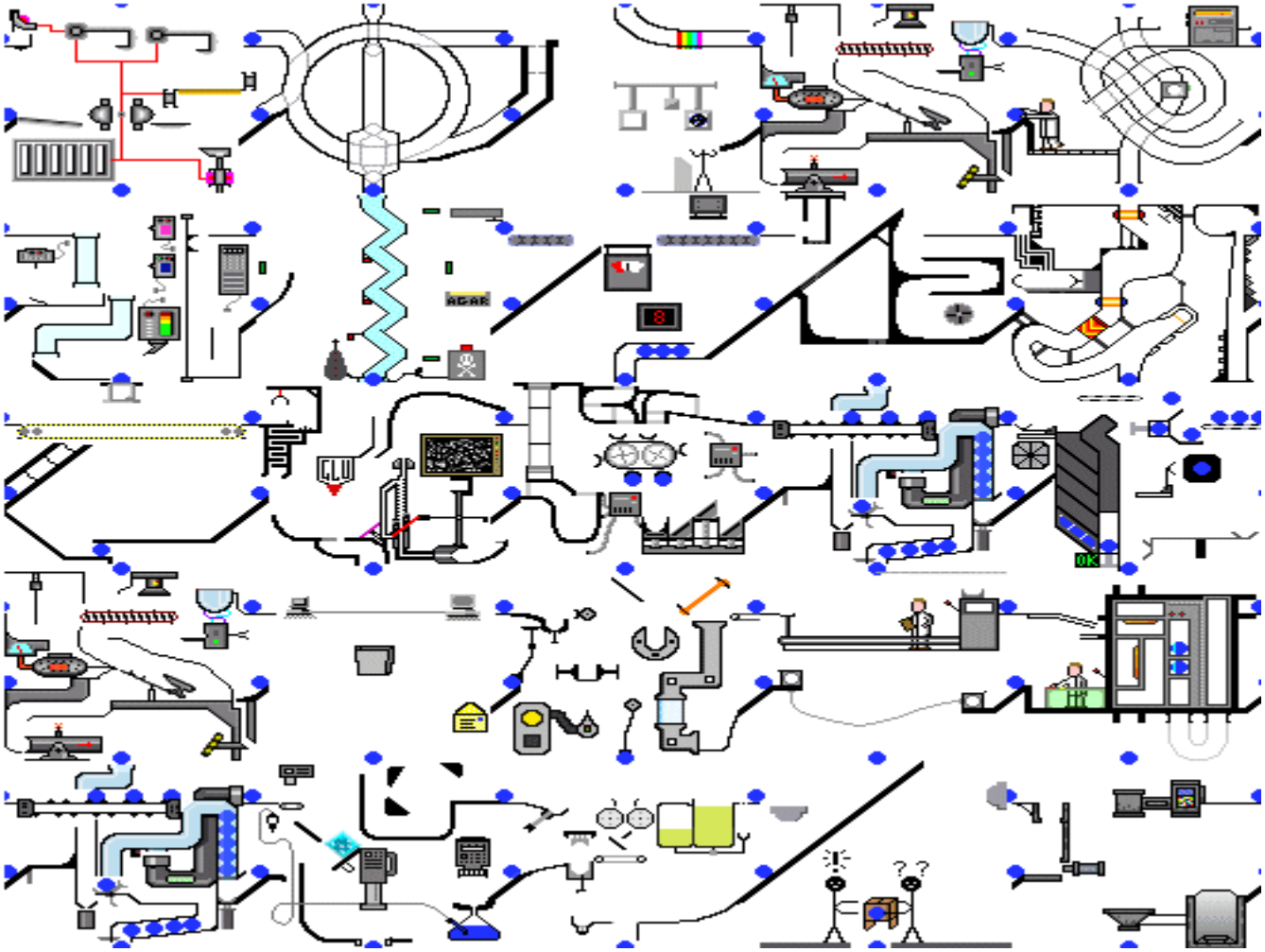
total care health services LTD
for quality care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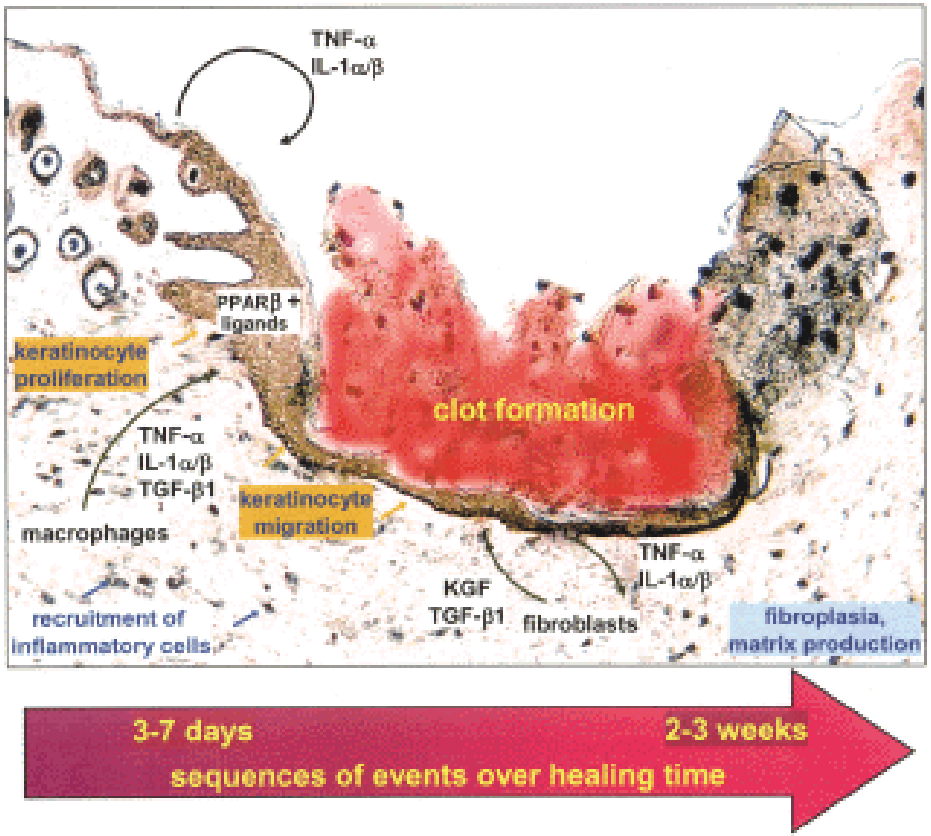
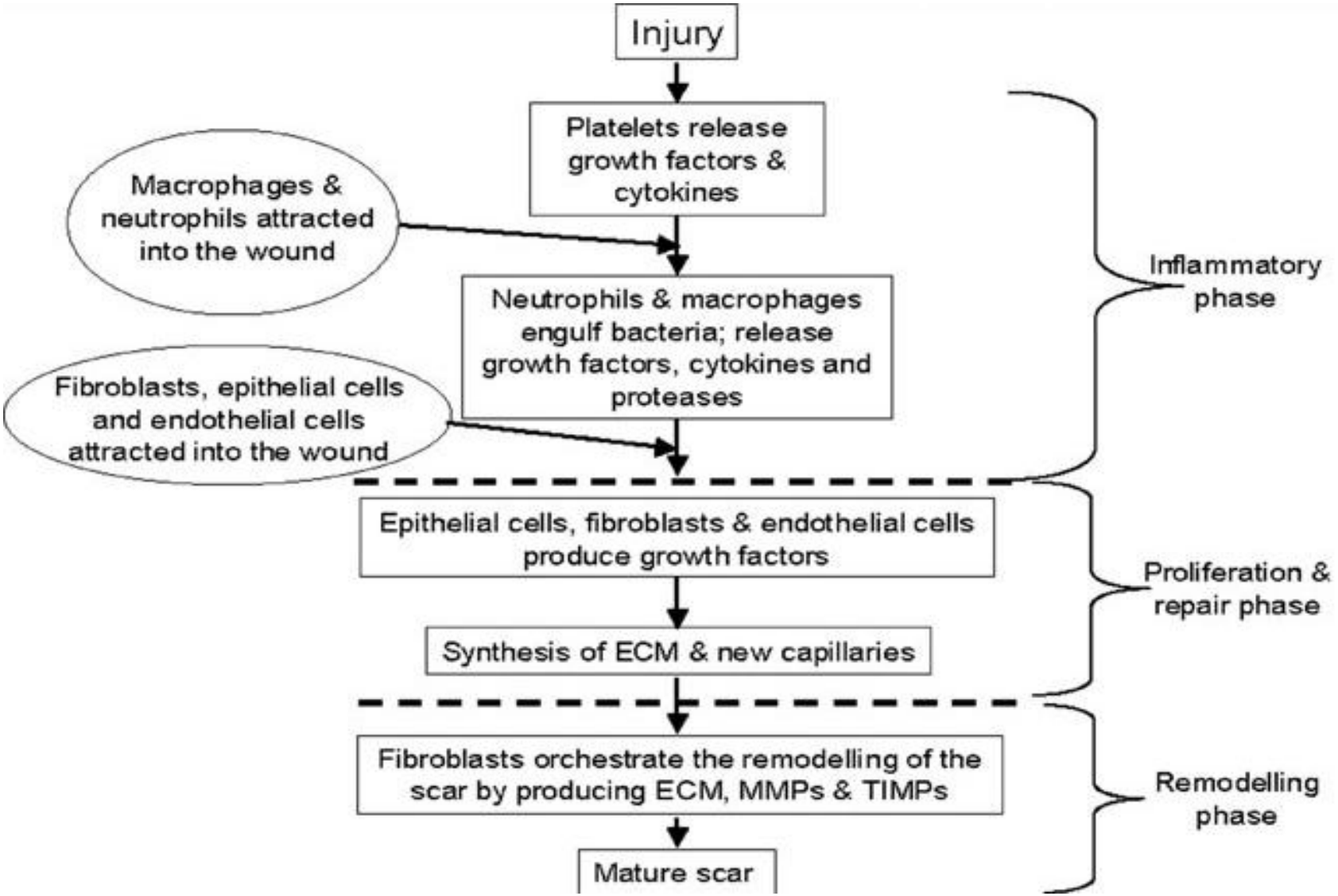
OK

Wound healing

- is a complex biologic process that involves chemotaxis and division of cells, neovascularization, synthesis of extracellular matrix, proteins, and remodeling of scar tissue.

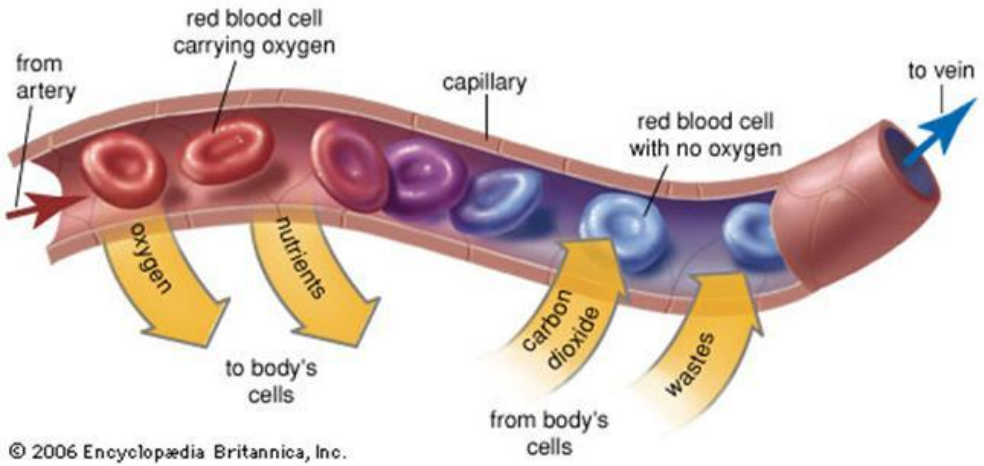
- The process begins when disruption of skin integrity occurs below the epidermis. The three phases of wound healing are the inflammatory, the proliferative, and the maturation phase



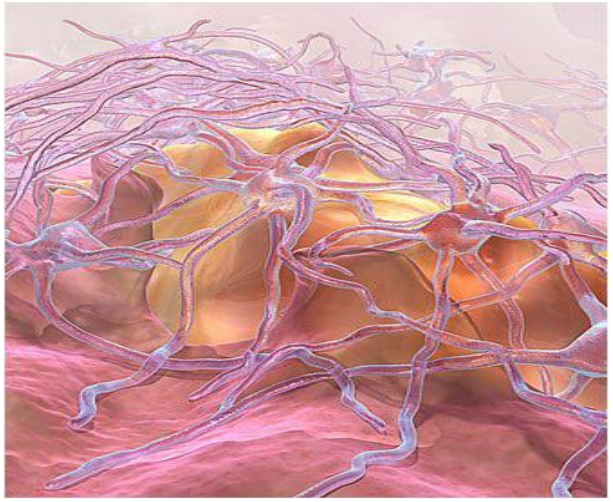

Injury
Platelets release growth factors & cytokines
Macrophages & neutrophils attracted into the wound
Neutrophils & macrophages engulf bacteria; release growth factors, cytokines and proteases
Fibroblasts, epithelial cells and endothelial cells attracted into the wound
Inflammatory phase
Epithelial cells, fibroblasts & endothelial cells produce growth factors
Synthesis of ECM & new capillaries
Proliferation & repair phase
Fibroblasts orchestrate the remodelling of the scar by producing ECM, MMPs & TIMPs
Mature scar
Remodelling phase

Injury and vascular response

- Haemostasis activates a series of overlapping events which provide a temporary barrier to bacterial infection
- Molecules of fibrin, a protein that acts as a molecular spring to keep blood clots structurally stable, but still flexible enough to allow blood to flow through them.

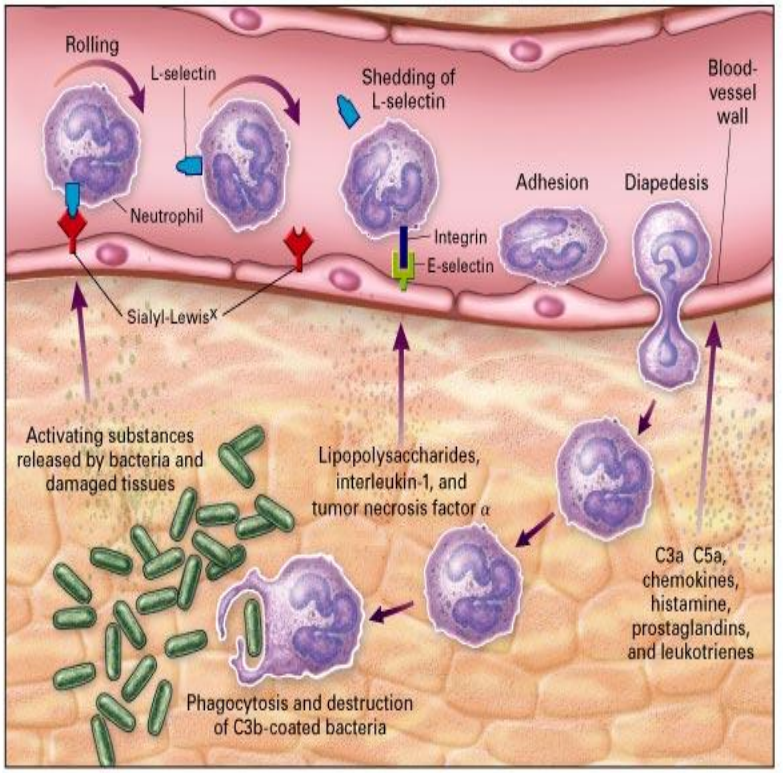


- Active platelets
  Activated platelets begin to seal over a blood vessel rupture by producing long cytoplasmic extensions.



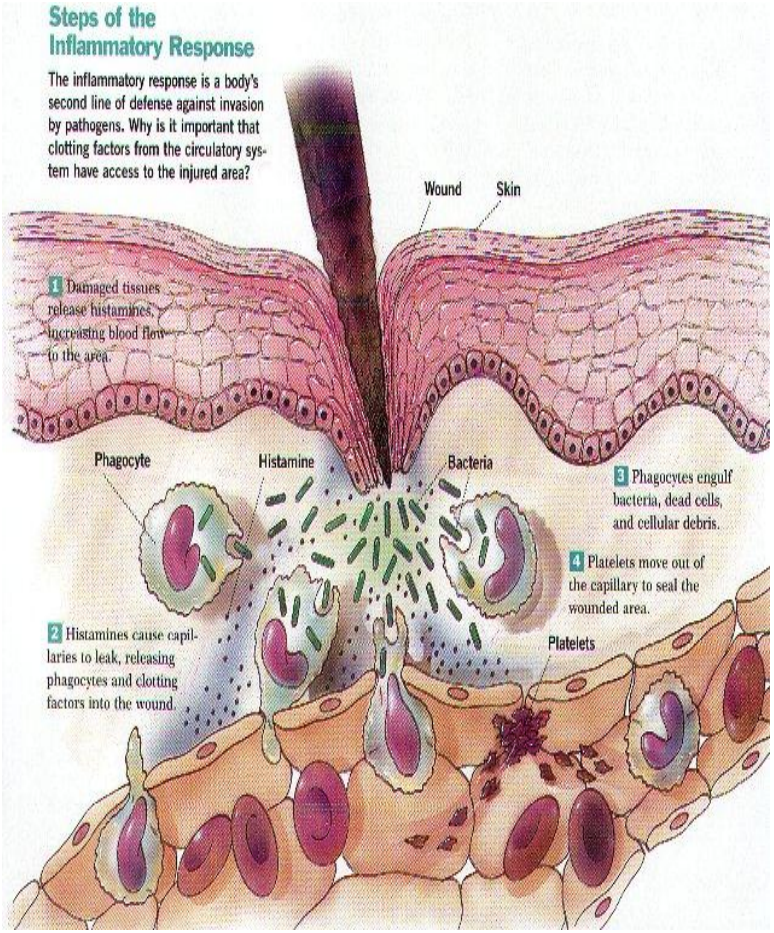
Polymorphonuclear neutrophils (PMN)

- Within an hour of wounding, polymorphonuclear neutrophils (PMNs) arrive at the wound site and become the predominant cells in the wound for the first three days after the injury occurs, with especially high numbers on the second day.
- They are attracted to the site by fibronectin, growth factors, and substances such as neuropeptides and kinins.



Inflammation phase

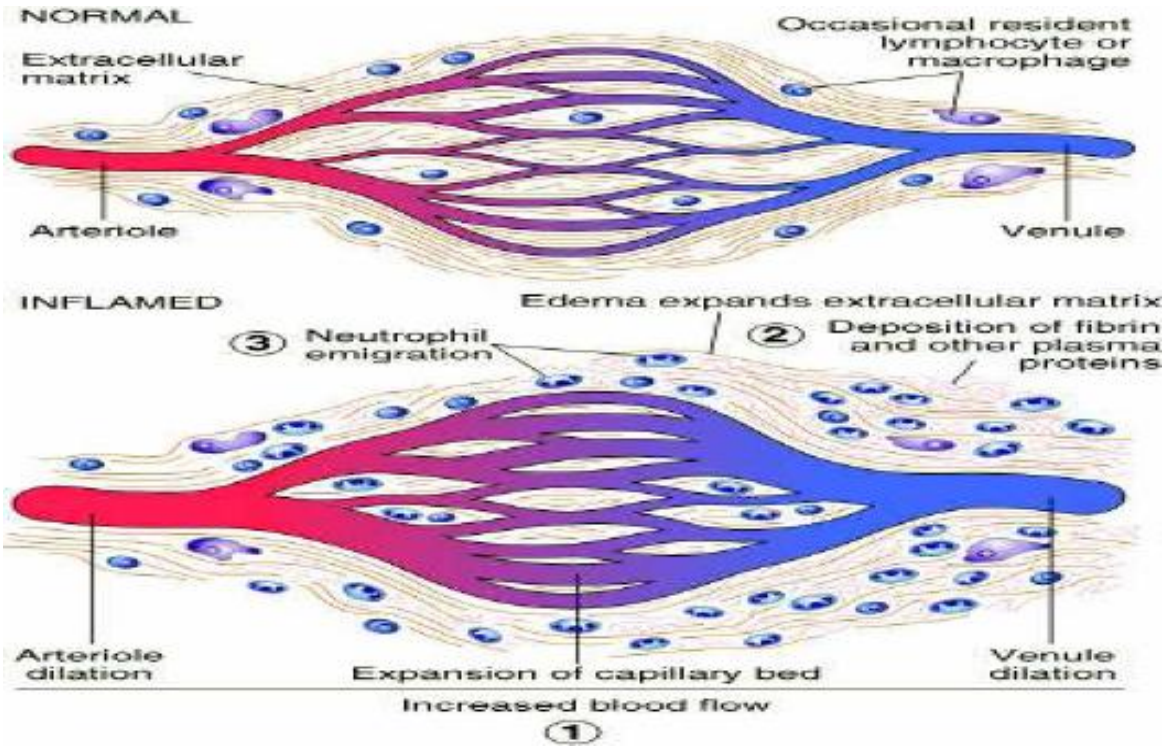
- Inflammation plays roles in fighting infection and inducing the proliferation phase, it is a necessary part of healing.
- The neutrophils and macrophages destroy bacteria and debris.
- The macrophages are also a source of cytokines, growth factors and proteolytic enzymes necessary for the wound to heal.
- As inflammation dies down, fewer inflammatory factors are secreted, existing ones are broken down, and numbers of neutrophils and macrophages are reduced at the wound site. These changes indicate that the inflammatory phase is ending and the proliferative phase is underway.

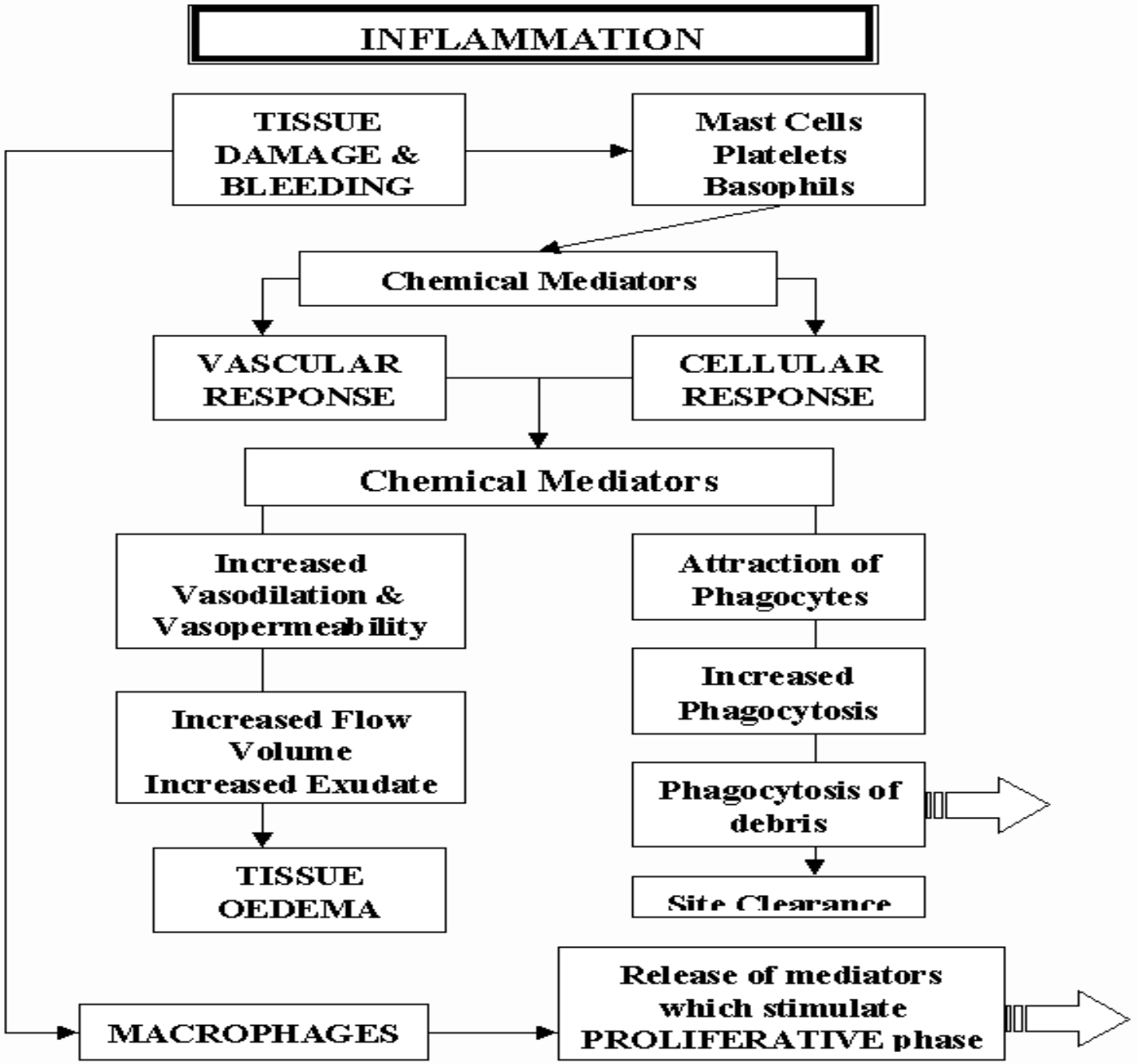


Inflammation

2 main steps to inflammation response.

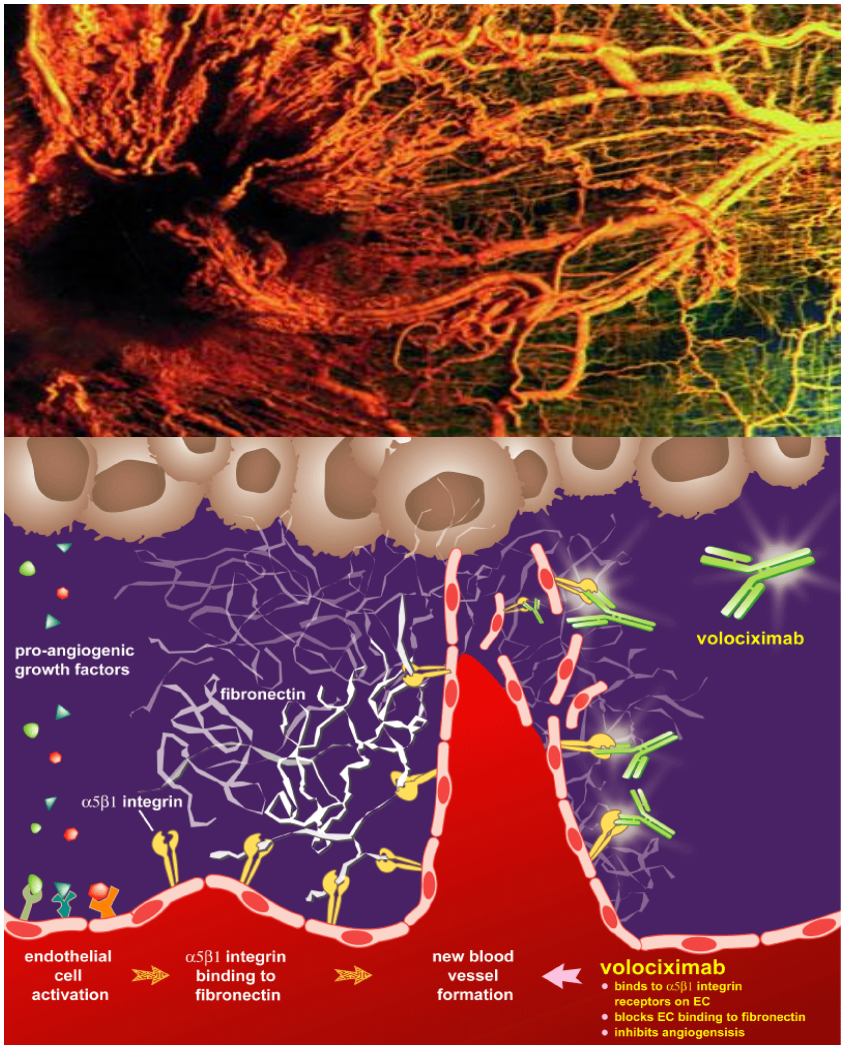
1. A vascular reaction
2. A cellular reaction




INFLAMMATION
TISSUE DAMAGE & BLEEDING
Mast Cells Platelets Basophils
Chemical Mediators
VASCULAR RESPONSE
CELLULAR RESPONSE
Chemical Mediators
Increased Vasodilation & Vasopermeability
Attraction of Phagocytes
Increased Flow Volume Increased Exudate
Increased Phagocytosis
Phagocytosis of debris
TISSUE OEDEMA
Site Clearance
MACROPHAGES
Release of mediators which stimulate PROLIFERATIVE phase

Angiogenesis: neovascularization

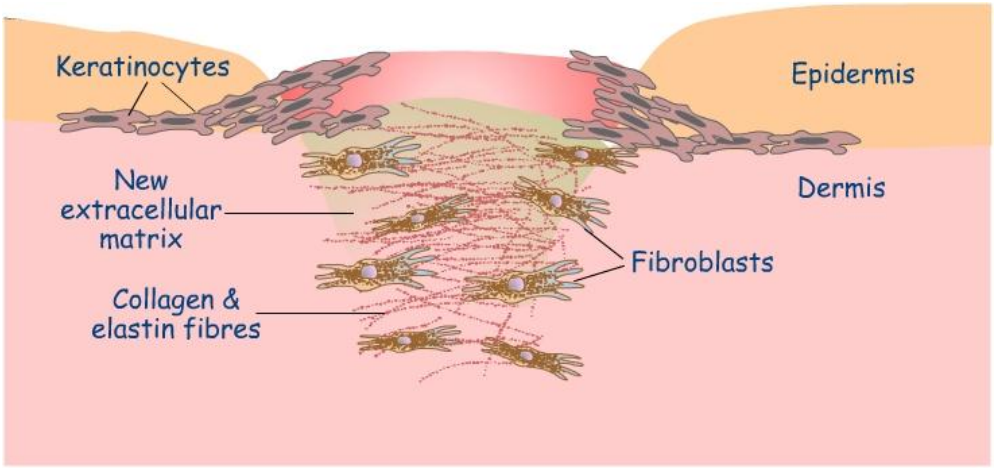
- the process of angiogenesis occurs concurrently with fibroblast proliferation when endothelial cells migrate to the area of the wound
- Because the activity of fibroblasts and epithelial cells requires oxygen, angiogenesis is imperative for other stages in wound healing, like epidermal and fibroblast migration.

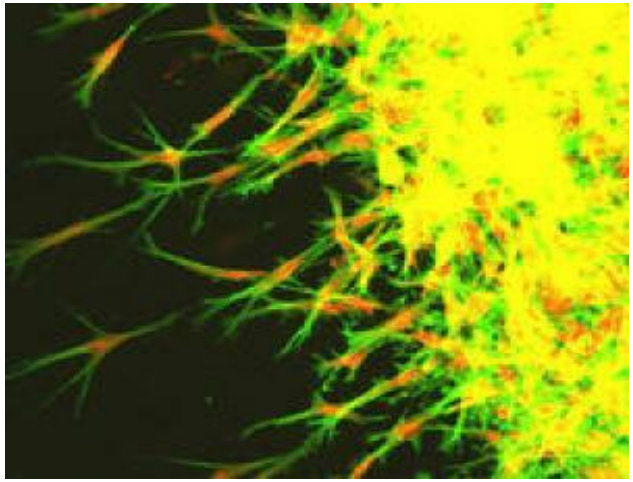


Proliferative phase

is characterized by the formation of granulation tissue.

- About two or three days after the wound occurs, fibroblasts begin to enter the wound site,
- As in the other phases of wound healing, steps in the proliferative phase do not occur in a series but rather partially overlap in time.





Fibroblast activity in wound healing

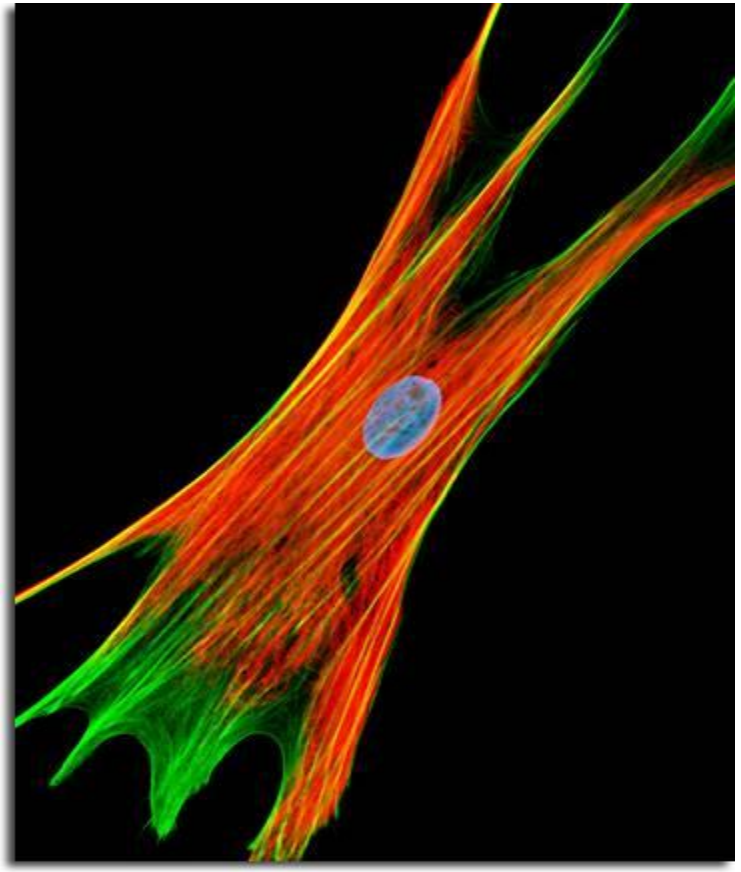
Fibroplasia and granulation tissue formation

- Simultaneously with angiogenesis, fibroblasts begin accumulating in the wound site. Fibroblasts begin entering the wound site two to five days after wounding as the inflammatory phase is ending, and their numbers peak at one to two weeks post-wounding.

- By the end of the first week, fibroblasts are the main cells in the wound

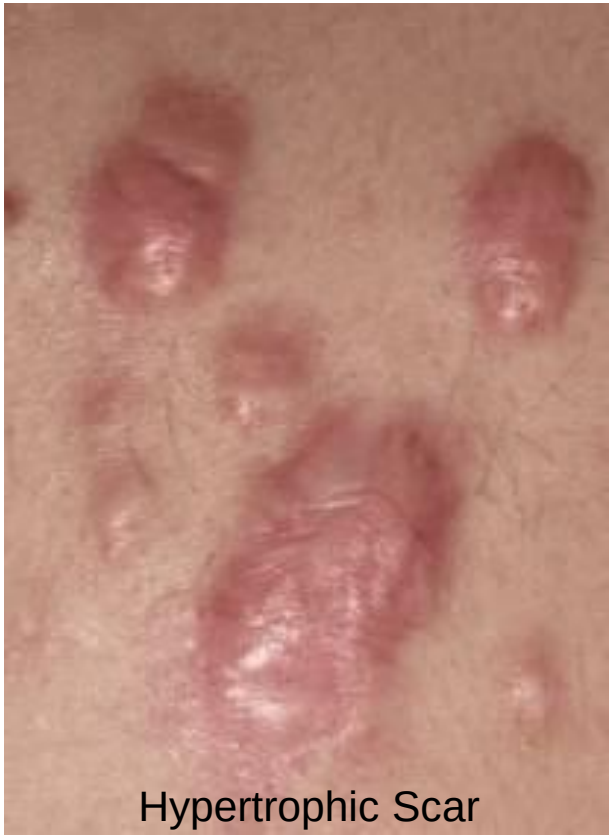
- Fibroplasia ends two to four weeks after wounding.

One of fibroblasts' most important duties is the production of collagen.



Collagen deposition

- Fibroblasts begin secreting collagen by the 2nd & 3rd post-wounding day, and peaks at one to three weeks.
- Even as fibroblasts are producing new collagen, collagenases and other factors degrade it. Shortly after wounding, synthesis exceeds degradation so collagen levels in the wound rise, but later production and degradation become equal so there is no net collagen gain. This homeostasis signals the onset of the maturation phase.

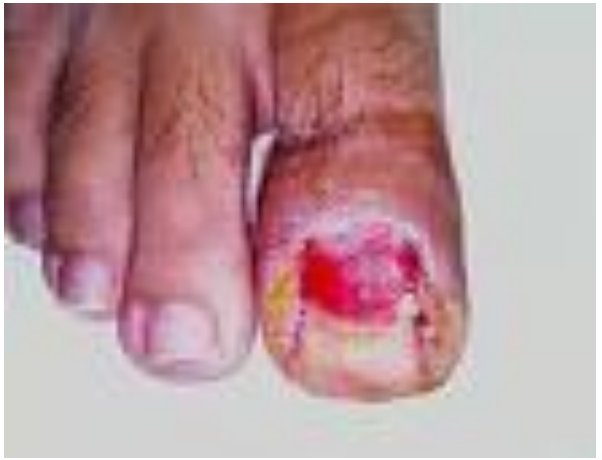


Hypertrophic Scar

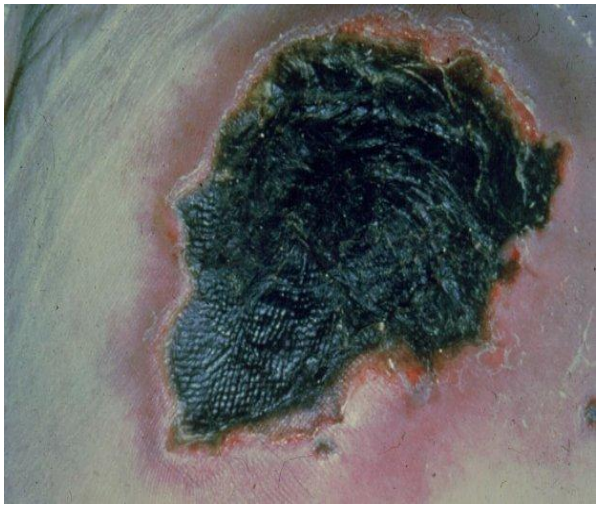
Granulation tissue

During the **maturation phase**, the wound closes, and scar tissue forms.

- Granulation gradually ceases and fibroblasts decrease in number in the wound once their work is done. At the end of the granulation phase, fibroblasts begin to commit apoptosis, converting granulation tissue from an environment rich in cells to one that consists mainly of collagen.



- Granulation tissue consists of new blood vessels, fibroblasts, inflammatory cells, endothelial cells, myofibroblasts, and the components of a new, provisional ECM.
- Fibroblasts deposit ECM molecules like glycoproteins,glycosaminoglycans (GAGs), proteoglycans, elastin, and fibronectin, which they can then use to migrate across the wound



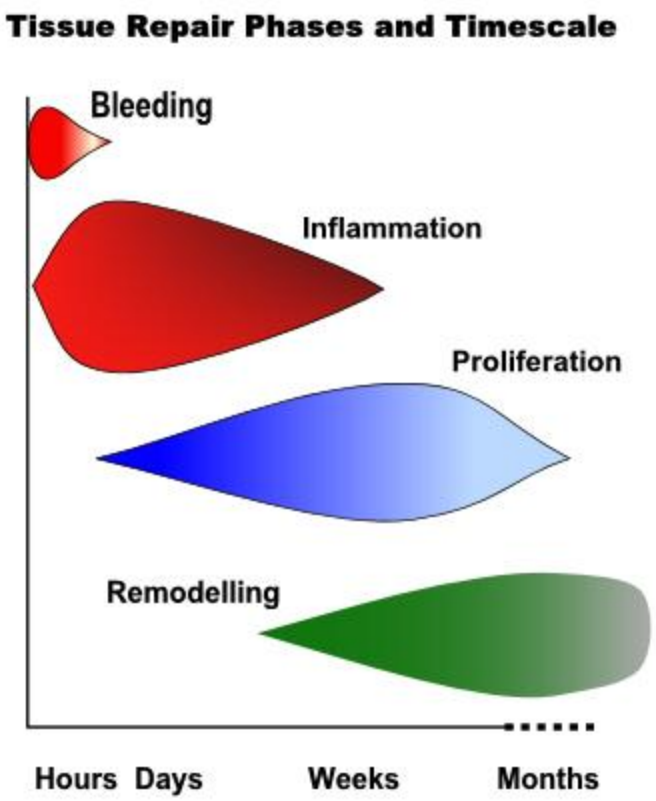


Figure 1 is a gross representation of the key phases of the tissue repair process. The phases identified are shown as separate entities, though in reality, they are interlinked in a very deliberate way (Fig 2). There are events associated with one phase that act as stimulants for the following phase. Some examples of these have been included in the main text.

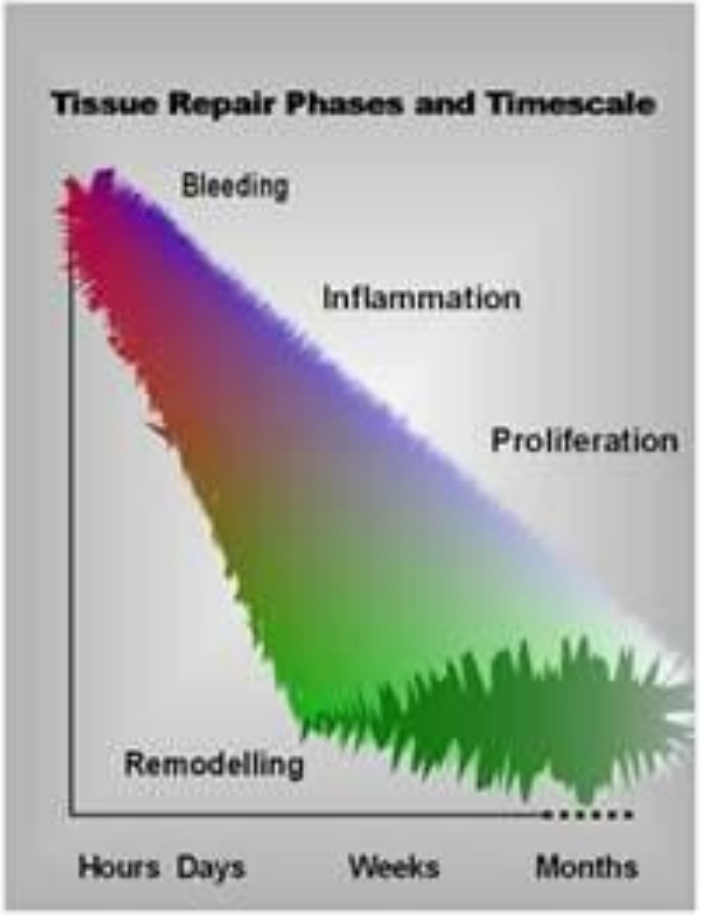


Figure 2 indicates the integrated reality of repair rather than the 'convenient' separate phase model.

Non viable Tissue

Non Viable Definition

‘Not alive

or

incapable of living,

developing, or reproducing,

as in a nonviable cell’.

http://www.biology-online.org/dictionary/Nonviable

NVT: Necrosis

‘**T**he morphological changes indicative of cell death caused by progressive enzymatic degradation;

it may affect groups of cells or part of a structure or an organ.

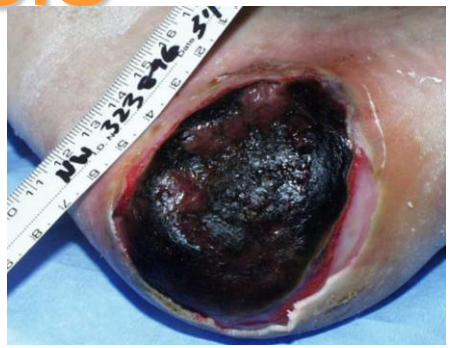
Cell or tissue death due to disease, trauma, hypoxia, radiation, acute infection, etc;

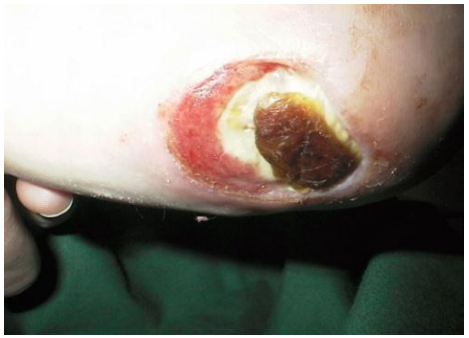
the constellation of changes that accompany and follow irreversible cell injury in living organisms.

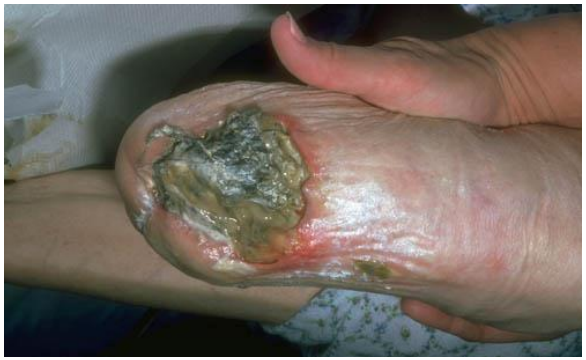
http://medical-dictionary.thefreedictionary.com/necrosis

Types NVT: Necrosis

- Eschar
  - Found in exposed [outer] areas
  - Often dry
    - Hard, firm layer of tissue damage
    - Classic color is black
      - Tan / brown / grey
- True extent of underlying damage may not been known unless the eschar is removed



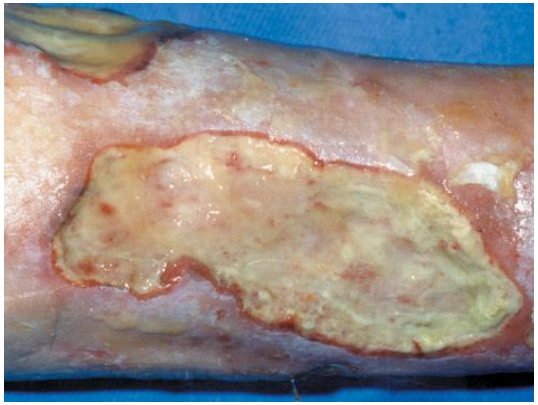


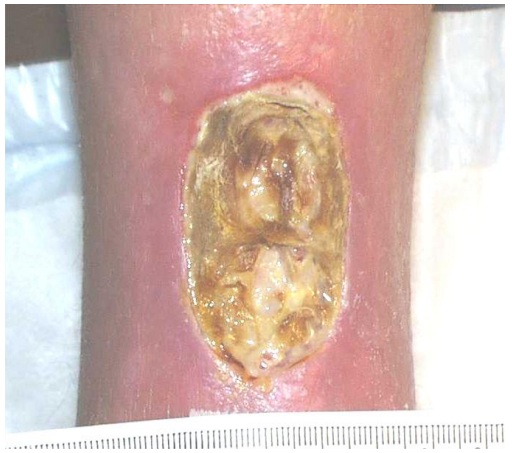


NICE Guidelines 2005 . Management of pressure ulcers in primary and secondary care. http://www.nice.org.uk/CG029

Types NVT: Necrosis

- Slough
  - Contains proteinaceous tissue, fibrin & neutrophils
  - Varying levels of moisture
    - Cream, yellow to tan
  - Layer separation between viable and NVT





NICE Guidelines 2005 . Management of pressure ulcers in primary and secondary care. http://www.nice.org.uk/CG029

NVT Characteristics: CMCA

- **Color**
  - Cream, yellow / tan, brown / grey, black
    - ‘Green tinge’ in the presence of ***Pseudomonas aeruginosa***
- **Moisture**
  - Dry & dehydrated
  - Moist / wet
- **Consistency**
  - Thin, mucinous, gelatinous, ‘stringy’ / thick ‘clumpy’
  - Soft, ‘soggy’ / dry, hard, ‘leathery’
  - Fibrinous / tough
- **Adherence**
  - Non adherent, loosely adhered, adherent, firmly adhered

Impact / Consequences

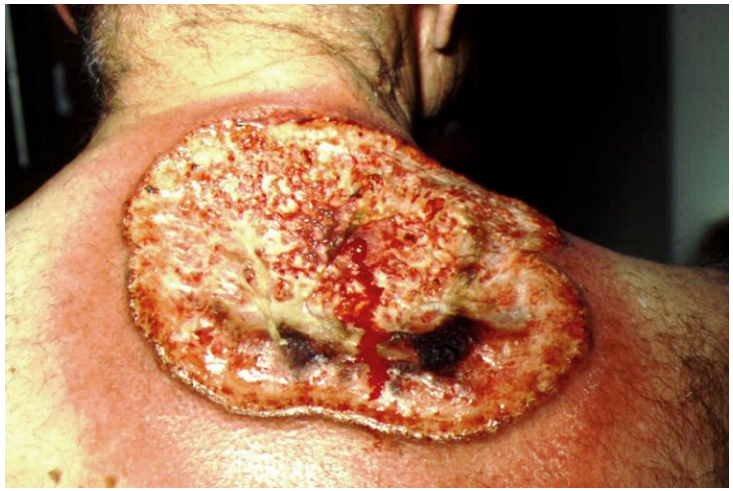
- Focal point bacteria
  - *NVT formation*
- Prolongs inflammation
  - Delay / inhibit healing
    - *NVT formation*
  - Increase exudate
    - Destruction ECM
    - *NVT formation*

- Mechanical barrier contraction / re-epithelisation

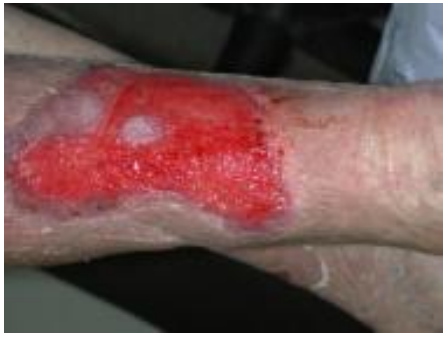
Enoch, S. Harding, K. Wound bed preparation: The science behind the removal of barriers to healing [Part 2]. 2003. Wounds15[7]:213-229

Important structures

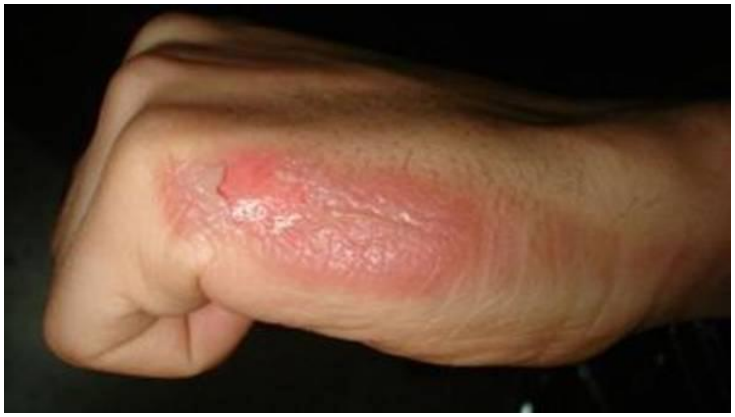
- Dead tissue
  - Avascular …. will not bleed
  - Insensate …. will not hurt
  - Smell …. often bad
  - Dead fat & fascia is brown-grey-black
  - Dead muscle is red-brown-grey

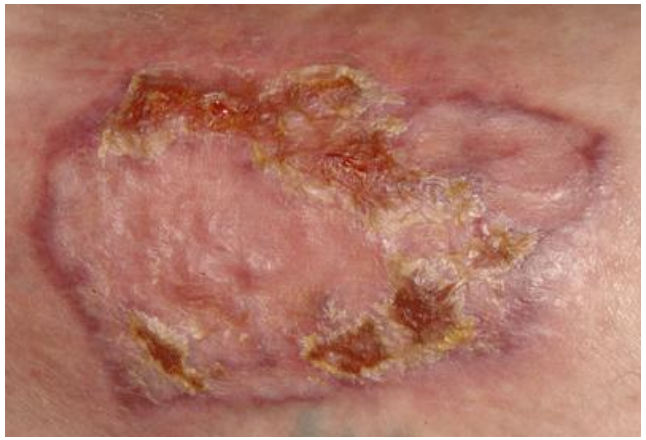


Important structures



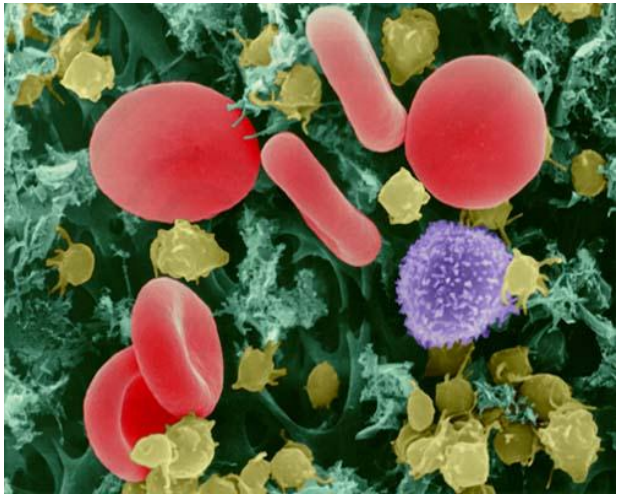
- Living Tissue
  - Vascular …. will bleed
  - Sensate …. will hurt when manipulated
  - Fascia is glistening white
  - Fat looks yellow (just darker that chicken fat)
  - Muscle is dull red and may contract if pinched





Exudate, what is it?

- Exudate is fluid that filters from the circulatory system into areas of the body that are inflamed.
- Inflammation leads to an increase in blood vessel dilatation and permeability with extracellular fluid formation, resulting in increased production of exudate.
- Wound exudate is protein-rich and cell-rich wound fluid that seeps out of blood vessels as a result of inflammation and is deposited in nearby tissues.
- The altered permeability of blood vessels permits the passage of large molecules through capillary walls.



Wound Assessment:

Characteristics of exudate

Types of Wound Exudate

Serous: clear, amber, thin and watery

Fibrinous: cloudy and thin, with strands of fibrin

Serosanguineous: clear, pink, thin and watery

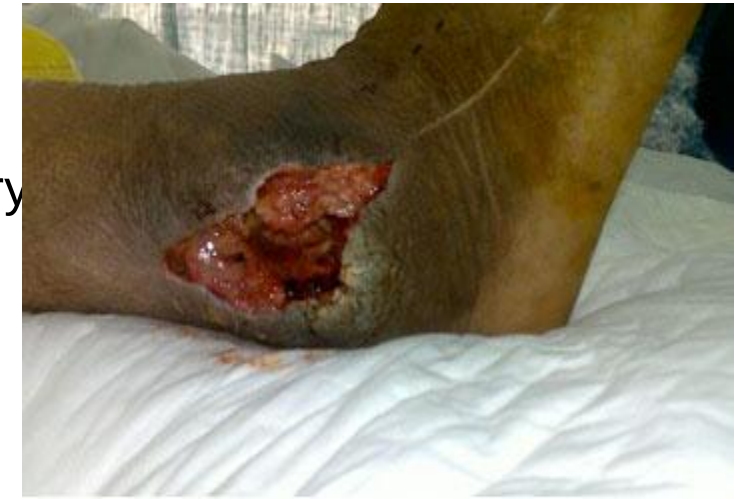
Sanguineous: reddish, thin and watery

Seropurulent: yellow or tan, cloudy and thick

Purulent: opaque, milky; sometimes green

Hemopurulent: reddish, milky and viscous

Hemorrhagic: red, thick



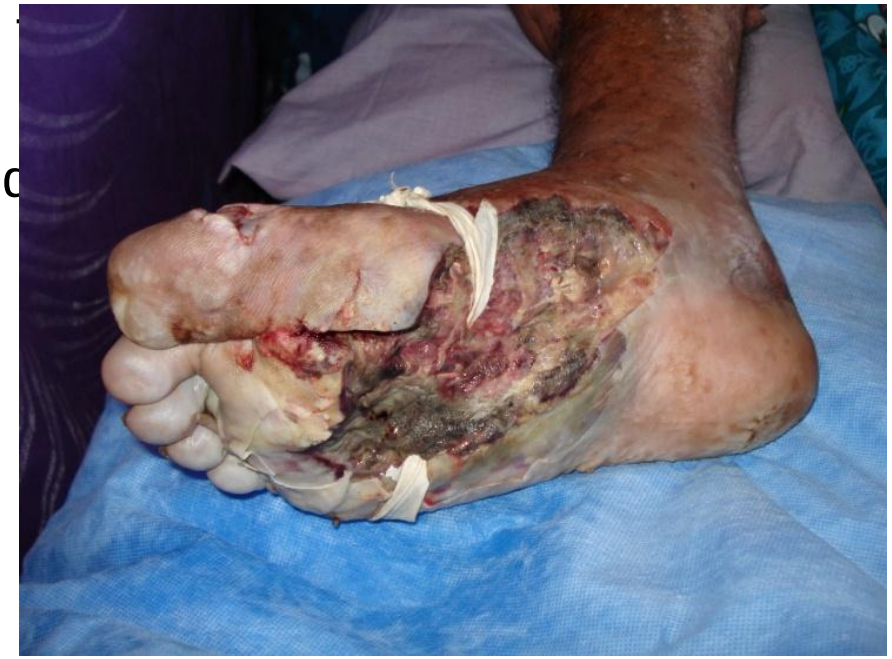
Wound Assessment:

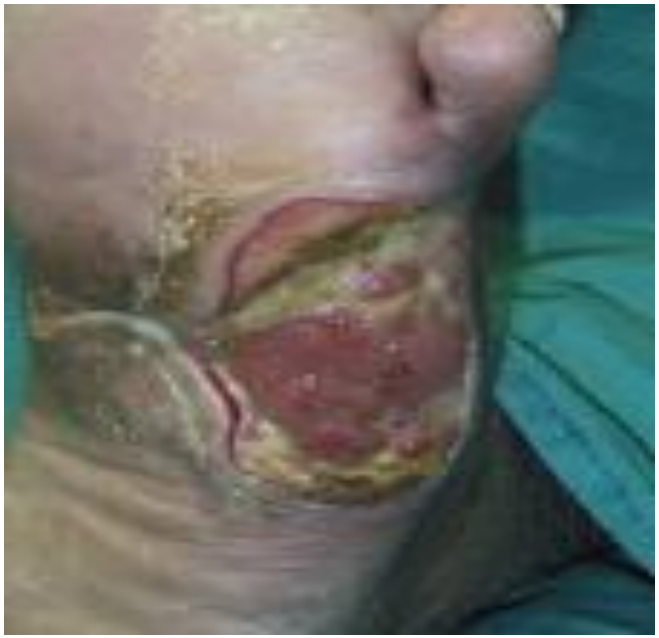
Characteristics of Exudate

Odour:

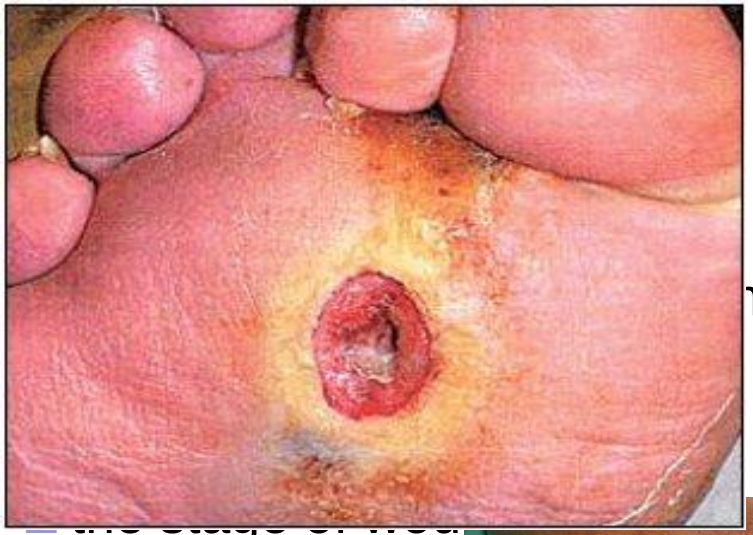
Wound odour can indicate

- Contamination with body fluids or faeces
- Infection
-
-

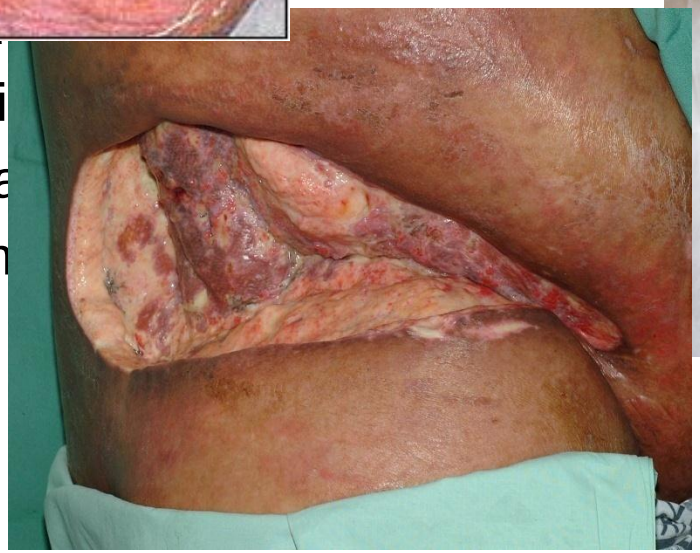


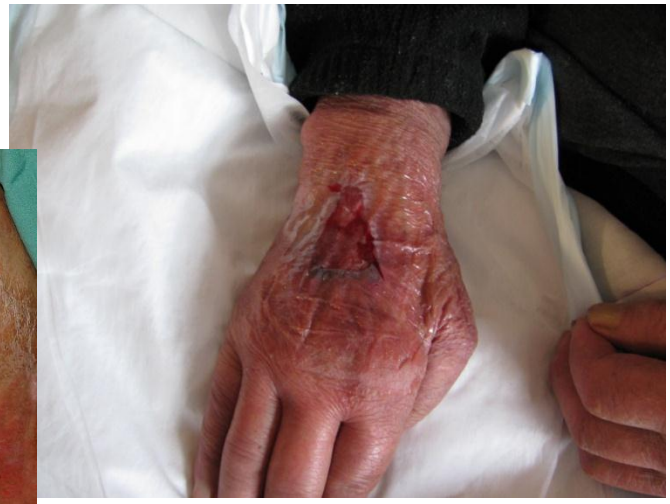


The Quantity of Exudate may be determined in part by wound type.



- with minimal exudate, and venous
- ne the quantity of exudate are as
  - the presence of i
  - underlying cardia
  - peripheral oeden





Wound Assessment: Characteristics of Exudate

Amount:
Exudate is difficult to measure accurately, often described as light, moderate or heavy.

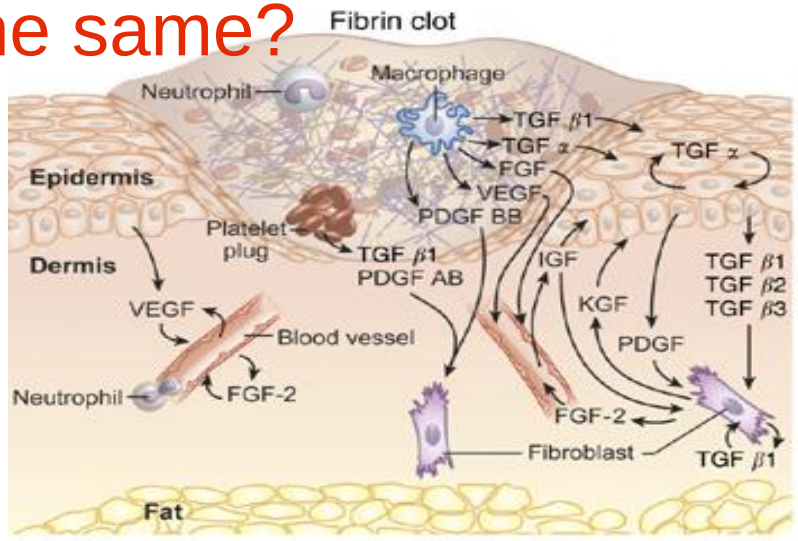
Heavy Exudate may result in Electrolyte imbalance. It may be useful to use a wound drainage bag if possible.

Status	Indicators
Dry	Wound bed is dry: there is no visible moisture • this may be the environment of choice for ischaemic wounds
Moist	Small amounts of fluid are visible when the dressing is removed. Dressing changes are appropriate for dressing type
Wet	Small amounts of fluid are visible when dressing is removed; the primary dressing is extensively marked
Saturated	Primary dressing is wet and strike through is occurring; dressing change is required more frequently or the dressing type needs to change
Leaking	Dressings are saturated and exudate is escaping from primary/secondary dressings ; dressing change is required more frequently or the dressing type needs to change

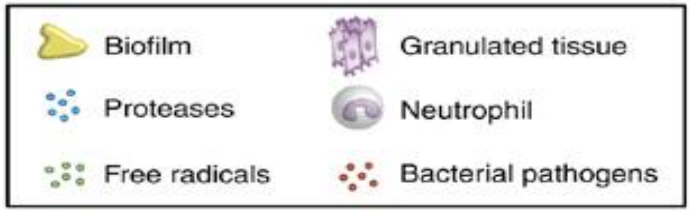
Acute & Chronic wounds

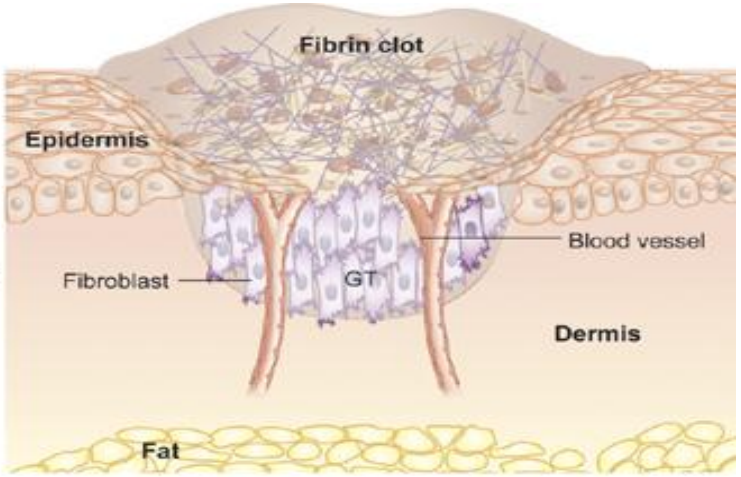
Is there a difference?

Do we treat these wounds the same?

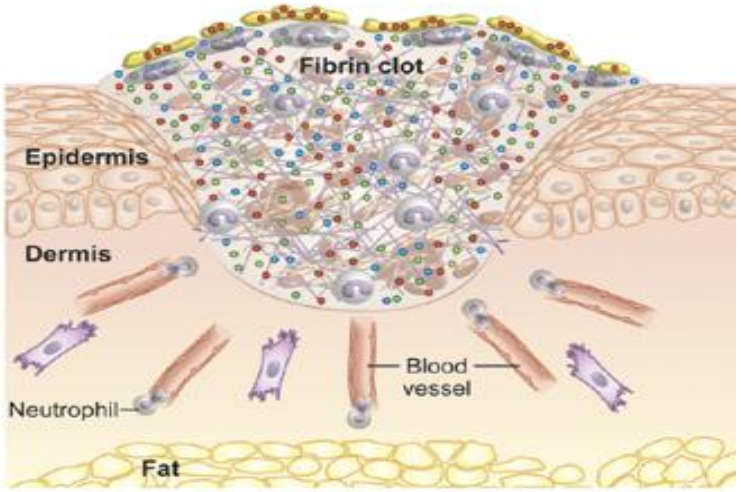


Initiation of wound repair





Repair of acute wound



Failure to heal: chronic wound

In Acute Wounds

- Wound exudate contains endogenous proteinases that promote wound bed preparation, removing any slough through autolysis and keeping the wound moist to promote epithelialization.

- Acute wound exudate is rich in platelet-derived growth factors, fibroblast growth factors and epithelial growth factors.

In Chronic Wounds

- Exudate may work against the healing process. It has been called a "wounding agent in its own right" because it can break down growth factors, macerate periwound skin and prolong the inflammatory phase.
  - Chronic wound exudate has higher concentrations of matrix metalloproteinases, which are proteolytic enzymes that break down the wound's cellular matrix.
  - It also has increased levels of inflammatory mediators such as histamine and bradykinin, which prolong the inflammatory phase of wound healing.

Inflammation is divided into:

- Acute inflammation.
- Chronic inflammation.

Acute inflammation

- rapid in onset (seconds or minutes)
- of relatively short duration, lasting for minutes, several hours, or a few days
- its main characteristics are the exudation of fluid and plasma proteins (oedema) and the emigration of Leukocytes, predominantly neutrophils.

Chronic inflammation

- is of longer duration
- associated histologically with the presence of lymphocytes and macrophages, the proliferation of blood vessels, fibrosis, and tissue necrosis.
- Less uniform.

Physical causes leading to persistence of chronic wounds

NEUROPATHY	Diabetes mellitus, Spinal injuries, Cerebral Palsy, Hansen's disease (Leprosy)
ISCHEMIA	Atherosclerosis, calcification, microangiopathy (diabetes mellitus), any form of peripheral vascular disease
PERIPHERAL OEDEMA	Venous hypertension (deep venous thrombosis, varicose veins), systemic causes (renal or cardiac failure), lymphoedema, decreased albumin, elephantiasis (common in tropical countries)
PRESSURE	Poor mobility, spinal cord injuries, dementia, diabetes mellitus, extremes of age, terminal illness
OTHER CAUSES	Connective tissue disorders leading to vasculitis, arterio-venous malformations, drugs such as corticosteroids and hydroxyurea, systemic diseases, malignancy, osteomyelitis, smoking, inherited neutrophil disorders, poor nutritional status

Cellular features of chronic wounds

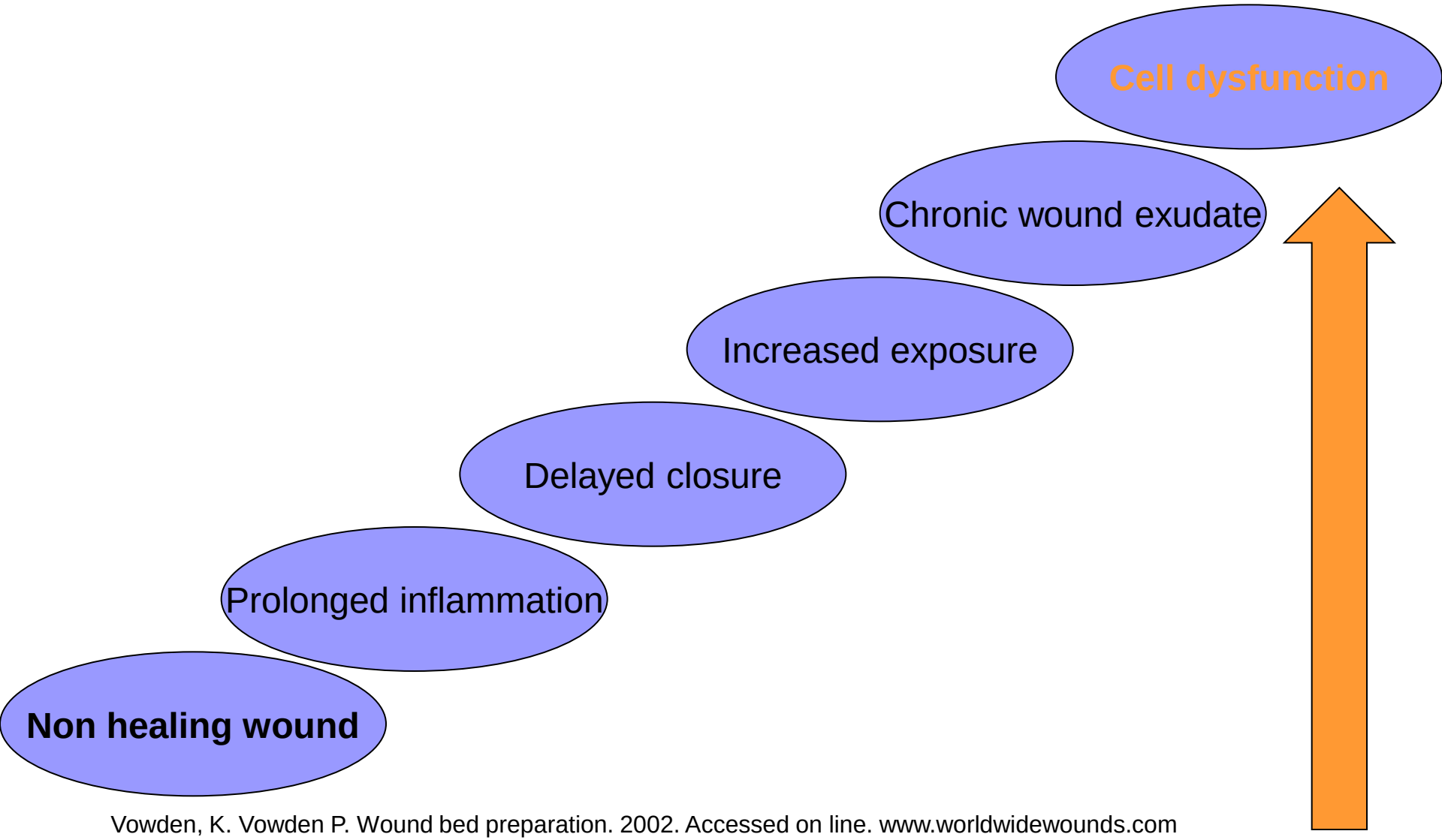
- Generally low mitotic activity
- Impaired migration of keratinocytes
- Altered fibroblast cellular phenotype
- Decreased fibroblast proliferation and migration
- Decreased response of fibroblasts to growth factors
- Presence of increased proportion of senescent cells

Clinical features and complications of chronic wounds

- Presence of necrotic and unhealthy tissue
- Excess exudate and slough
- Lack of adequate blood supply
- Absence of healthy granulation tissue
- Failure of re-epithelialisation
- Cyclical or persistent pain
- Recurrent wound breakdown
- Clinical or sub-clinical infection

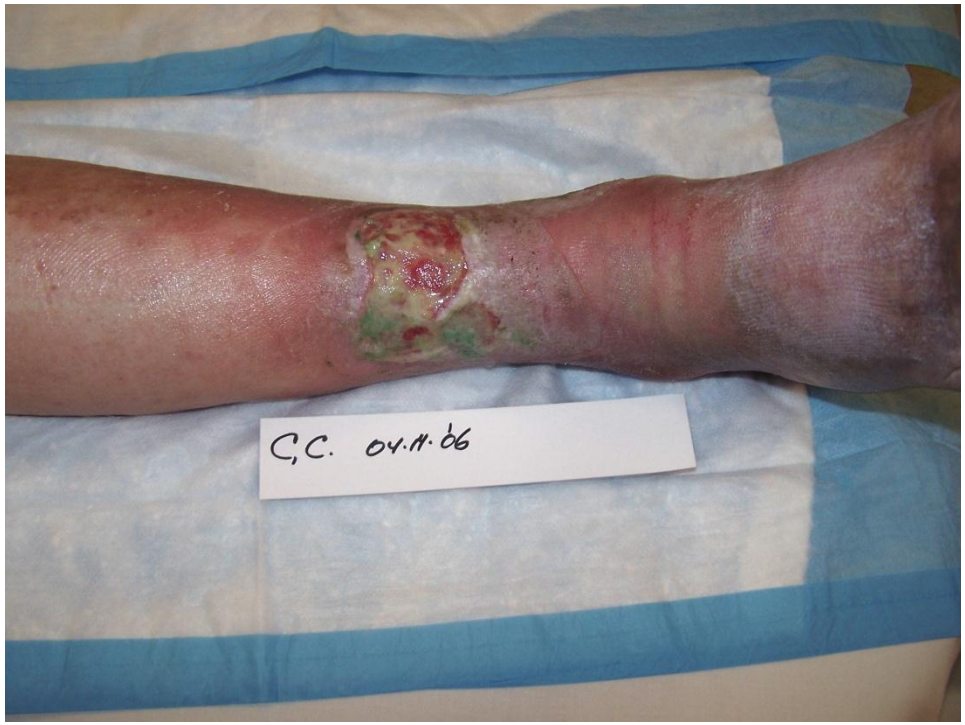
- Sinus formation
- Fistulous communication
- Malignancy in the fistulous tract
- Malignant transformation in the
- ulcer bed (Marjolin's ulcer)
- Osteomyelitis
- Contractures and deformity in
- surrounding joints
- Systemic amyloidosis
- Heterotopic calcification
- Colonisation by multiple drug-resistant
- pathogens leading to antibiotic resistance

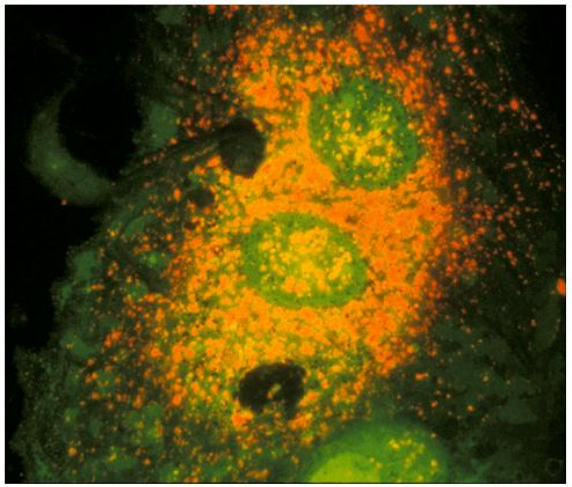
Senescent Cells



Vowden, K. Vowden P. Wound bed preparation. 2002. Accessed on line. www.worldwidewounds.com

Senescent Cells



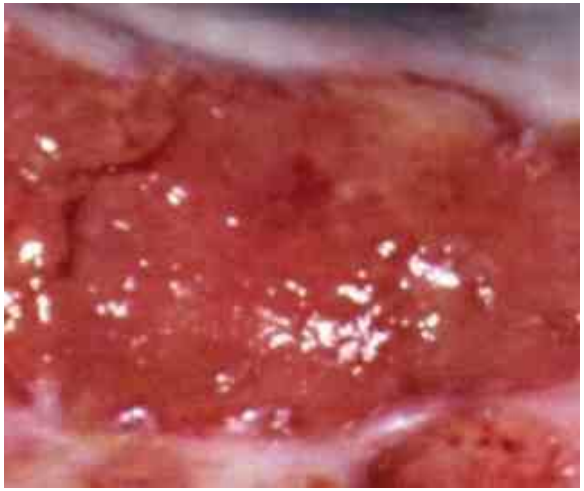


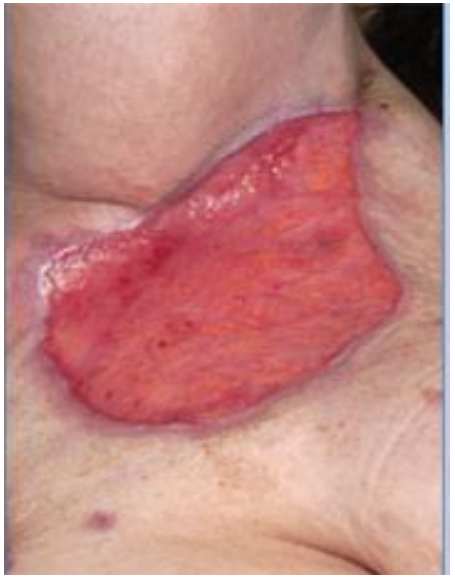
Cells non responsive to GF
Incapable of mitotic activity

Enoch

‘All that glitters is not gold’

‘All that is red is not healthy’





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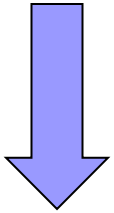
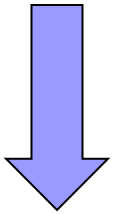
‘Chronically Inflamed’

- Non viable tissue – inflammation / infection



- Inflammation – increasing exudate



- Exudate inactivates / damages wound environment – creates more non viable tissue

Falanga, V. Wound bed preparation and the role of enzymes. Wounds. 2002.14[2]:47-57

Barriers in Wound Environment

- A wound which fails to progress:
  - **non viable tissue**
  - **bioburden**
  - **excessive moisture**
  - **pain** (Enoch & Price, 2004)

Sound Familiar??

Identifying the aim of treatment

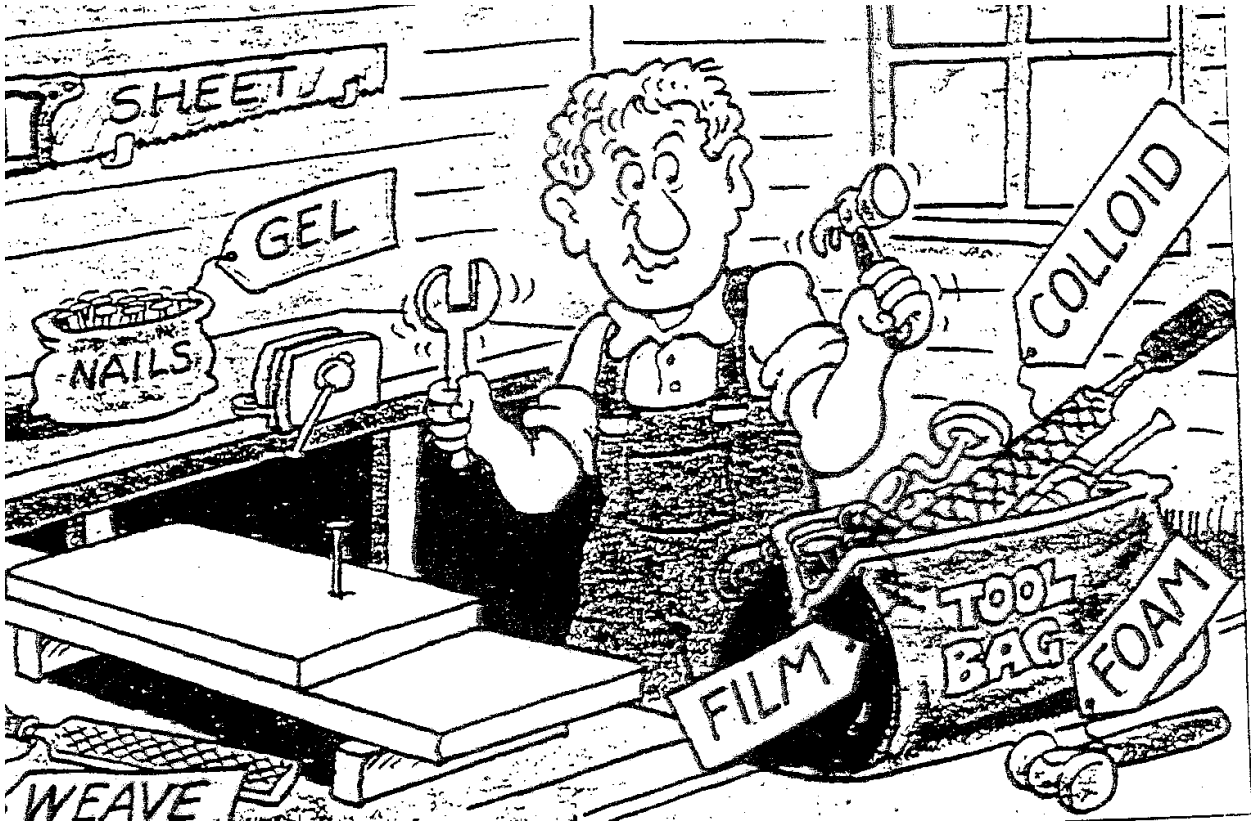
- Before the dressing selection process can begin, it is first necessary to identify the purpose or principal aim of the proposed treatment.
- The principal reasons for applying a dressing can be summarised as follows.
  - To produce rapid and cosmetically acceptable healing,
  - To remove or contain odour,
  - To reduce pain,
  - To prevent or combat infection,
  - To contain exudate,
  - To cause minimum distress or disturbance to the patient,
  - To hide or cover a wound for cosmetic reasons.
  - A combination of two or more of the above.

Wound Management Traditional Theory

1. Wounds should be kept clean and dry so that a scab forms over the wound
2. Wounds should be exposed to air and sunlight as much as possible
3. Where tissue loss is present the wound should be packed to prevent surface closure before the cavity is filled
4. Wounds should be covered with a dry dressing

Traditional Theory disadvantages

1. The scab (dehydrated exudate and dying dermis) is a physical barriers to healing the delay because epidermal cells cannot move easily under the scab there may be poor cosmetic results and scarring
2. Exposure to air reduces surface temperatures of the wound and dries the wound causing further delays healing
3. Gauze packing impairs the quality of healing
4. The dressing may adhere to the wound and cause trauma when is removed.


SHEET
GEL
NAILS
COLLOID
TOOL BAG
FOAM
FILM
WEAVE

1.Differential diagnosis?

2. Wound Bed assessment using the TIME(s) principles:

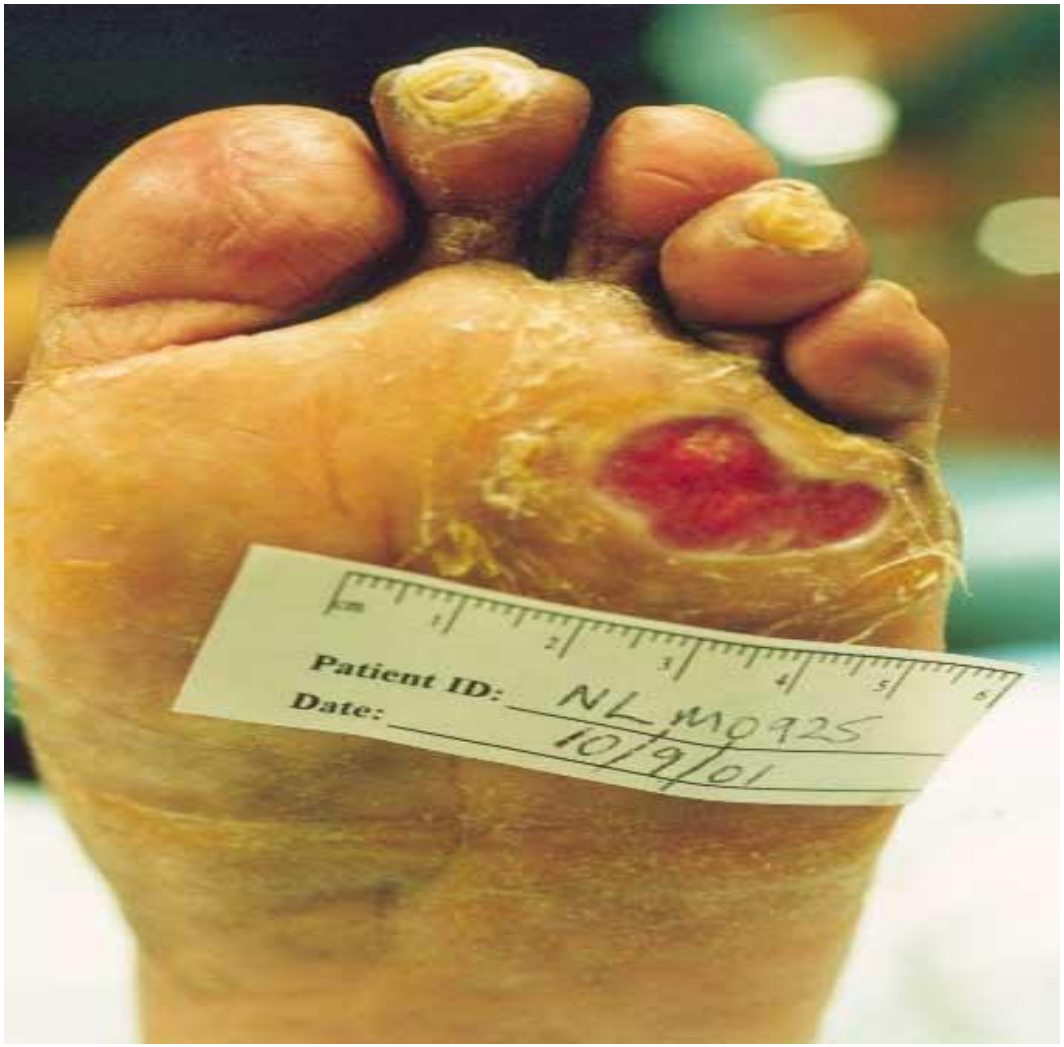
T" tissue

I: inflammation and/or Infection

M: moisture balance

E: epidermal margins

S: Surrounding skin.



3. Wound Management plan.

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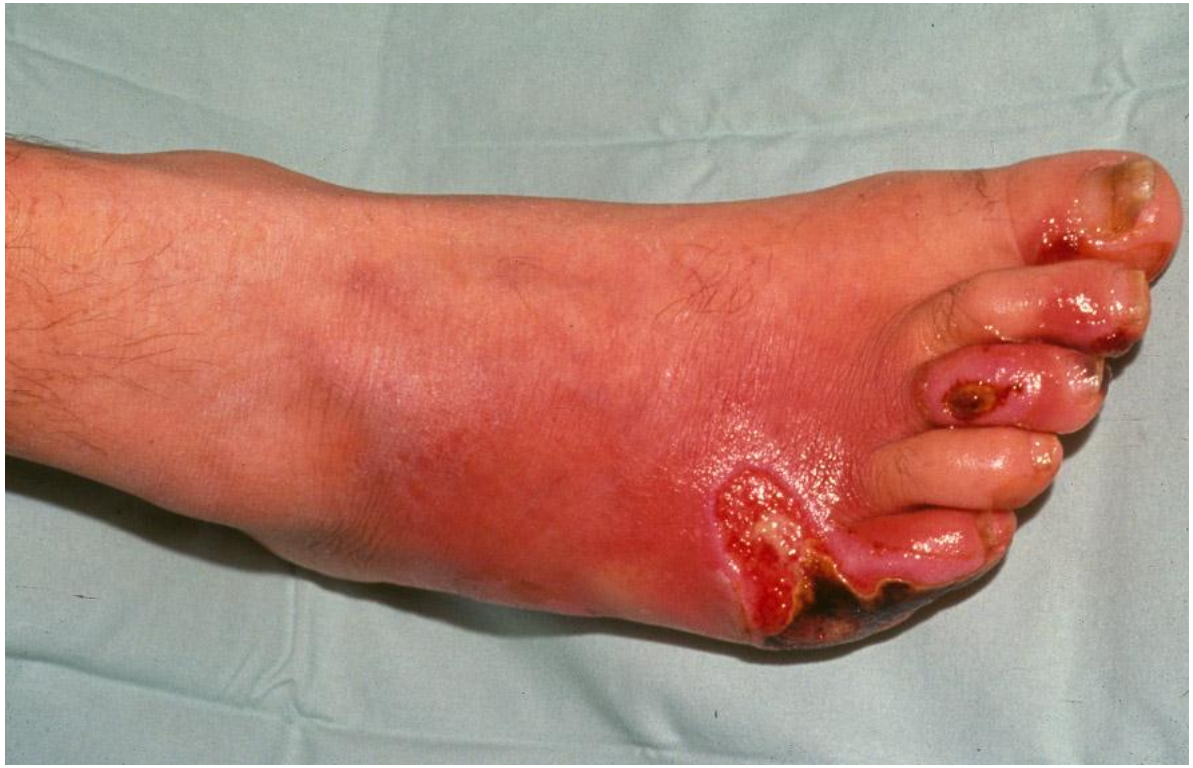

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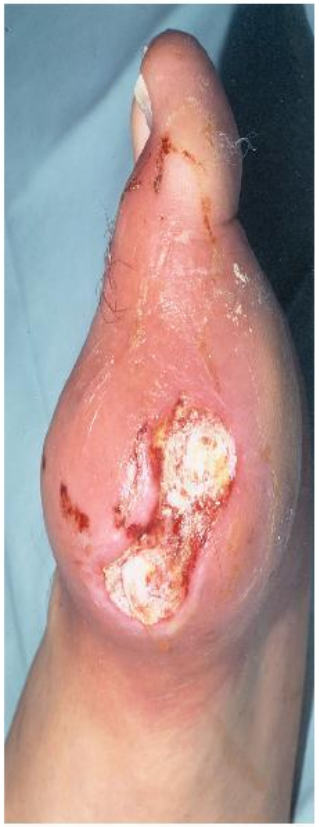

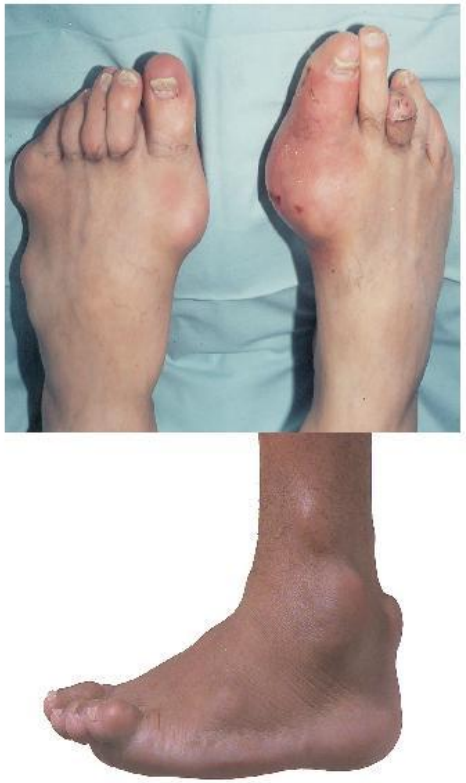

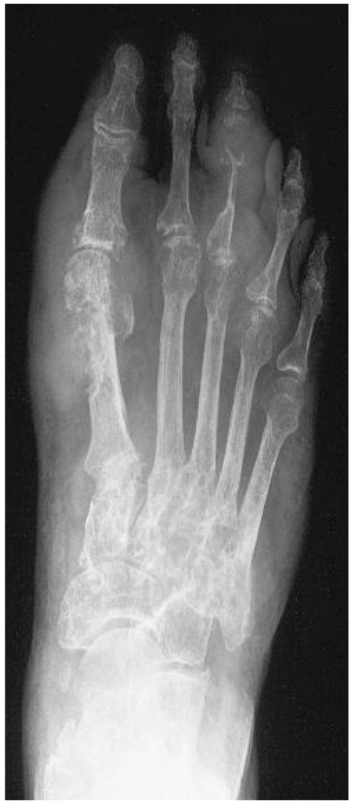

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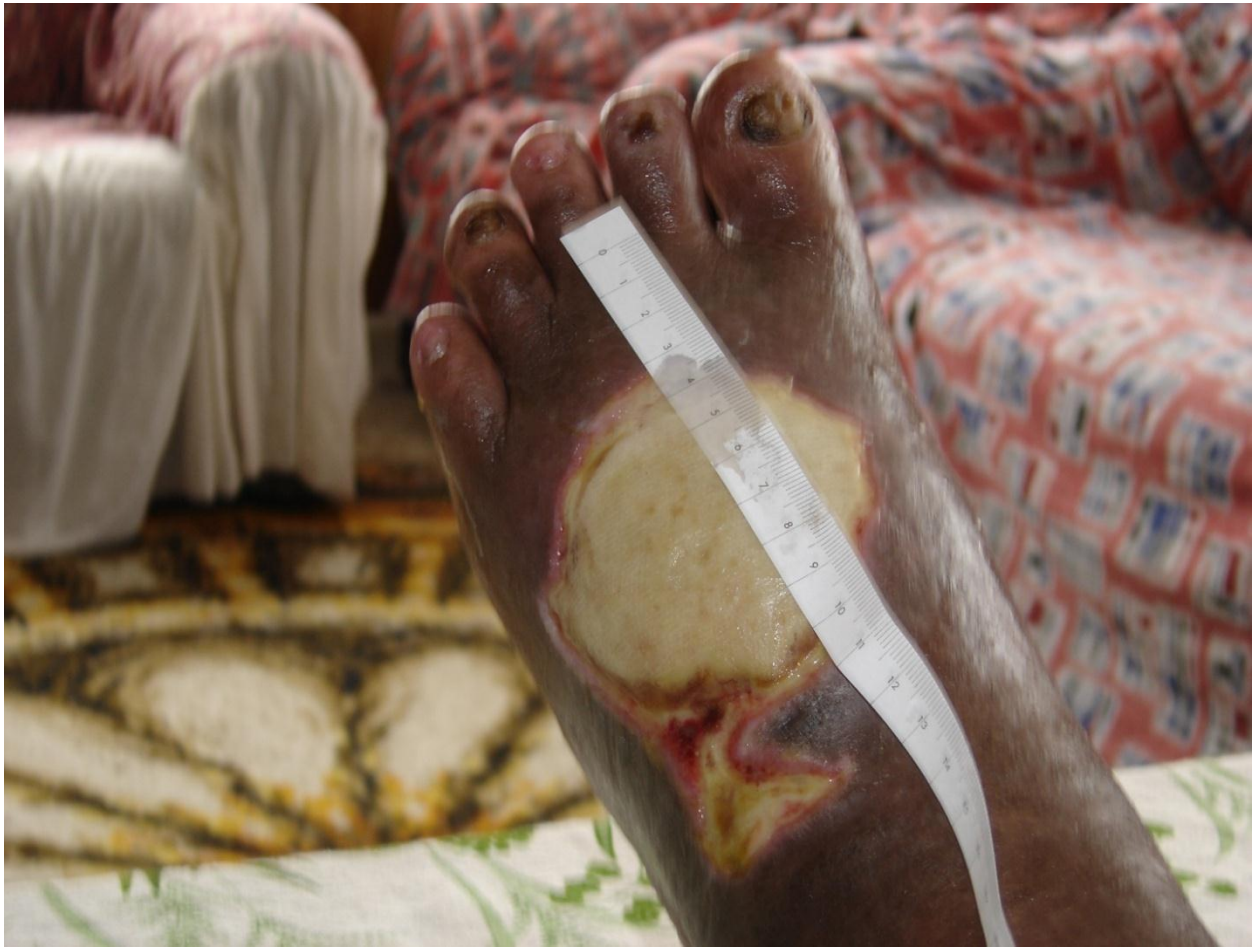

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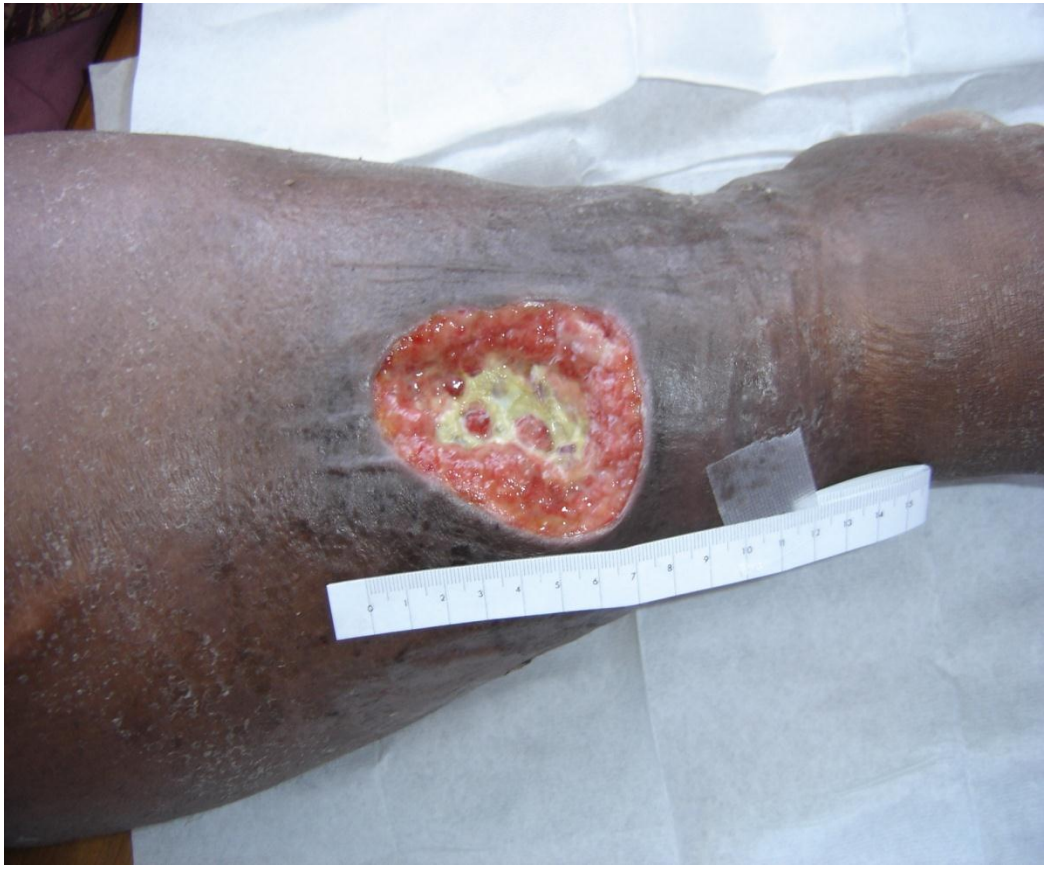

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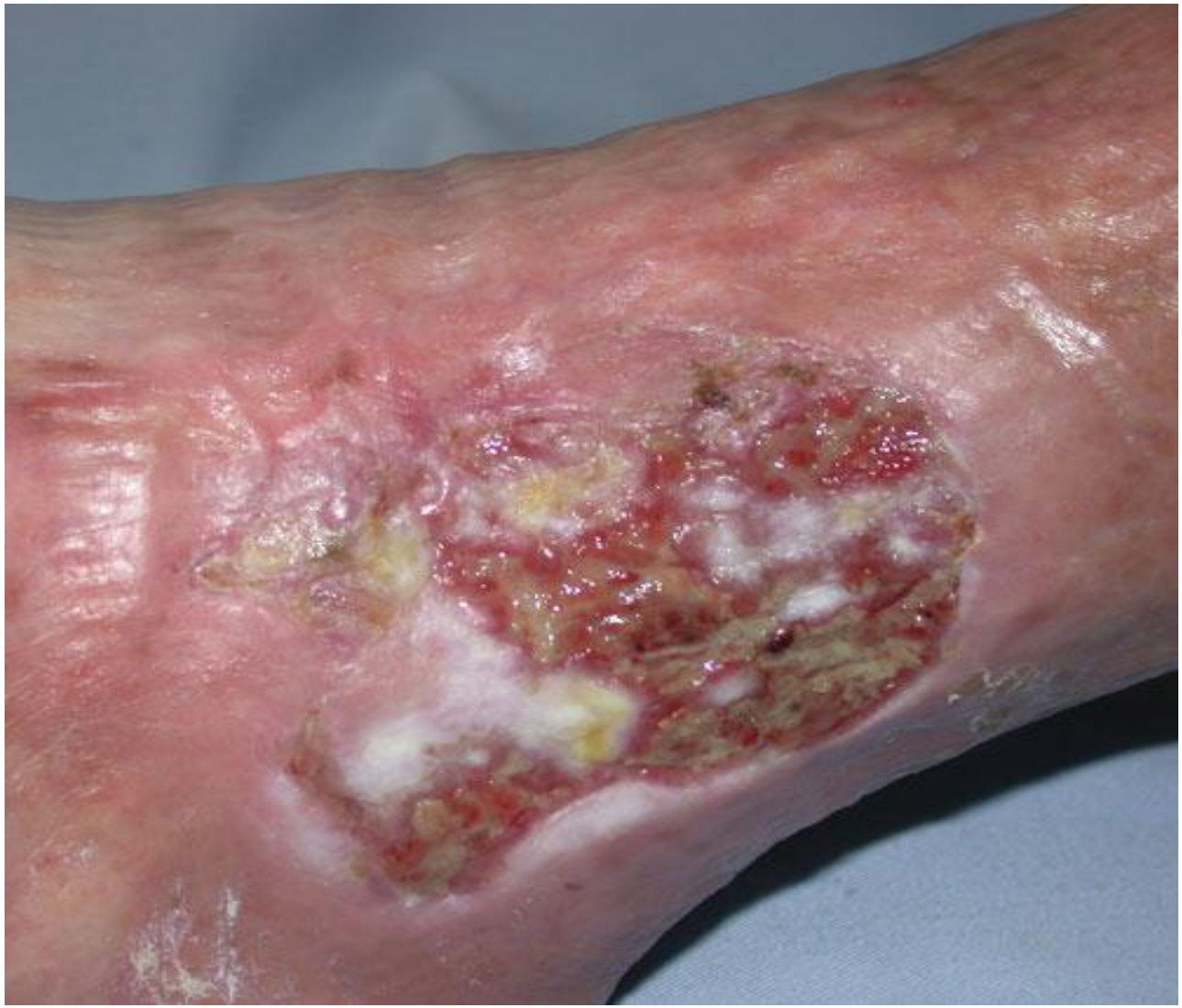

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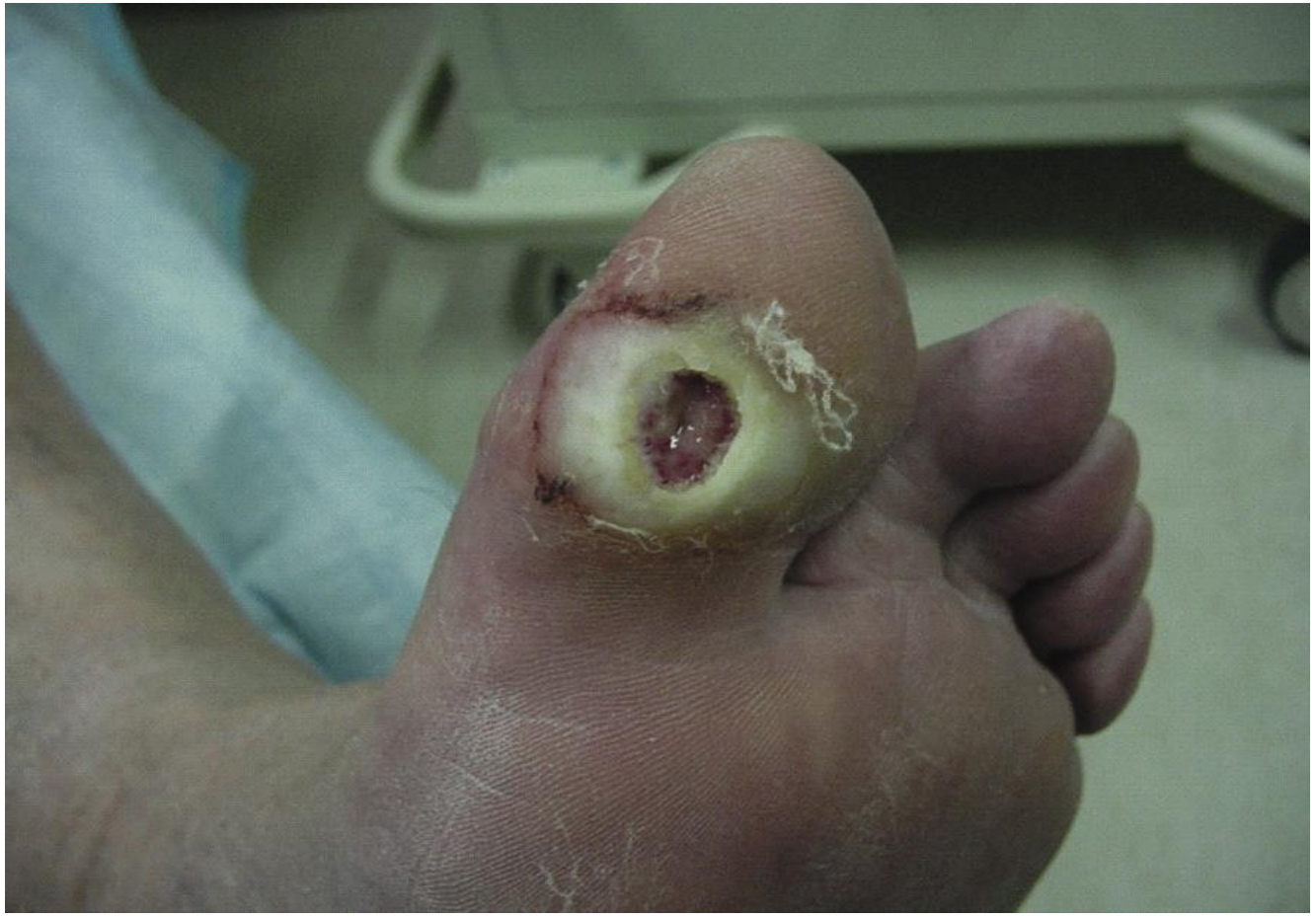

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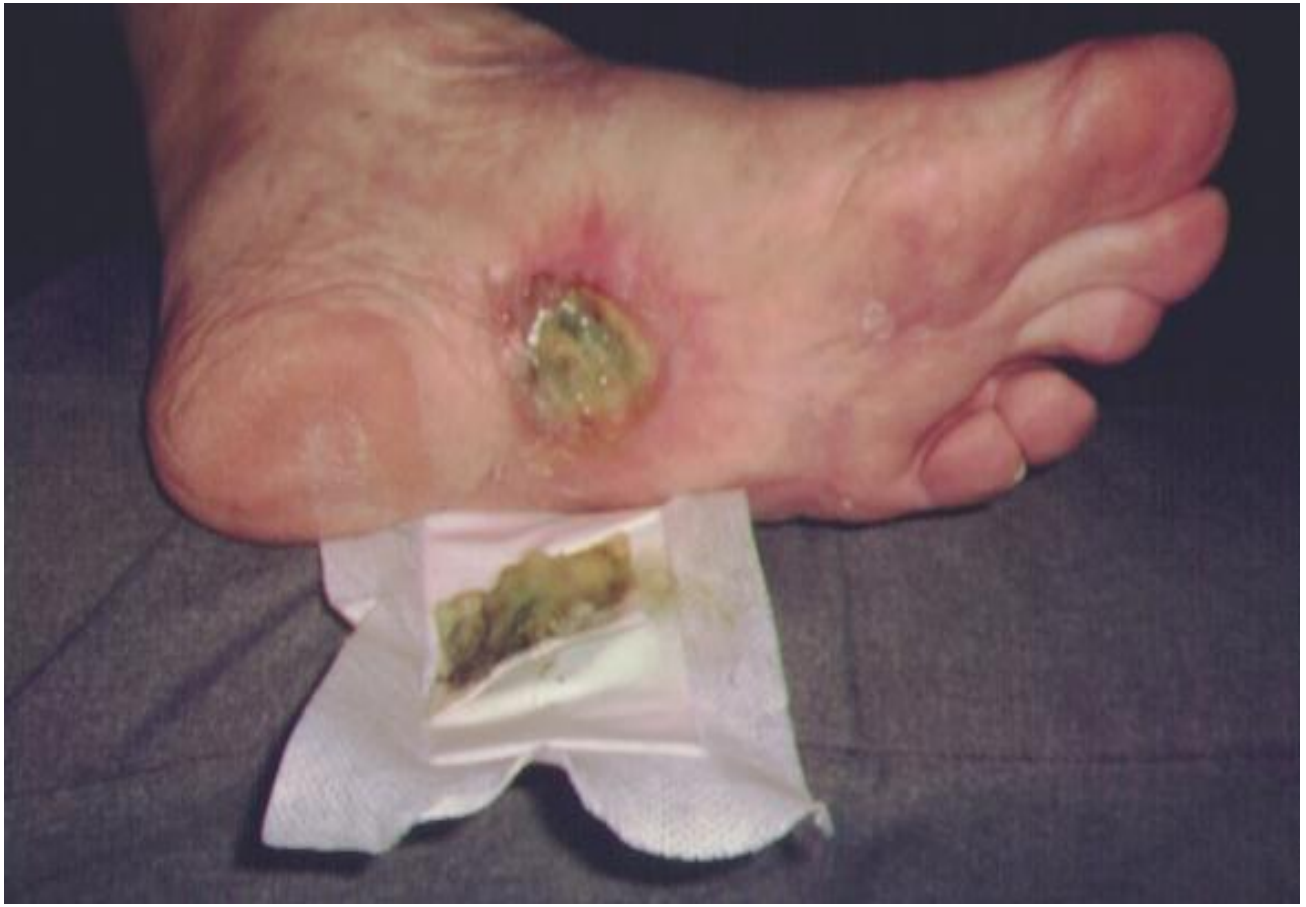

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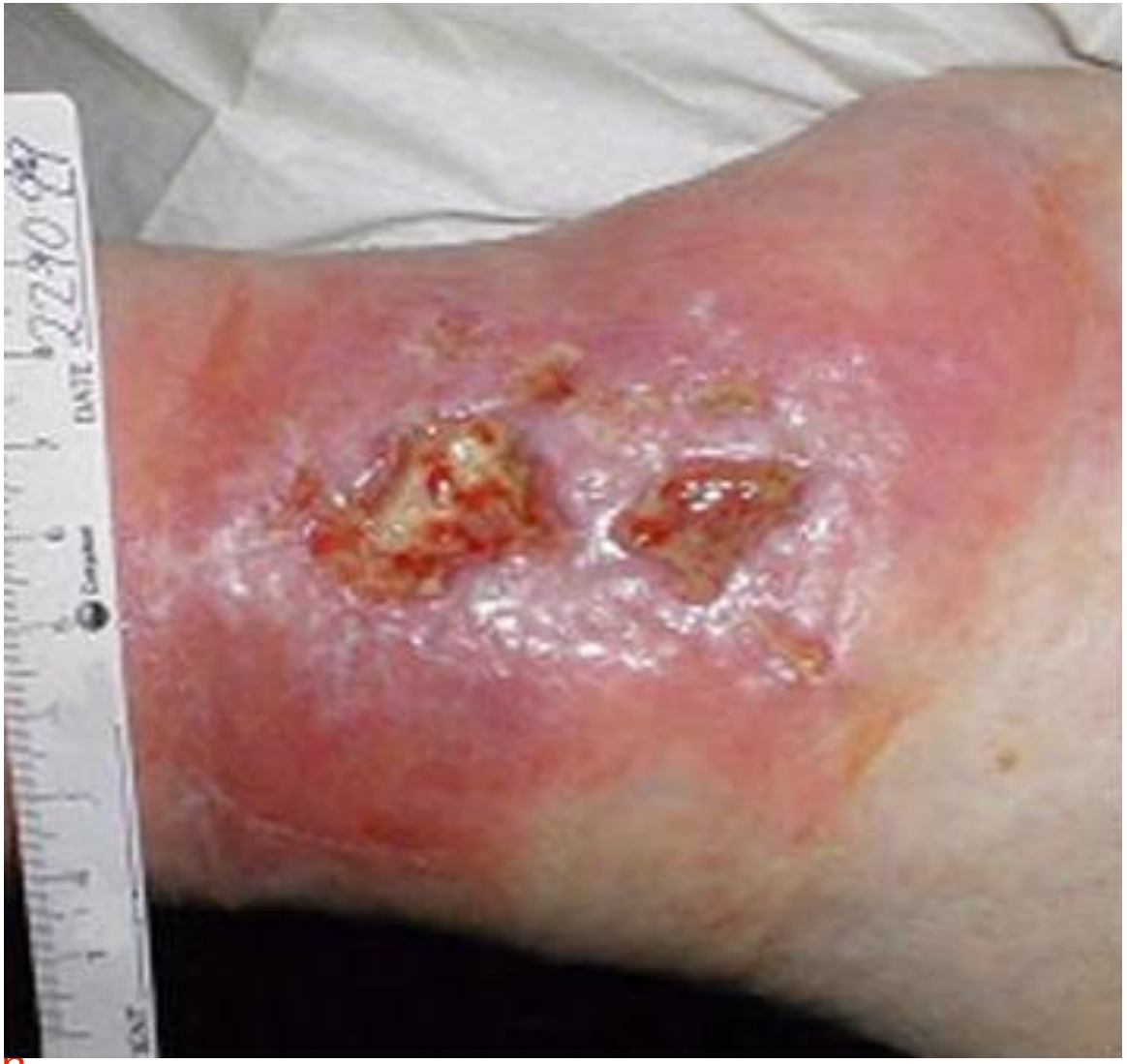

3. Wound Management plan.