Wound healing and Wound care.

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Learning outcome

by the end of this presentation, you should be able to:

- Describe the process of wound healing.
- Discuss complications of wounds healing.

Learning outcome

by the end of this presentation, you should be able to:

- Discuss the total care of a patient who presents with a wound / ulcer
- Compare the present mode of wound care with the past
- Contrast the present mode of wound care with the past

A wound is an area of the body whose normal integrity has been compromised

Wound can be found in:

- Skin,
- Mucosa,
- Bone,
- Brain

Can be:

- Acute
- Chronic

Aetiology

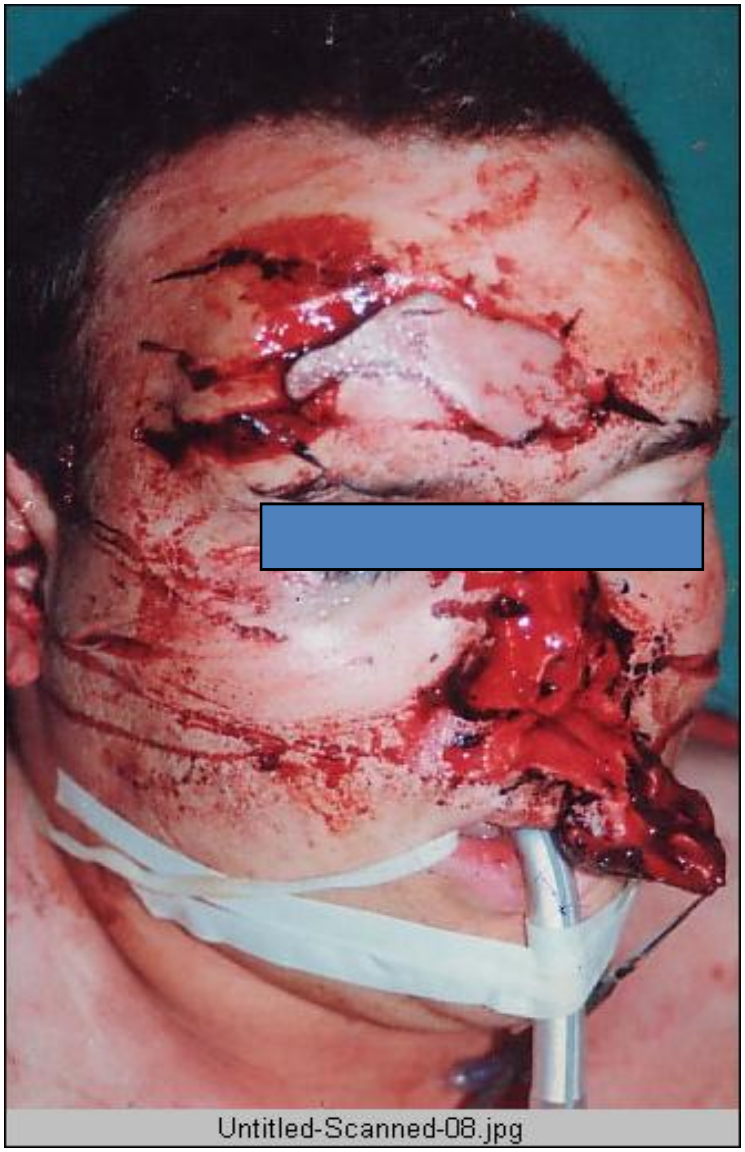
Acute wound

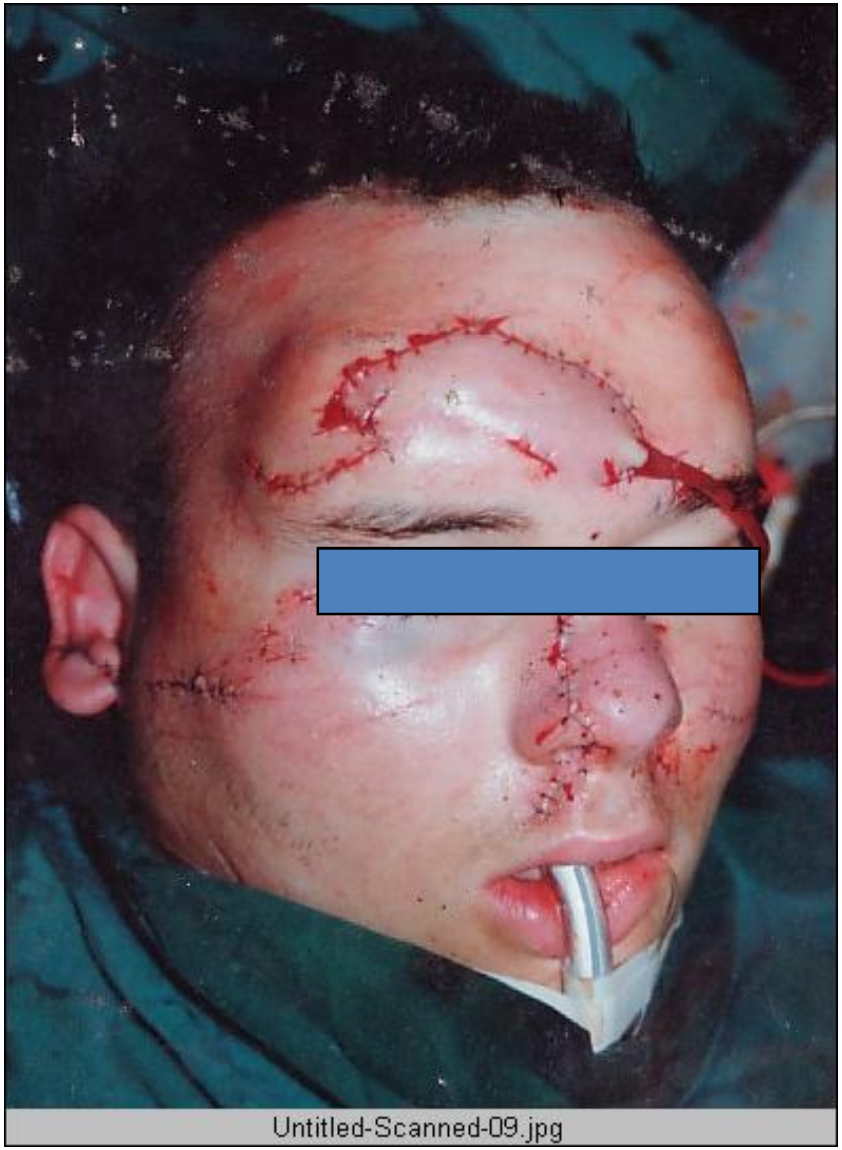
- Trauma
- Surgery
- Infection
- Inflammation

Chronic wound

- Poorly treated trauma
- Vascular disease
- Haematological disease
- Pressure
- Infection
- Endocrine
- Malignancy

Traumatic wounds on the face

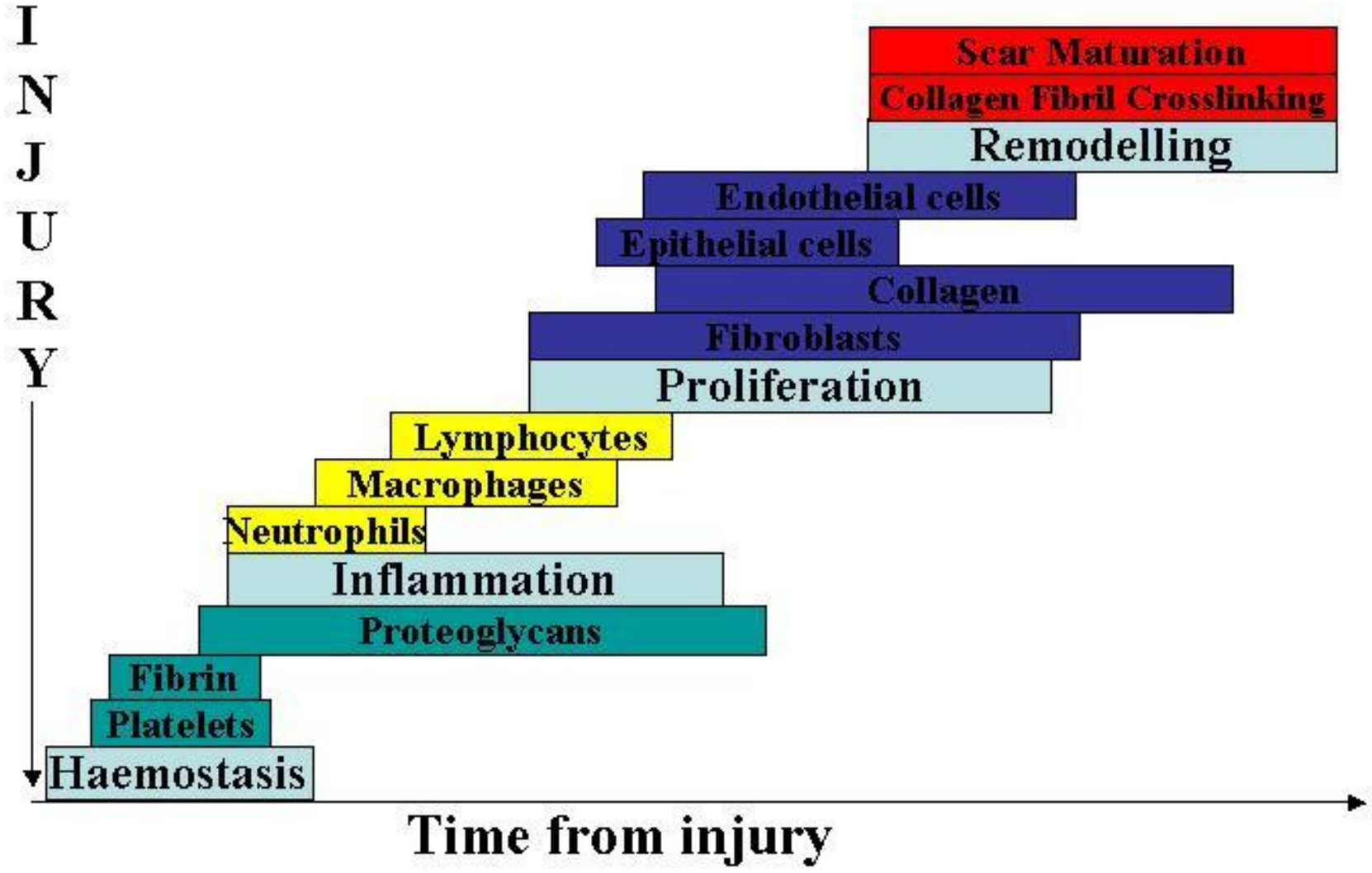




Phases of wound healing

- haemostasis
- inflammation
- proliferation
- maturation

Towards healing



Haemostasis

- microvascular injury leads to extravasation of blood,
- activation of coagulation cascade,
- constriction of injured vessels,
- clot formation,
- platelet aggregation.
- Fibrin, fibronectin, vW (von Willibrandt) factor, and thrombospondin, provide initial matrix for cellular migration

Inflammation

- Initial inflammatory exudate contains mainly polymorphs that later gets replaced by monocytes and lymphocytes.

- Monocytes must be present to trigger off fibroblast invasion of the wound as well as proliferation

Proliferation

- Fibroplasia
- Epithelialisation
- Angiogenesis

- commences 3 days into wound healing
- lasts weeks depending on the size of wound, and type of tissue involved.
- replacement of provisional fibrin/fibronectin matrix by a more definitive framework comprises

Fibroplasia

- commences 2-4 days after wounding.
- fibroblasts are attracted to site by PDGF and TGF-β
- they proliferate and construct new extracellular matrix (ECM) which initially comprises fibronectin and hyaluronan but later, collagen and proteoglycan.

Fibroplasia

- During the first three weeks, all wounds gain strength at the same rate
- thereafter the gain in strength becomes variable depending on the tissue.
- for the skin, peak tensile strength is achieved 60 days after injury.

Epithelialisation

- Epithelial mobilisation, migration, division, and differentiation are stimulated by an apparent loss of contact inhibition.
- EGF stimulates mitogenesis and chemotaxis
- β-FGF and keratinocyte growth factor (KGF) stimulate epithelial proliferation.

Epithelialisation

- Advancing cells bridge the wound
- Cellular differentiation from the base to the surface occurs.
- The rate of epithelialisation increases if:
  - the wound does not require debridement,
  - the basal lamina is intact
  - the wound is kept moist.

Angiogenesis

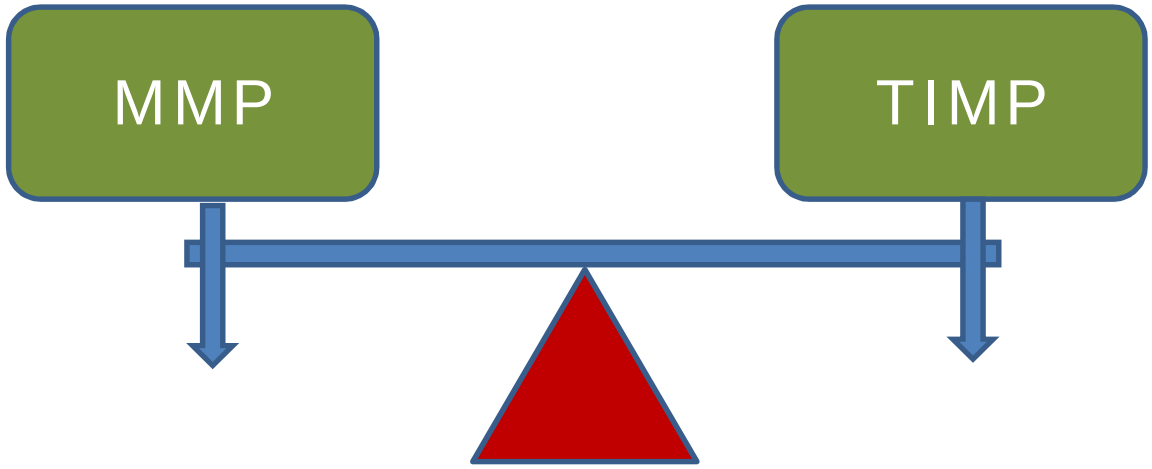
- Stimulated by:
  - TGF-β and PDGF from platelets
  - TNF-α and β-FGF from macrophages.
- The capillary sprouts invade fibrin to form granulation tissue network
- With time, blood vessel density reduces and scar tissue develops.

Maturation or remodelling

- Balance develops between collagen formation and degradation
- This reaches a steady state at 21 days after wounding.
- The eventual tensile strength achieved is only 80% of normal.

Maturation

- Collagen degradation is by matrix metalloproteinases (MMPs)
- Produced by: fibroblasts, granulocytes, macrophages.
- Tissue inhibitors of MMP (TIMP) deactivate MMPs.



Maturation

- While early collagen deposition is disorganised, local forces cause the laid down collagen to orientate in an organised fashion.
- Subsequently, activity of MMPs decreases
- Tissue inhibitors of MMP (TIMP) activity increase.

Maturation

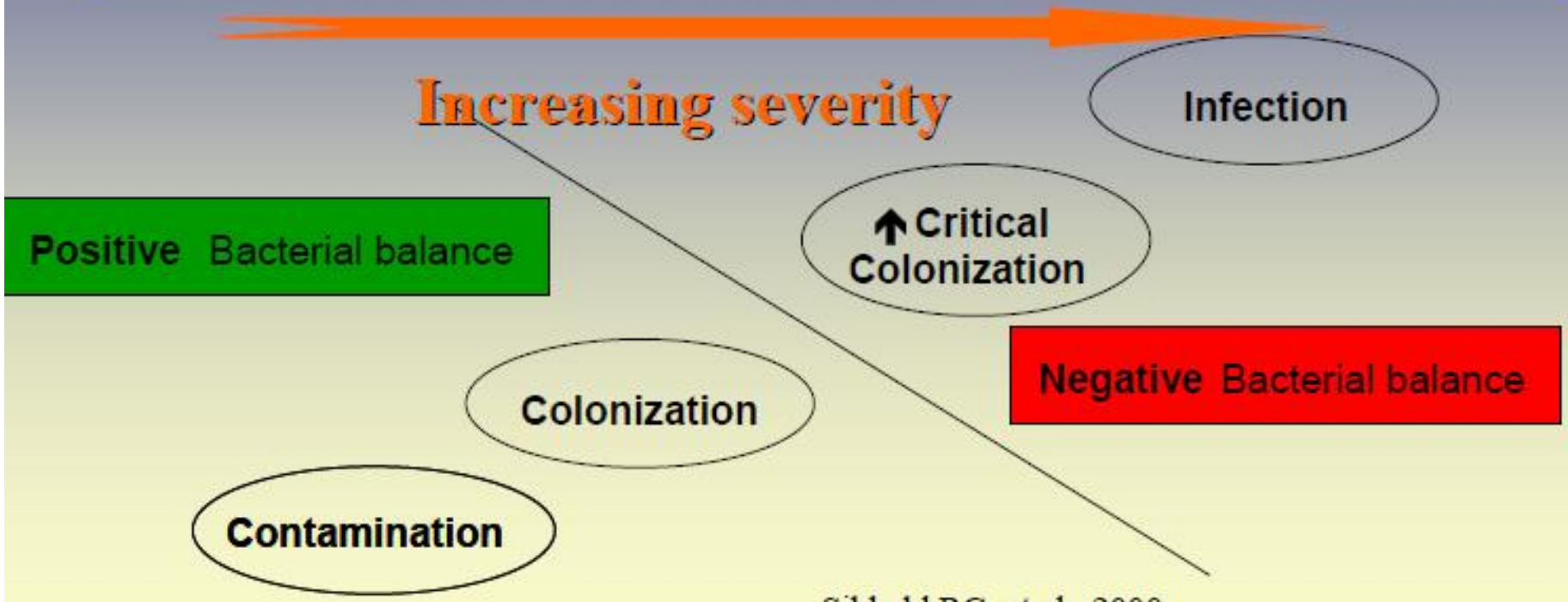
- macrophage, and fibroblast density becomes reduced by apoptosis
- capillary outgrowth stops
- acellular and avascular scar results

Complications of wound healing

- Wound infection
- Systemic infections
- Chronic wounds and ulcers
  - Scars and contractures
  - keloids
  - Lymphoedema
  - Bone complications: osteitis, osteomyelitis
  - Marjolin's ulcer
- Tetanus
- Pressure ulcers

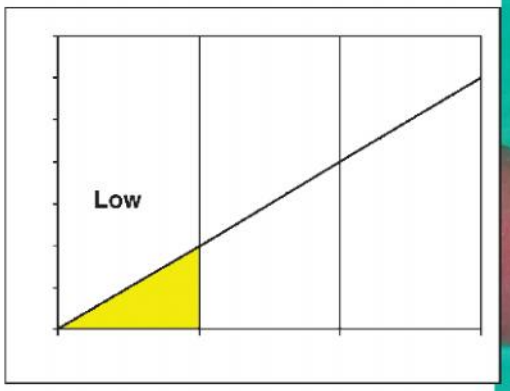
Bacterial Invasion: A Continuum

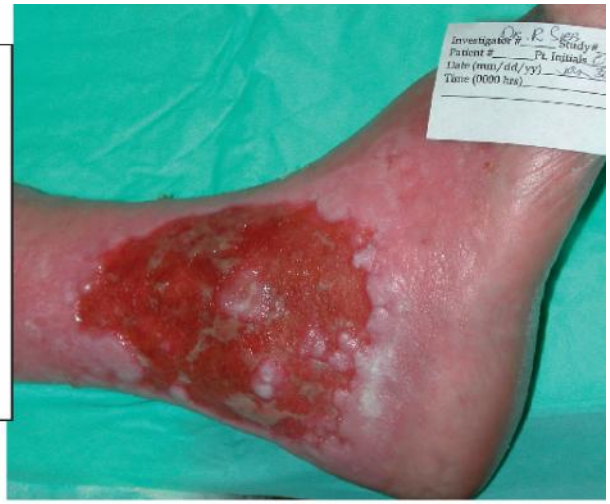
Risk of Infection = Organism number x Virulence / Host Immune Function (resistance)



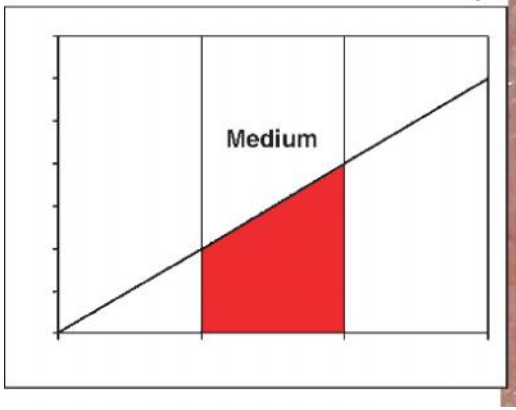
Sibbald RG, et al. 2000
Sibbald RG. In: Wound Bed Preparation, 2001.
Slide – adapted from Smith &Nephew TIME: to Prepare

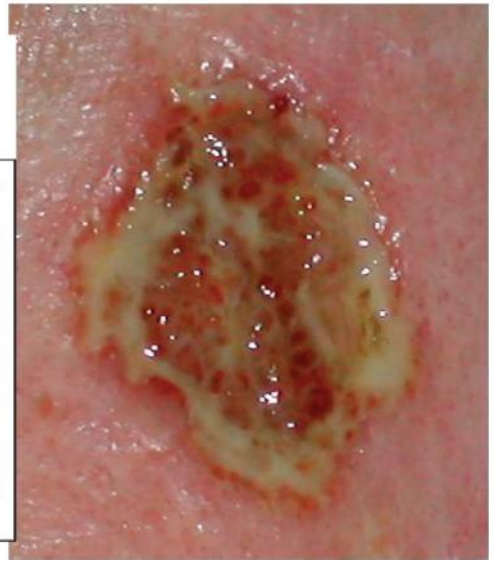
Contaminated or colonized



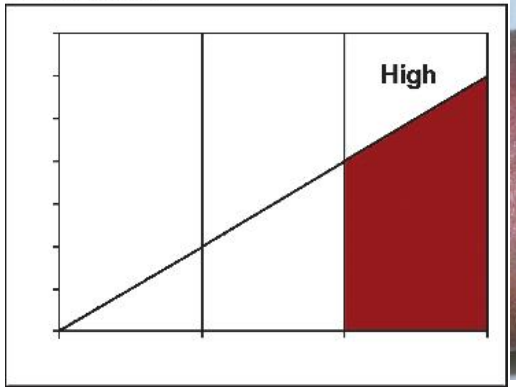


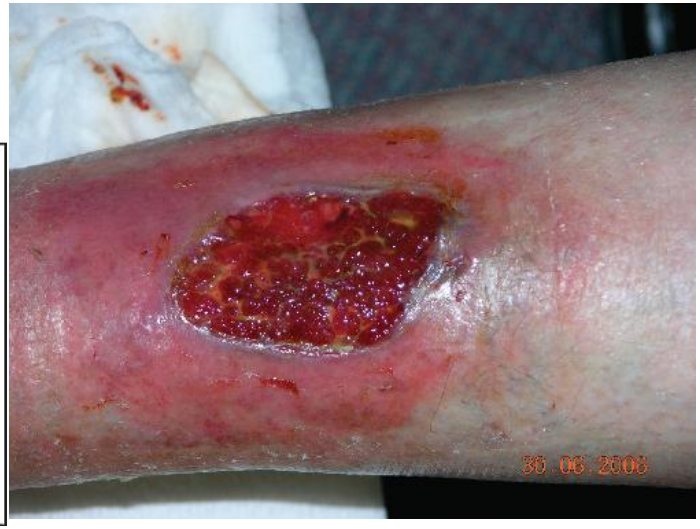
Critically colonized (local infection, covert infection, increased bacterial burden)



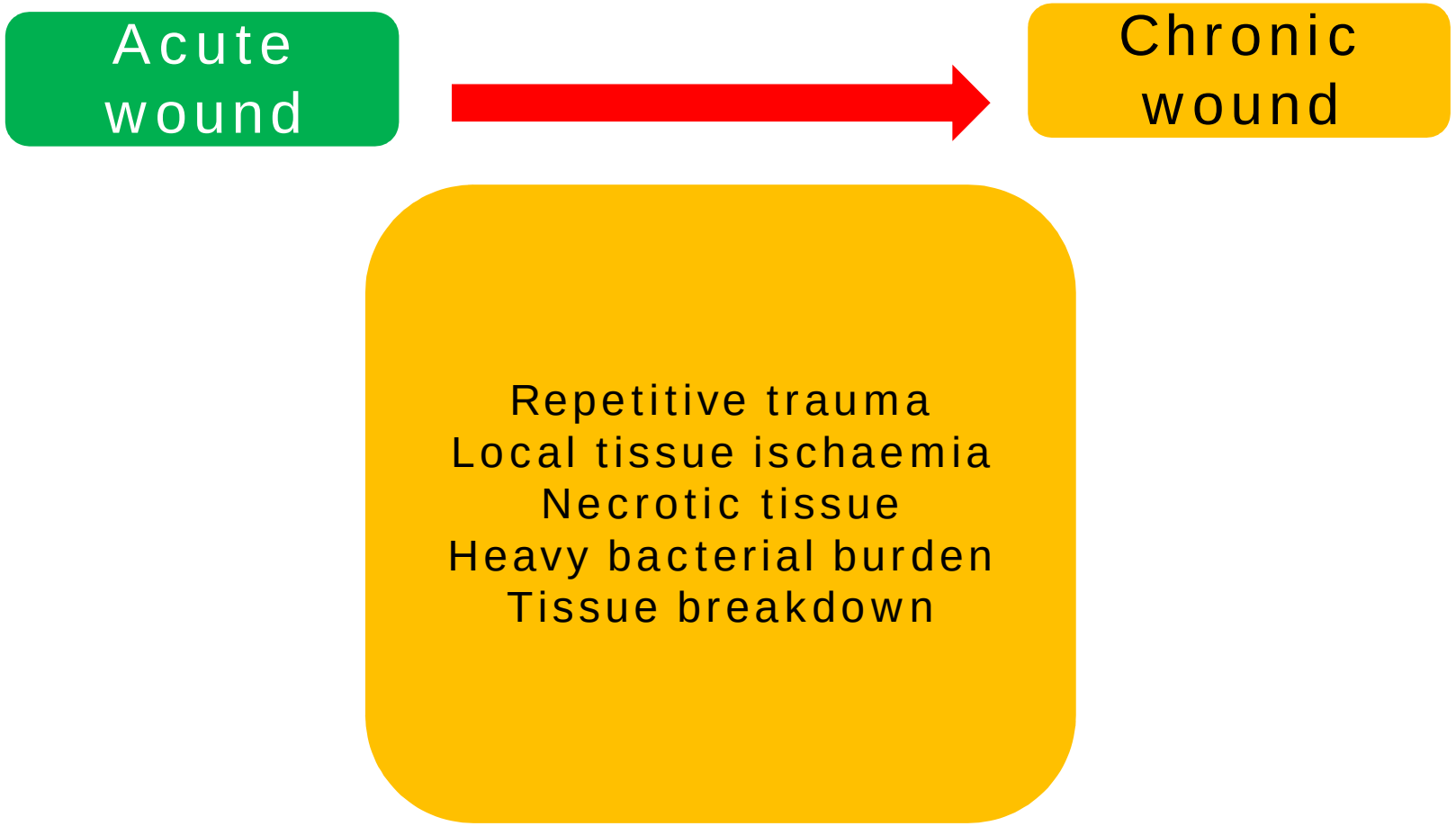


Infected





R. Gary Sibbald, Kevin Woo, Elizabeth A. Ayello. Increased Bacterial Burden and Infection: The Story of NERDS and STONES. ADV SKIN WOUND CARE 2006;19:447-61


Acute wound
Chronic wound
Repetitive trauma
Local tissue ischaemia
Necrotic tissue
Heavy bacterial burden
Tissue breakdown

Differences between acute and chronic wound healing

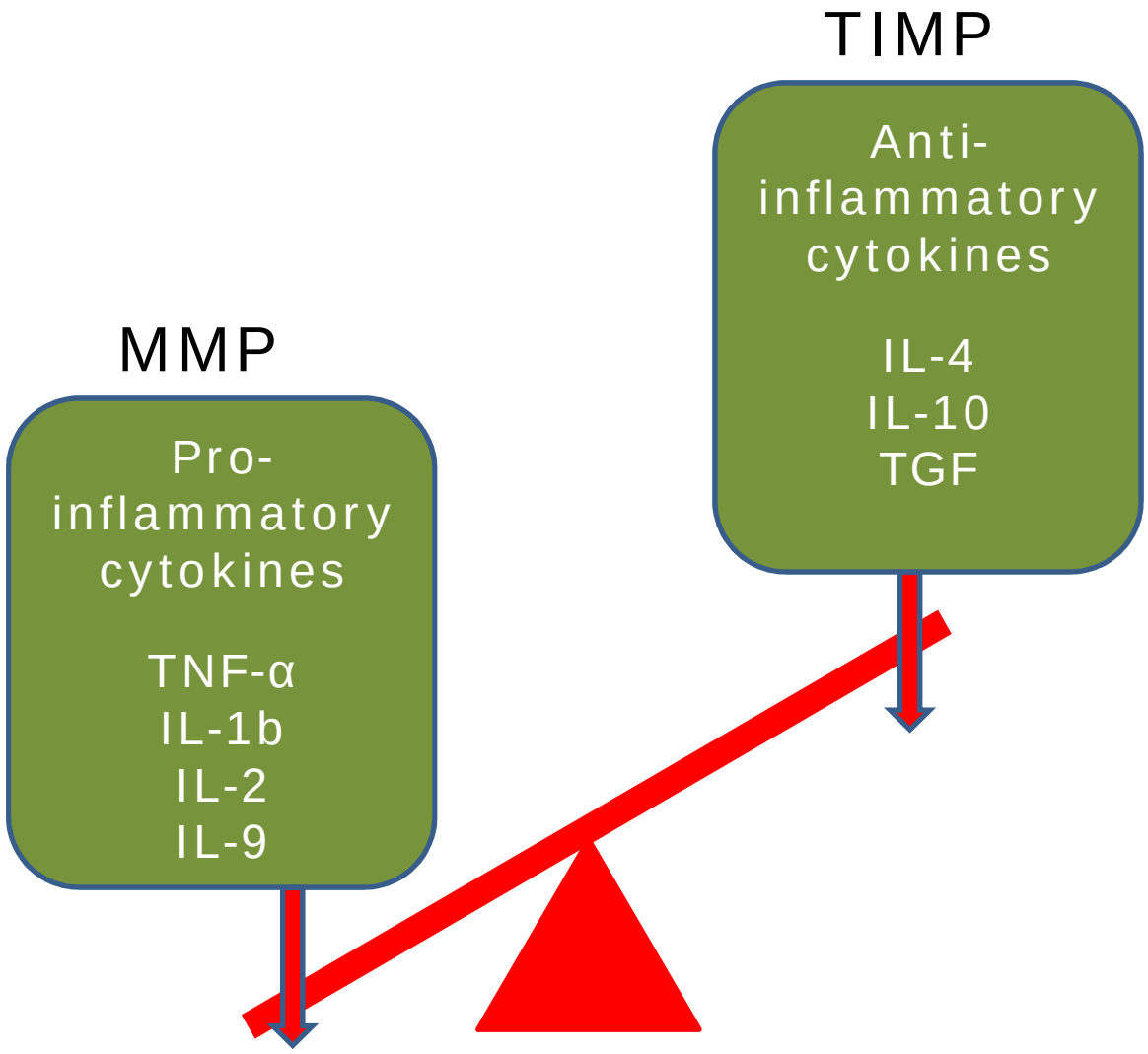
- In a chronic wound, the timely and orderly manner of acute wound healing enumerated previously is disrupted.

- This disruption occurs in most cases in the inflammatory and the proliferative phases

Differences between acute and chronic wound healing

The disruption manifests in:

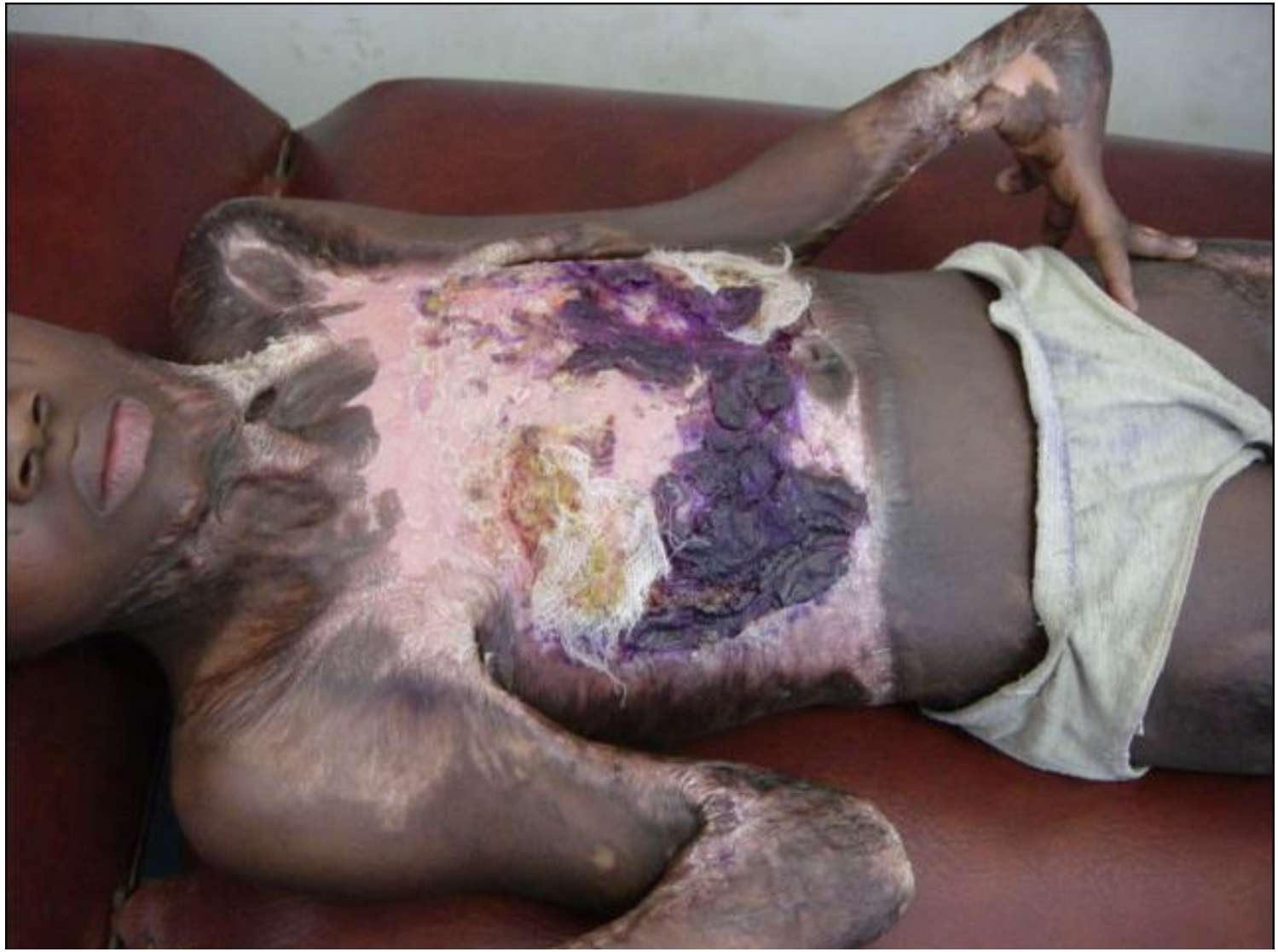
- alterations in protease activity,
- alteration in cytokine profile and inflammatory response,
- changes in cellular profile and activity,
- changes in the composition of extracellular matrix and environment,

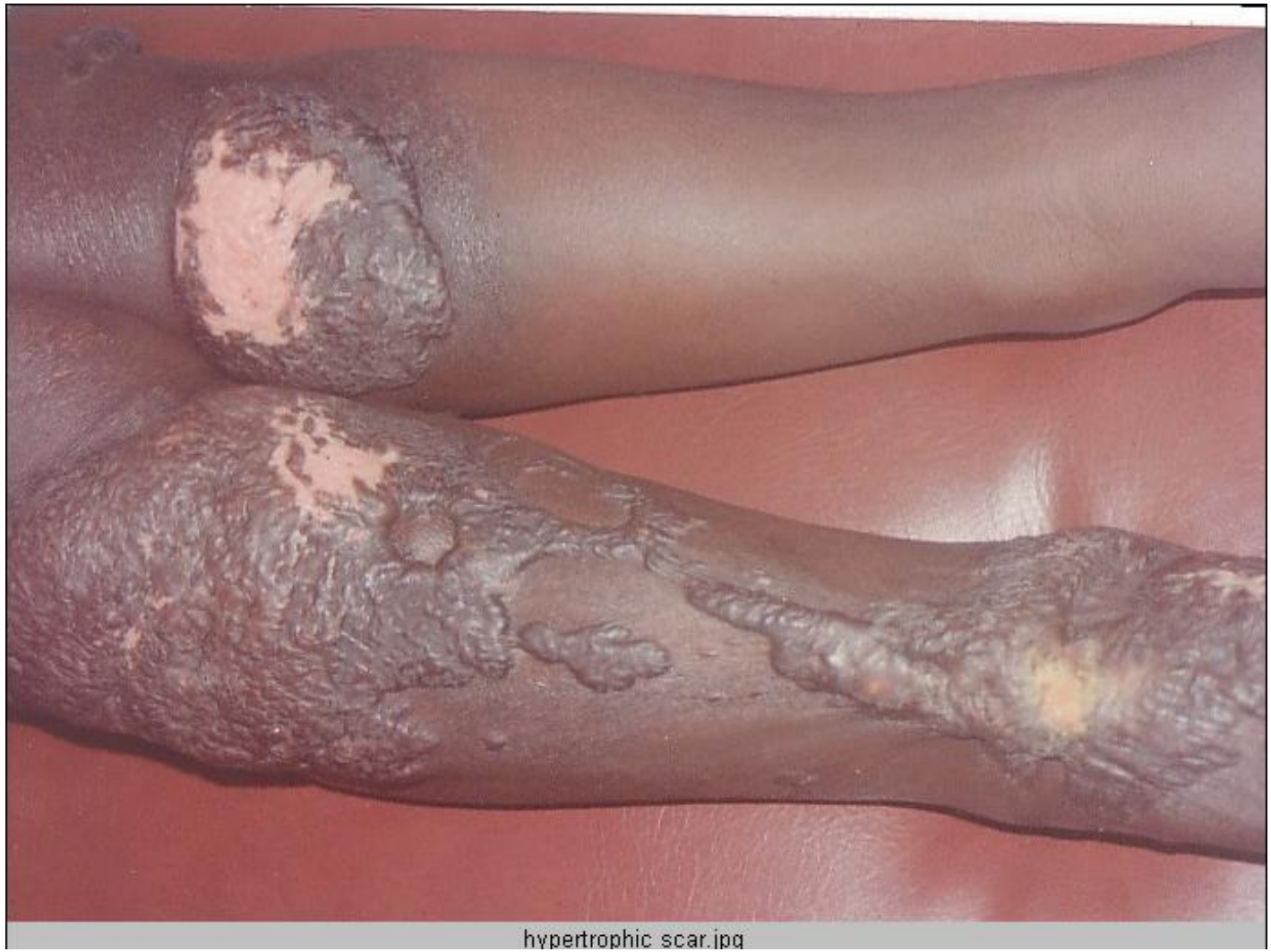

TIMP
Anti-inflammatory cytokines
IL-4
IL-10
TGF
MMP
Pro-inflammatory cytokines
TNF-α
IL-1b
IL-2
IL-9

Differences between acute and chronic wound healing

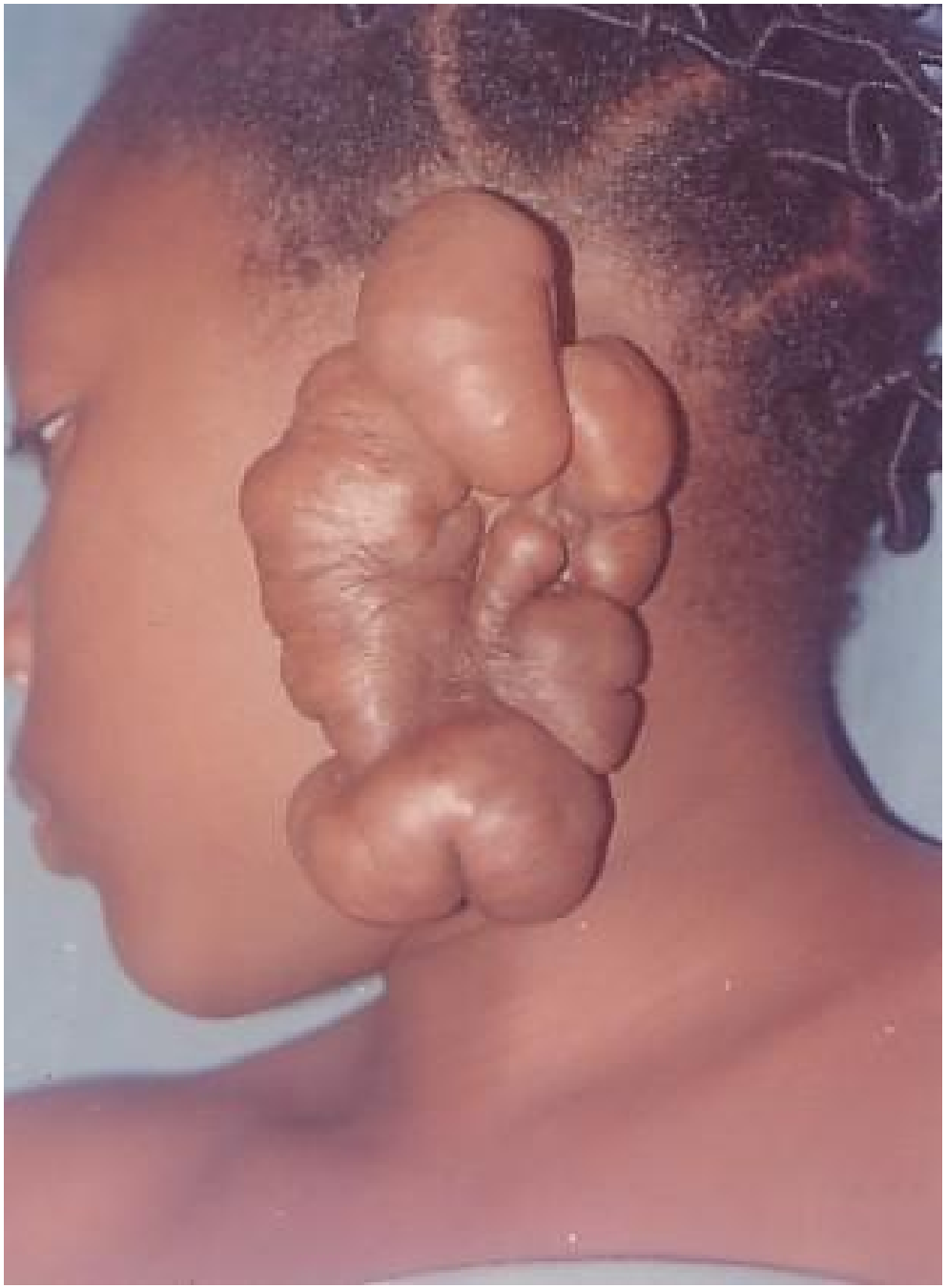
The disruption manifests in:

- presence of free radicals and role of nitric oxide,
- accumulation of necrotic tissue and slough,
- presence of micro organisms,
- disease specific pathological change.



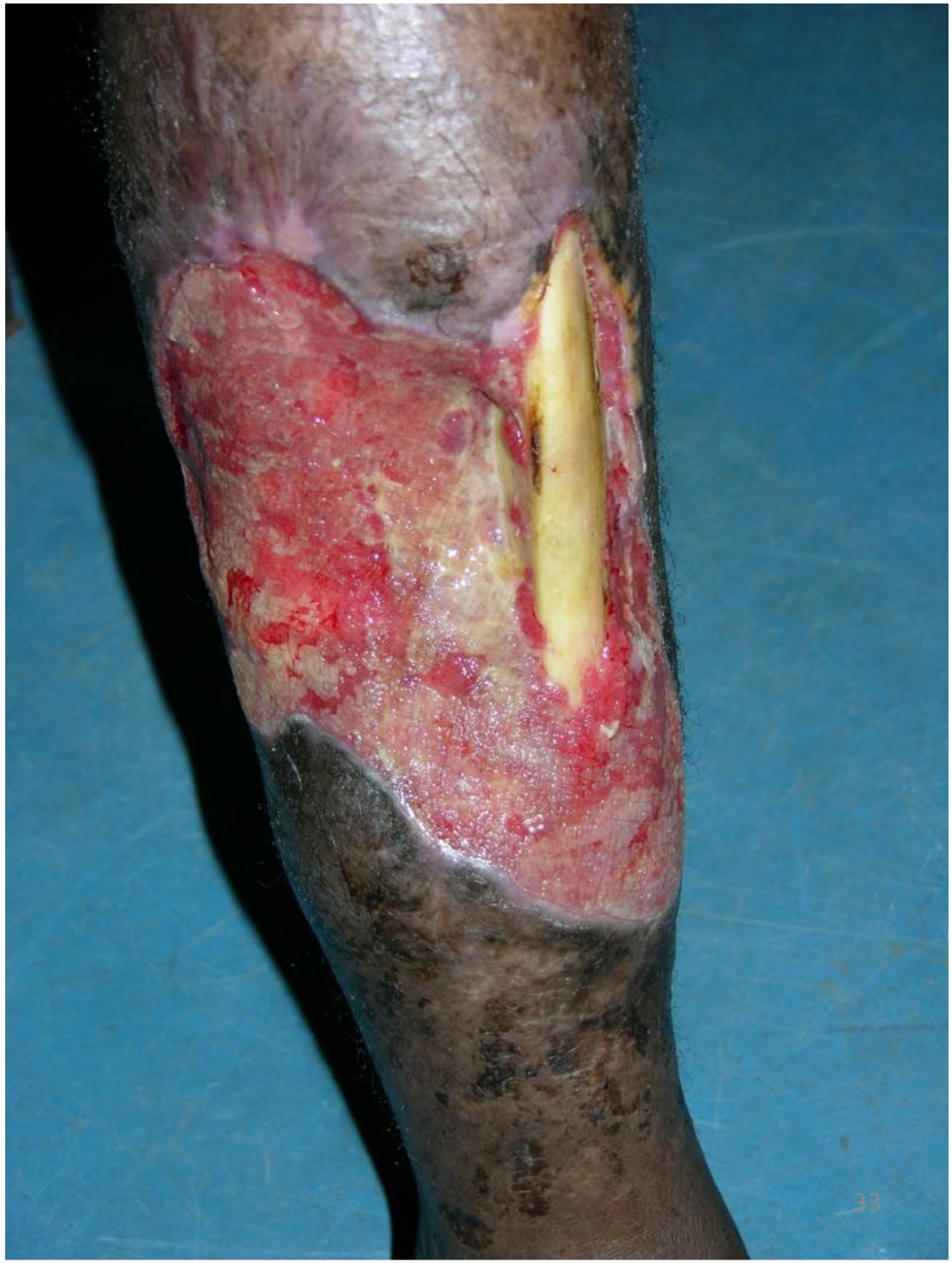


hypertrophic scar.jpg



Keloid

Bone complications



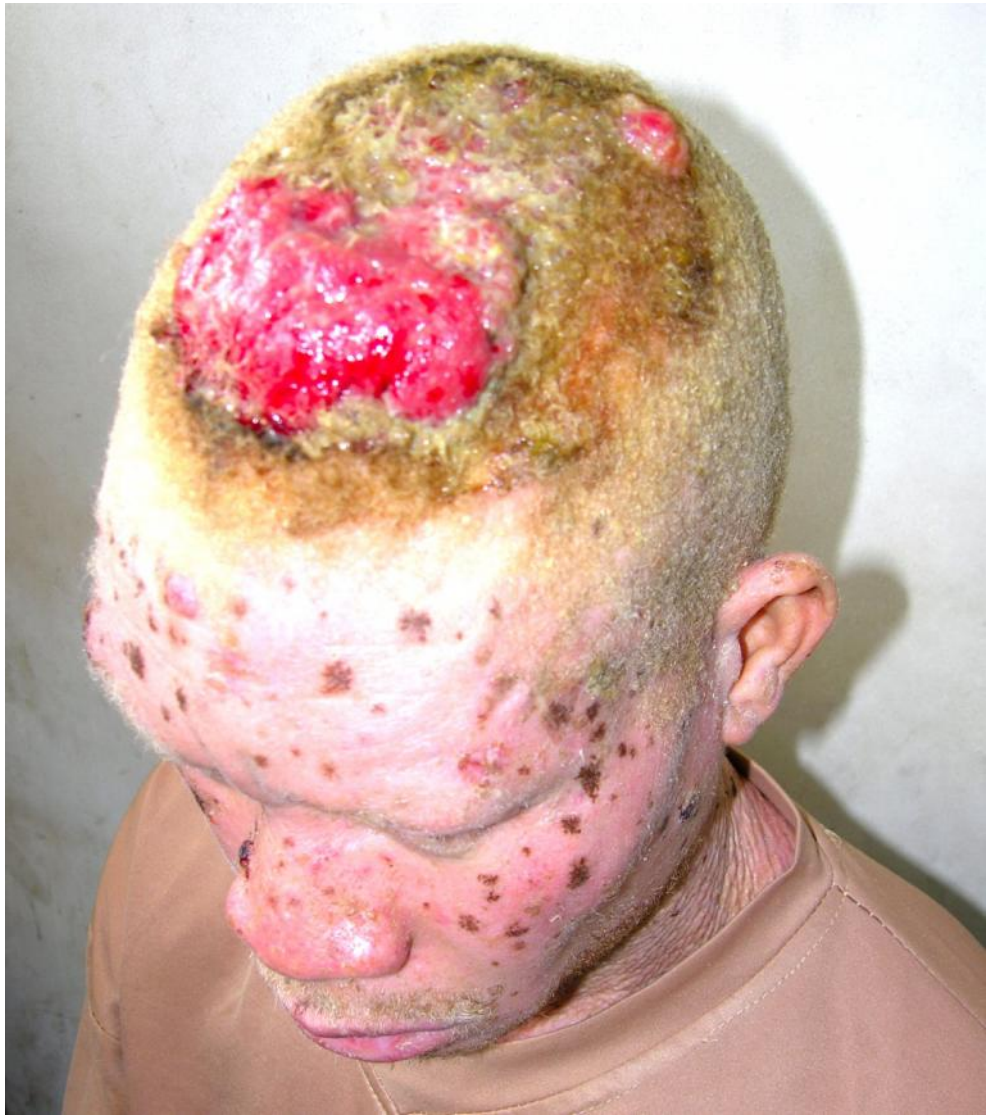
Lymphoedema

Total care of a patient who presents with a wound / ulcer

- Identify and treat the cause
- Address patient-centered concerns
- Provide local wound care

- Denis Okan, Kevin Woo, Elizabeth A. Ayello, R. Gary Sibbald. The Role of Moisture Balance in Wound Healing. Adv Skin Wound Care 2007; 20: 39-53

Identify and treat the cause

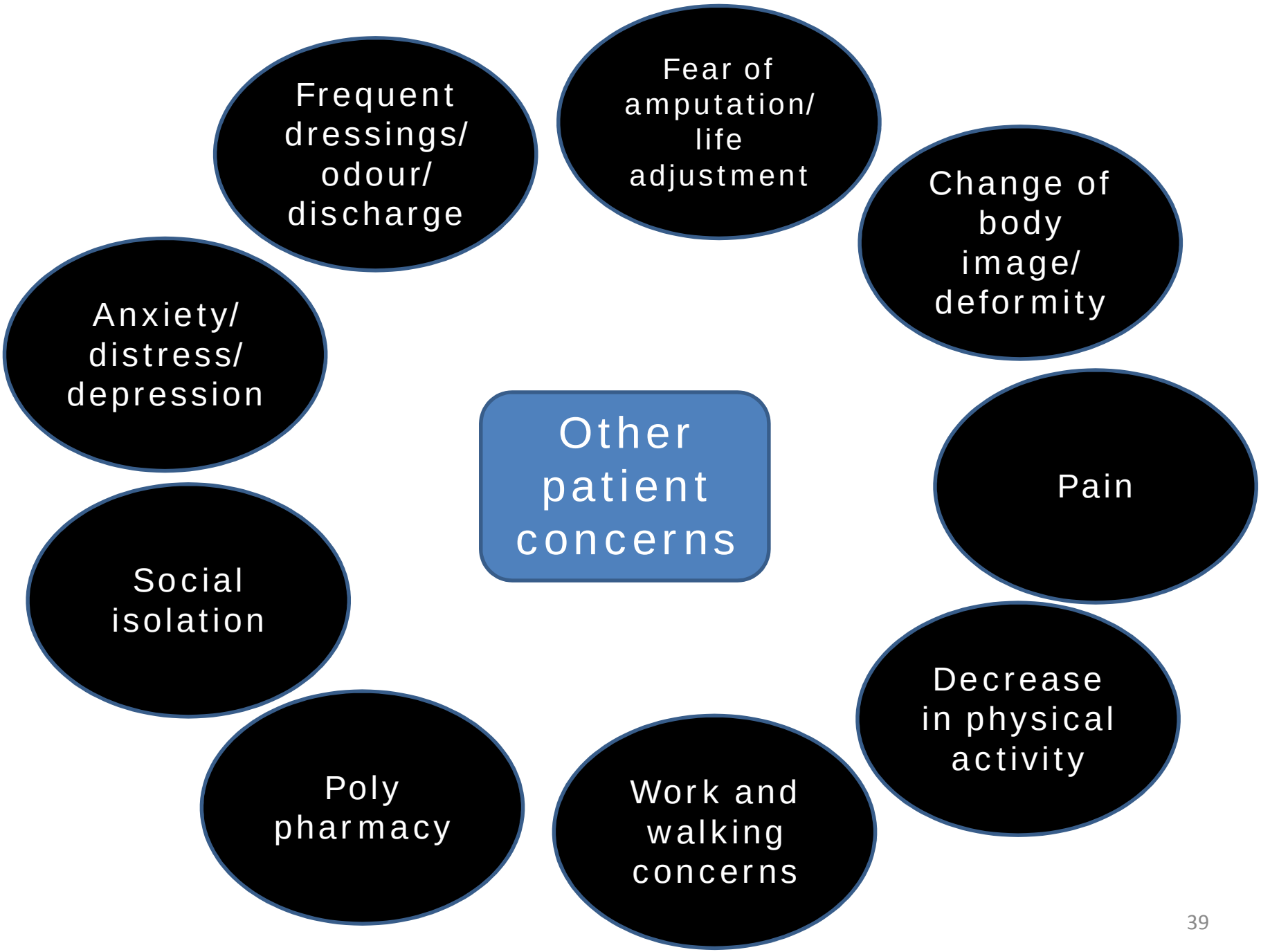


Identify and treat the cause

- Venous leg ulcers
- Arterial ulcers
- Pressure ulcers
- Diabetic foot ulcers
- Malignant ulcers and Marjolin’s ulcer

Address patient-centered concerns

- Provide emotional support
- Assess and consider financial situation
- Provide patient and family education
- Assess and provide/facilitate optimum health care


Other patient concerns
Frequent dressings/ odour/ discharge
Fear of amputation/ life adjustment
Change of body image/ deformity
Anxiety/ distress/ depression
Pain
Social isolation
Decrease in physical activity
Poly pharmacy
Work and walking concerns

Wound information	Is tetanus-prone	Is not tetanus prone
Time since injury	>6 hours	<6 hours
Depth of injury	> 1 cm	< 1 cm
Mechanism of injury	Crush, burn, gunshot, frostbite, penetration through clothing	Sharp cut
Dead tissue present	Yes	No
Foreign material (grass, dirt, etc.) contamination	Yes	No

Pain management

- Injury pain (immediate, severe, regressive), background pain which is prolonged until wounds are healed
- Procedural pain (dressing changes, physiotherapy, post operative) which is severe and repetitive.

- WHO pain ladder
- Appropriate timing

Total care of a patient who presents with a wound / ulcer

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Provide local wound care

Determine the potential for healing

Assess the wound

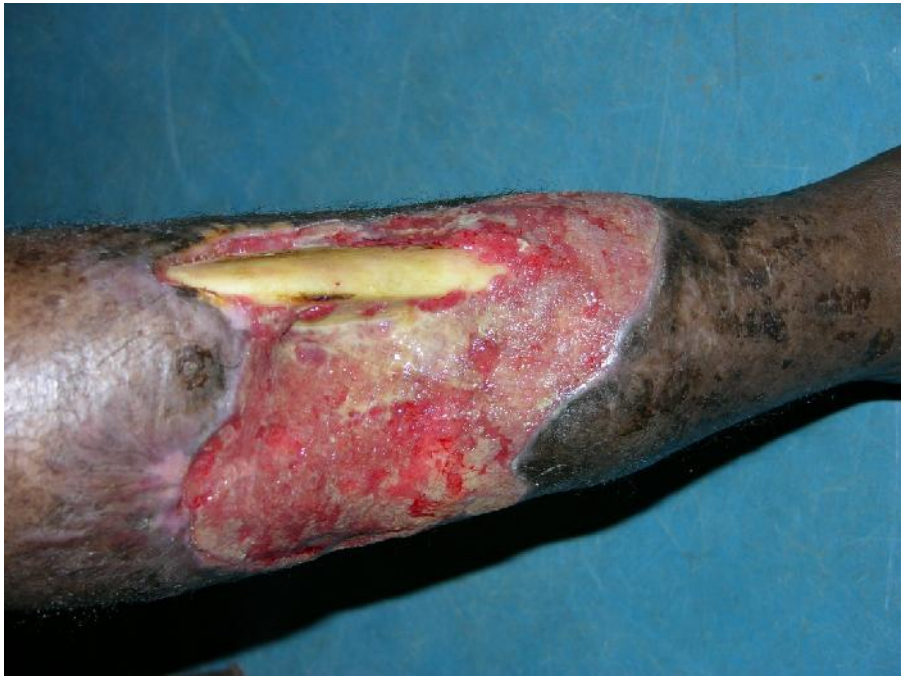
a) Obtain the wound history
b) Assess and monitor the physical characteristics of the wound
c) Assess and manage wound pain

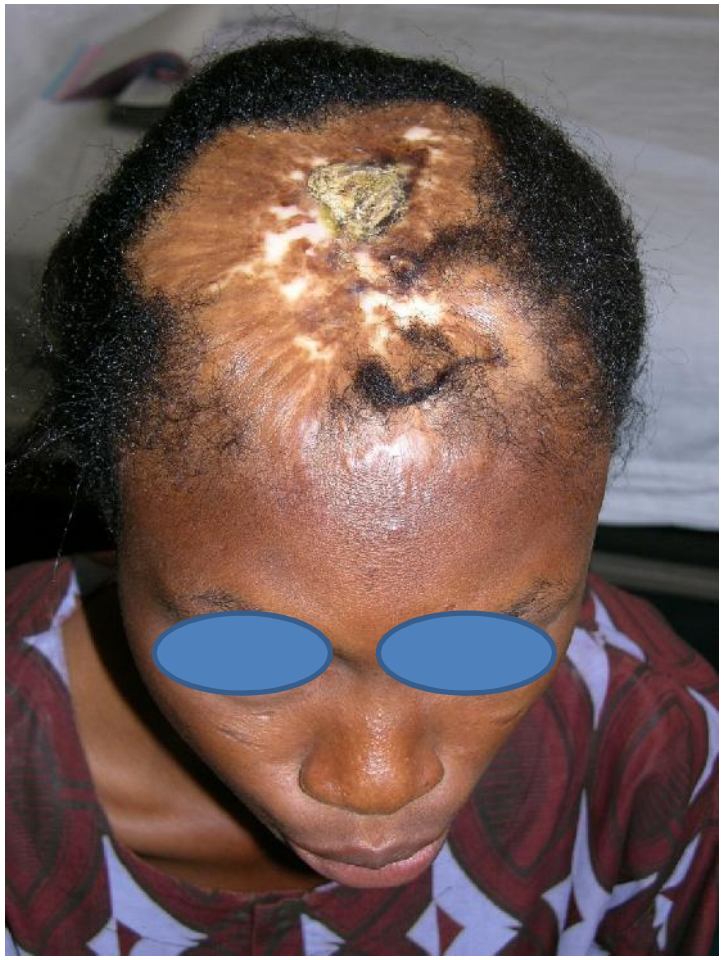
Local Wound Care

a) Tissue concerns
b) Infection/Inflammation concerns
c) Moisture concerns
d) Edge concerns

Determine the potential for healing

Blood supply





Determine the potential for healing

Risk factors and co-morbidities that may affect wound healing:

a) Drugs i.e. immunosuppressive agents and systemic steroids
b) Periwound edema in a chronic wound
c) Serum albumin: <30g/L delays healing, <20g/L very hard to heal or non-healing wounds
d) Hemoglobin: <100g/L delayed healing, ≤ 70-80g/L very hard to heal or non-healing wounds
e) Diseases or treatments that impair immunity such as rheumatoid arthritis and collagen vascular diseases (lupus, scleroderma, dermatomyositis), chemotherapy and radiation therapy
f) Chronic diseases such as uncontrolled diabetes, hepatic/renal/lung disease and vascular disease

Treat the wound

Determine the potential for healing

Assess the wound

a) Obtain the wound history
b) Assess and monitor the physical characteristics of the wound
c) Assess and manage wound pain

Local Wound Care

a) Tissue concerns
b) Infection/Inflammation concerns
c) Moisture concerns
d) Edge concerns

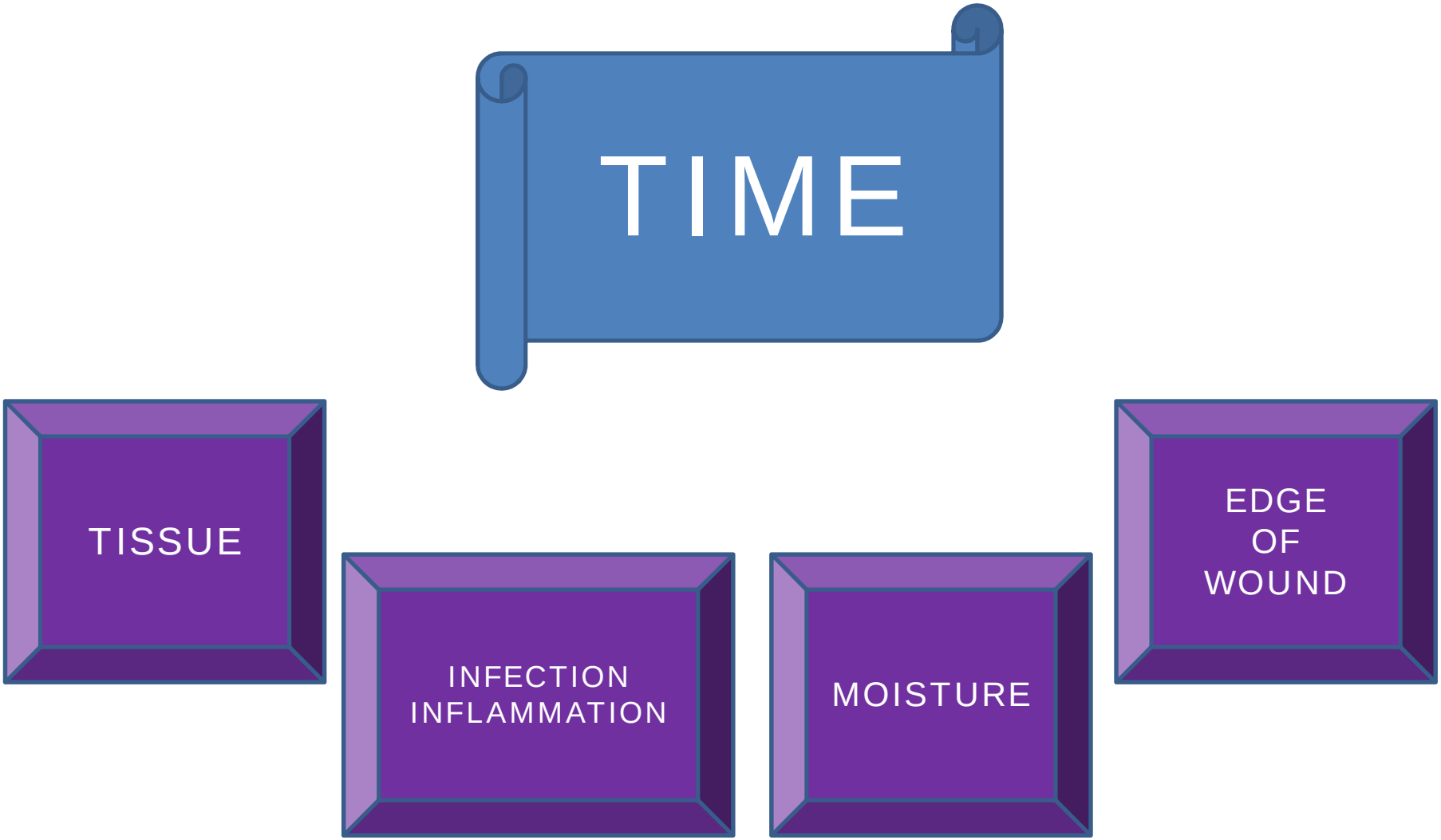
Local Wound Care

Wound Bed Preparation:

To accelerate endogenous healing

To facilitate the effectiveness of other therapeutic measures

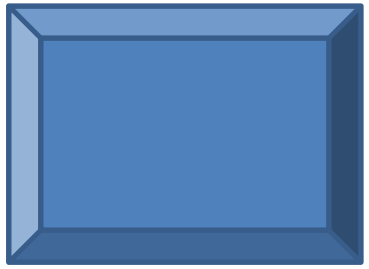
TIME principles of Wound Bed Preparation



Wound cleansing

(from page 12 of UK policy doc.)

- Normal saline
- High quality pure water
- Chlorhexidine gluconate 0.015 - 0.05% w/v
- Povidone-iodine 10%
- Cetrimide 0.15%



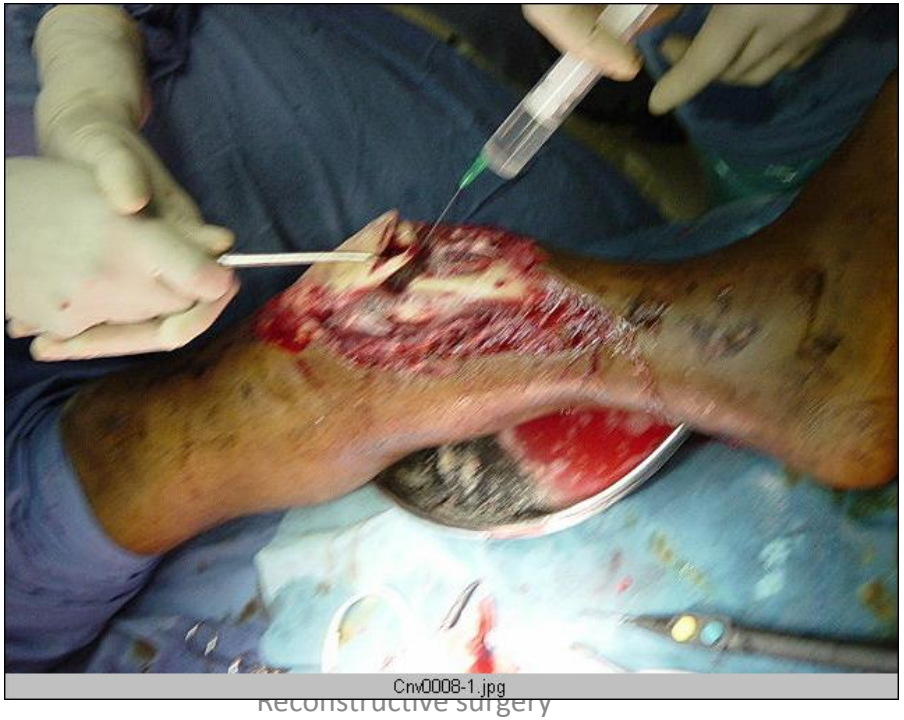
The effects of water compared with other solutions for wound cleansing

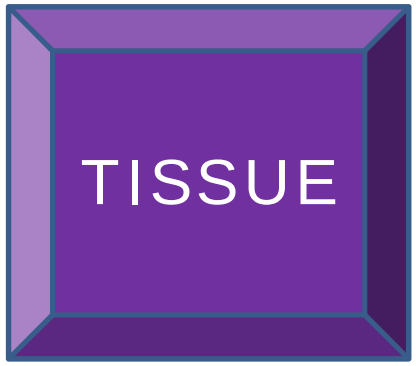
- Water is frequently used for cleaning wounds to prevent infection. This can be tap water, distilled water, cooled boiled water or saline.
- Using tap water to cleanse acute wounds in adults does not increase the infection rate, however, there is no strong evidence
- That cleansing per se is better than not cleansing. The reviewers concluded that where tap water is high quality (drinkable), it may be as good as other methods such as sterile water or saline (and more cost-effective),
- But more research is needed.

Wound cleansing

Swab

Irrigate



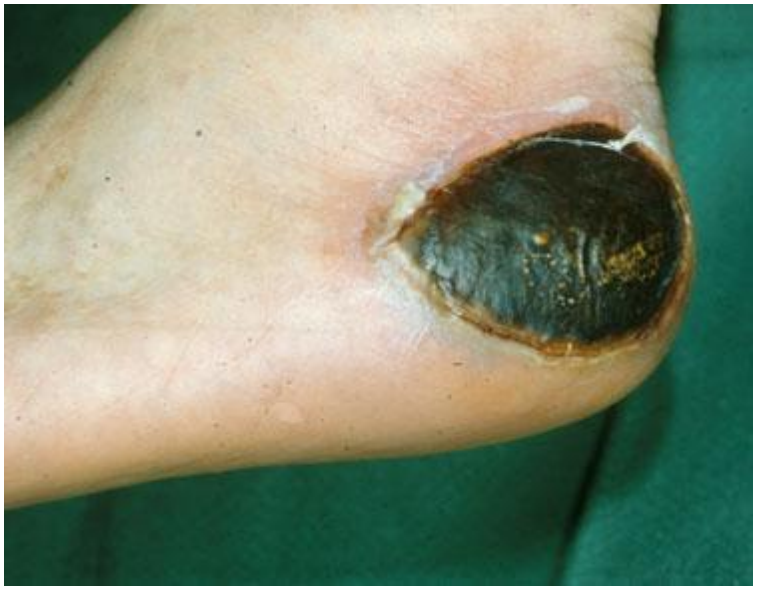


For clean wounds, no raw surface:
Gauze dressing

For raw surface like skin graft donor site, partial thickness burn, abrasion:
Non-adherent dressings
Opsite, Tulle gras, Jelonet, Sofratulle, Vaseline gauze, Bactigras, Melolin, Mepitel,

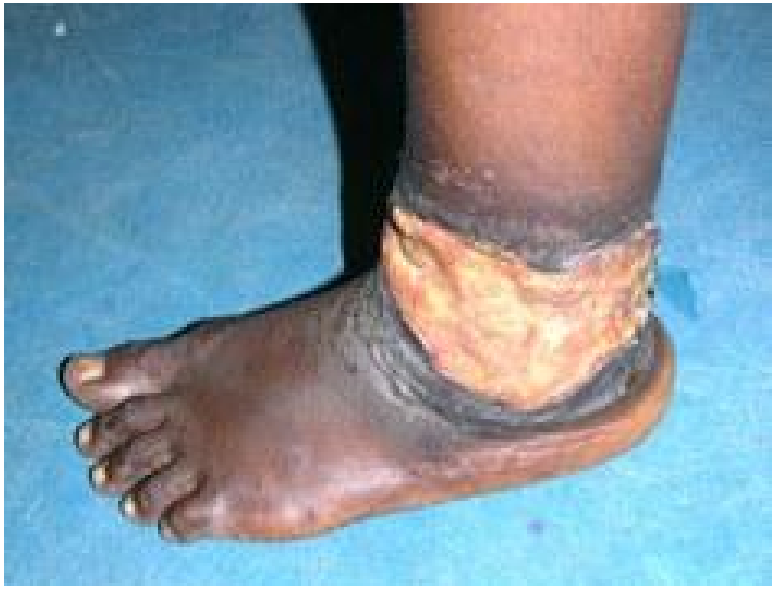
Temporary biosynthetic skin

Non viable TISSUE



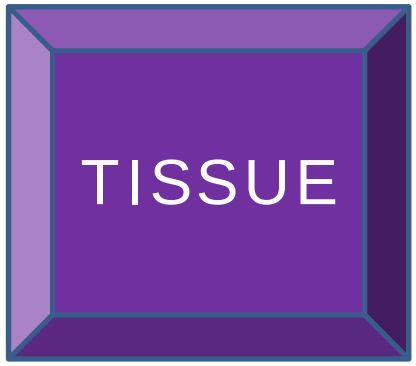
NECROTIC TISSUE

Dehydrates, shrinks, inhibits autolysis and separation becomes delayed indefinitely



SLOUGH

Mixture of fibrin, deoxyribonucleo-protein, serous exudates, leukocytes and bacteria

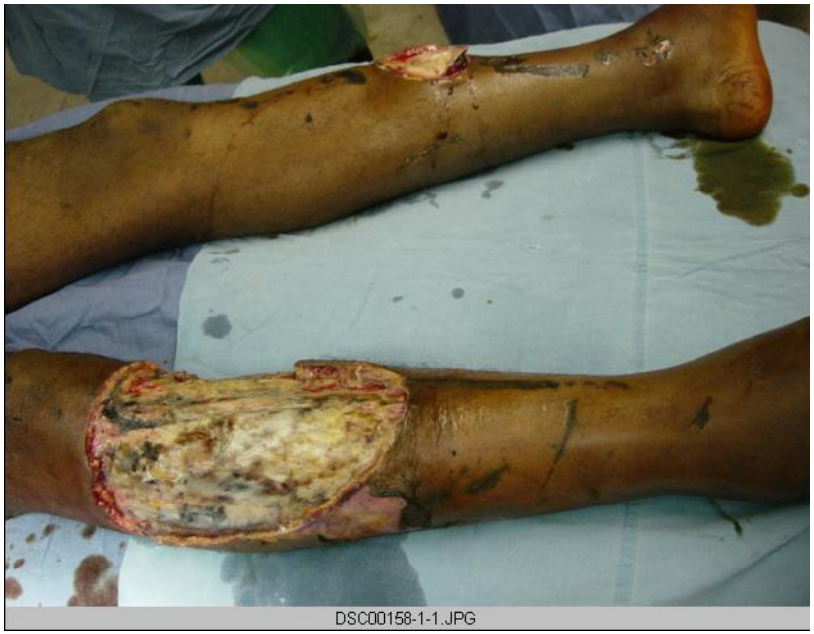


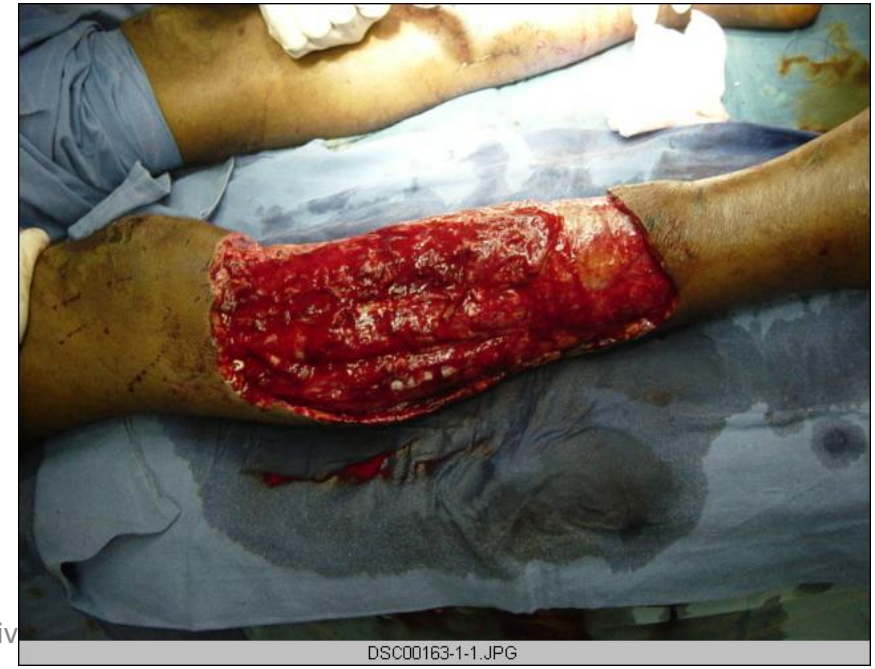
Methods of debridement of non-viable tissue:

- surgical
- mechanical
- autolytic
- chemical
- biological
- enzymatic

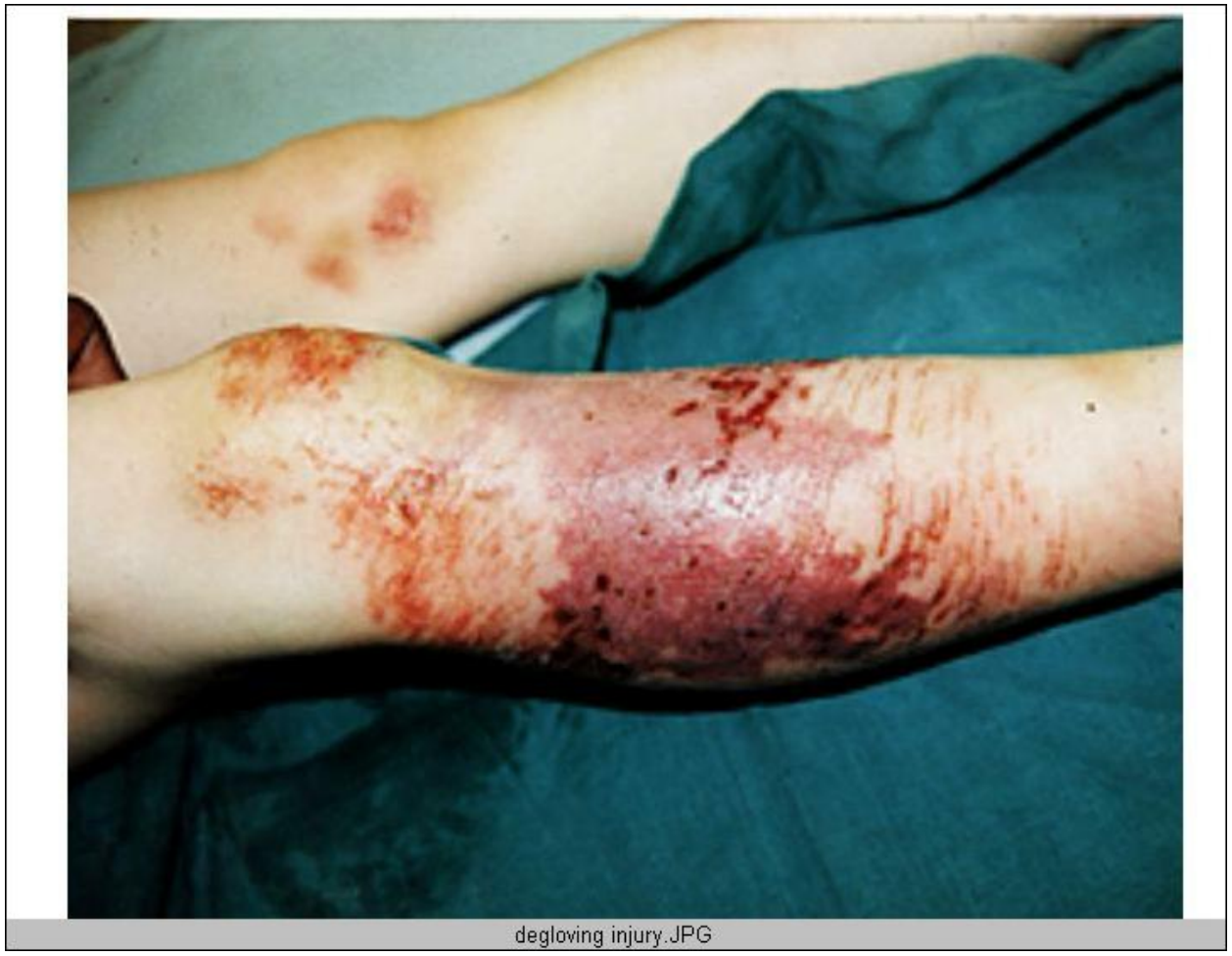
TISSUE

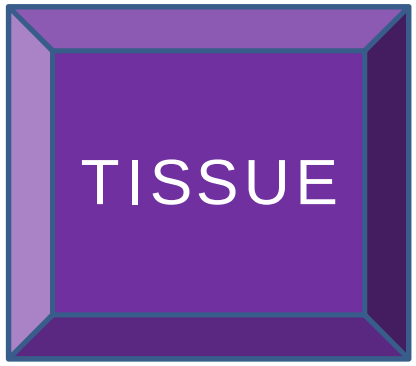
Sharp debridement and wound excision must be thorough





Beware of the closed degloving wound


degloving injury.JPG



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TISSUE

Methods of debridement of non-viable tissue:

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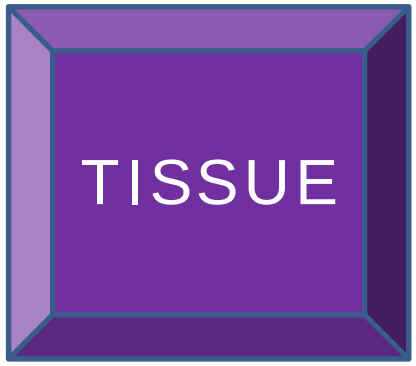
Hydrocolloids, Hydrogels, Films, Honey:

- Absorb fluid and progressively move bacteria and cellular debris away from the surface of the wound
- Facilitate tissue digestion and separation of the slough



Hypochlorite solution
Aserbine

Methods of debridement of non-viable tissue:

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TISSUE

Methods of debridement of non-viable tissue:

- surgical
- mechanical
- autolytic
- chemical
- biological
- enzymatic

Larvae of Lucilia sericata (greenbottle fly) digest necrotic tissue and pathogens

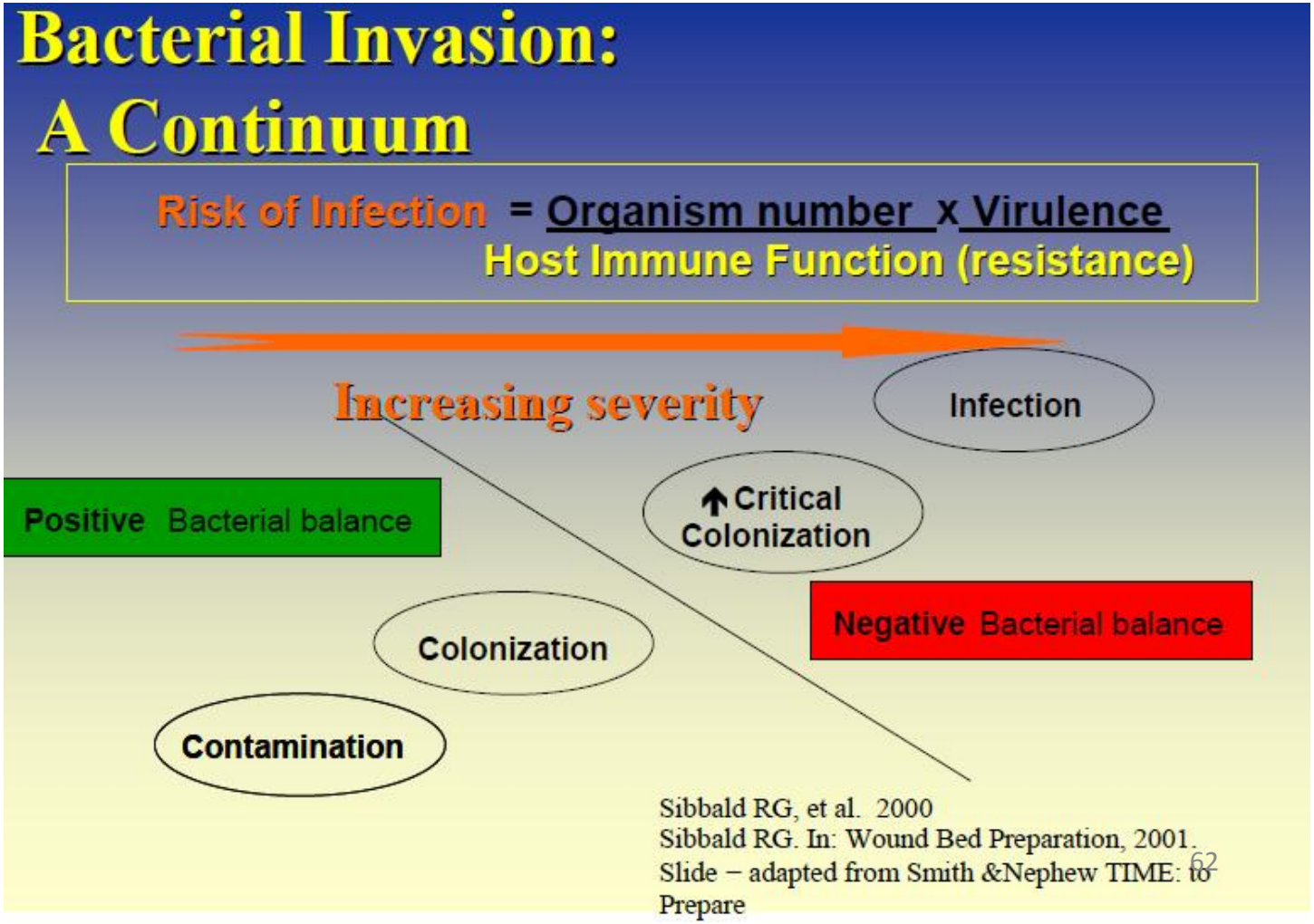
TISSUE

Methods of debridement of non-viable tissue:

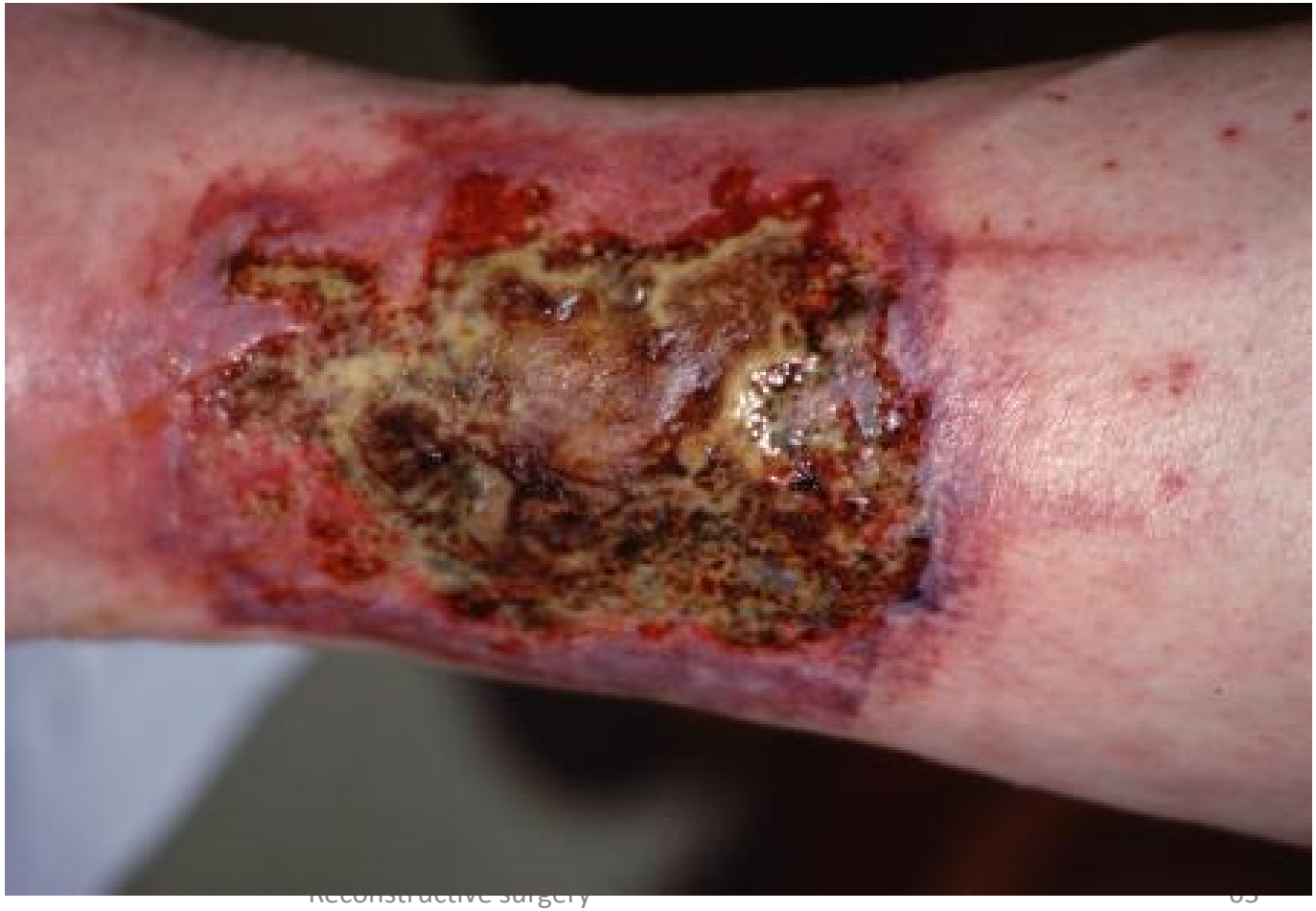
- surgical
- mechanical
- autolytic
- chemical
- biological
- enzymatic

Varidase
Iruxol

INFECTION
INFLAMMATION



INFECTION
INFLAMMATION



Topical antibiotics and antiseptics:

- Honey
- Silver sulphadiazine
- Impregnated gauze – sofratulle, bactigras,
- Inadene
- Iodine based polysaccharide bead dressings e.g. Iodosorb, Iodoflex
- Metronidazole gel,
- Mupirocin (Bactroban), fusidic acid (Fucidin)
- Nanocrystalline silver

“A moist wound healing environment enhances re-epithelialization in open wounds, but excessive wound fluid can slow down wound healing and cause maceration”

Absorbent dressings:

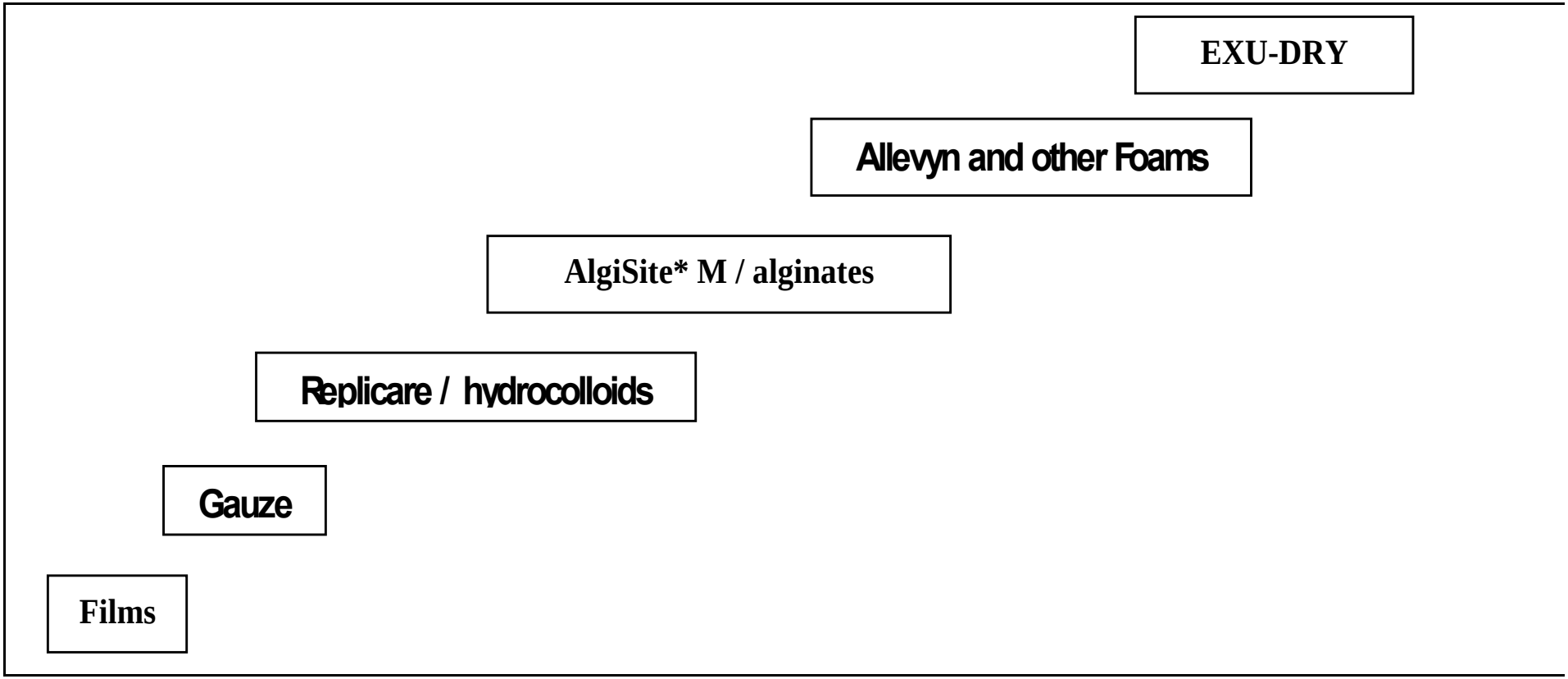
Foam
Hydrofibre
Calcium alginate
Crystalline saline
Hydropolymer foam
Acrylic dressing

Proper moisture balance produces:

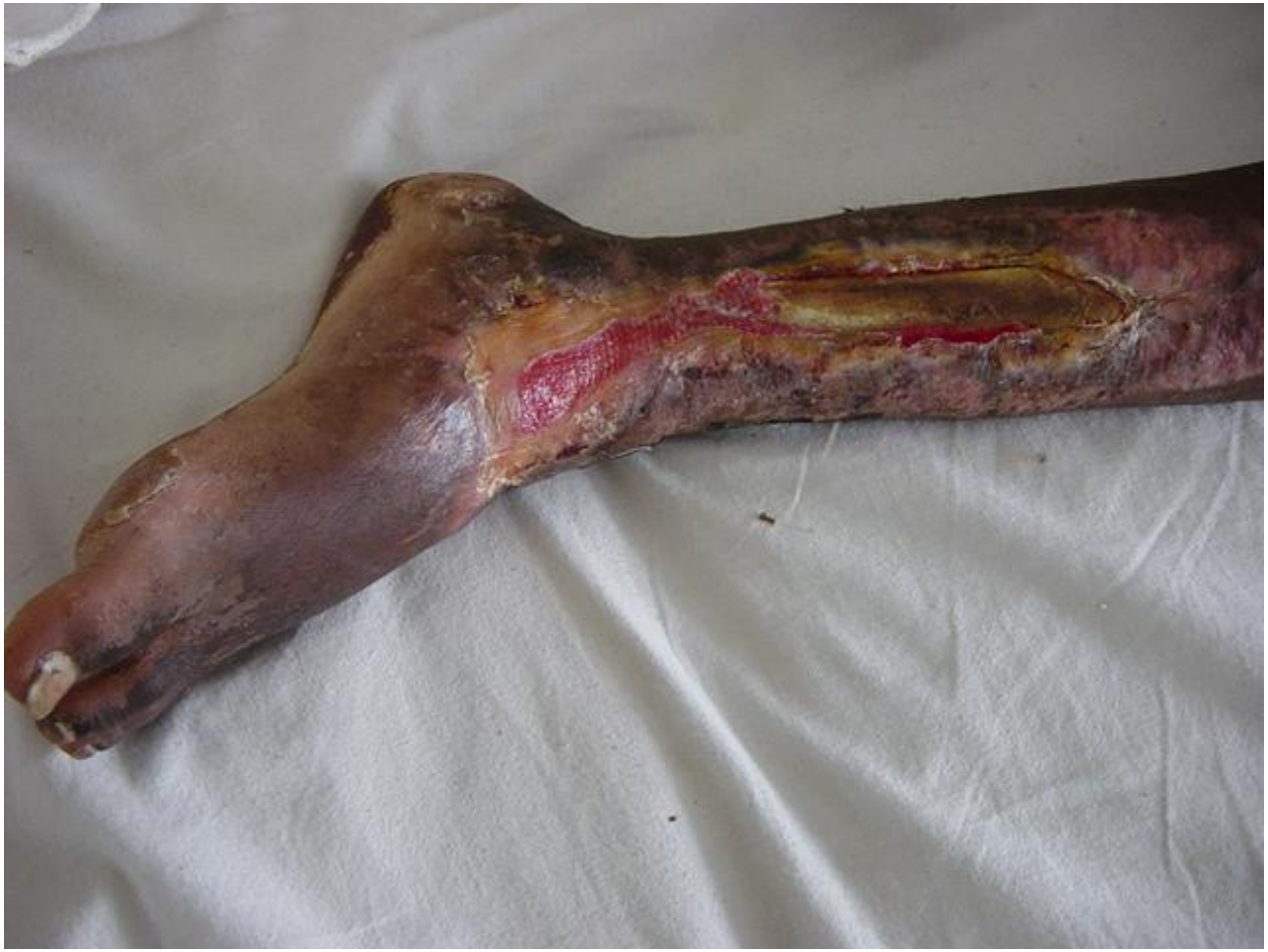
- Decreased healing time
- Decreased rate of infection
- Reduced wound trauma
- Fewer dressing changes
- Reduced pain
- Increased cost effectiveness

RELATIVE MOISTURE MANAGEMENT

Dressing	Water Absorbed (grams)
Gauze 6” x 9” 4-ply	28.0
ABD Pad 5” x 9”	111.9
EXU-DRY 6” x 9”	214.8



- 20 to 40% reduction in two and four weeks is likely to be a reliable predictor of healing
- Non-healing edge is cliff like; healing edge is sloppy and bluish tinged
- Consider removal of hyper-keratotic and fibrotic rim
- Use growth factors



WOUND MANAGEMENT PRODUCTS FOR TYPES OF WOUNDS

(page 8 of UK policy paper)

- Healing is a matter of time, but it is sometimes also a matter of opportunity.
  Read more:
  http://www.brainyquote.com/quotes/authors/h/hippocrates.html#ixzz1kM4R2jiv

- Make a habit of two things: to help; or at least to do no harm.
  Read more:
  http://www.brainyquote.com/quotes/authors/h/hippocrates.html#ixzz1kM4fNvM0

- Science is the father of knowledge, but opinion breeds ignorance.
  Read more:
  http://www.brainyquote.com/quotes/authors/h/hippocrates.html#ixzz1kM4xMqEj