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

<table>
<thead>
<tr>
<th>Acute wound</th>
<th>Chronic wound</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Trauma</td>
<td>• Poorly treated trauma</td>
</tr>
<tr>
<td>• Surgery</td>
<td>• Vascular disease</td>
</tr>
<tr>
<td>• Infection</td>
<td>• Haematological disease</td>
</tr>
<tr>
<td>• Inflammation</td>
<td>• Pressure</td>
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<tr>
<td></td>
<td>• Infection</td>
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<tr>
<td></td>
<td>• Endocrine</td>
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<tr>
<td></td>
<td>• Malignancy</td>
</tr>
</tbody>
</table>
Traumatic wounds on the face
Phases of wound healing

- haemostasis
- inflammation
- proliferation
- maturation
Towards healing

- Haemostasis
- Fibrin Platelets
- Neutrophils
- Macrophages
- Lymphocytes
- Inflammation: Proteoglycans
- Proliferation: Fibroblasts
- Collagen
- Endothelial cells
- Scar Maturation: Collagen Fibril Crosslinking
- Remodelling
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
- Epithelialisiation
- 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 = \( \text{Organism number} \times \text{Virulence} \)

Host Immune Function (resistance)

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Increasing severity

- **Critical Colonization**
- **Infection**
- **Colonization**
- **Contamination**
- **Positive Bacterial balance**
- **Negative Bacterial balance**

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Sibbald RG, et al. 2000
Slide – adapted from Smith &Nephew TIME: to Prepare
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,
Pro-inflammatory cytokines

- TNF-α
- IL-1b
- IL-2
- IL-9

Anti-inflammatory cytokines

- IL-4
- IL-10
- TGF

MMP

TIMP
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.
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

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
- Anxiety/distress/depression
- Social isolation
- Polypharmacy
- Fear of amputation/life adjustment
- Change of body image/deformity
- Pain
- Decrease in physical activity
- Work and walking concerns
- Pain
<table>
<thead>
<tr>
<th>Wound information</th>
<th>Is tetanus-prone</th>
<th>Is not tetanus prone</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time since injury</strong></td>
<td>&gt;6 hours</td>
<td>&lt;6 hours</td>
</tr>
<tr>
<td><strong>Depth of injury</strong></td>
<td>&gt; 1 cm</td>
<td>&lt; 1 cm</td>
</tr>
<tr>
<td><strong>Mechanism of injury</strong></td>
<td>Crush, burn, gunshot, frostbite, penetration through clothing</td>
<td>Sharp cut</td>
</tr>
<tr>
<td><strong>Dead tissue present</strong></td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td><strong>Foreign material (grass, dirt, etc.) contamination</strong></td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>
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

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

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

TIME

TISSUE

INFECTION INFLAMMATION

MOISTURE

EDGE OF WOUND
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%
• Cetrime 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
TISSUE

Non viable

NECROTIC TISSUE

Dehydrates, shrinks, inhibits autolysis and separation becomes delayed indefinitely

SLOUGH

Mixture of fibrin, deoxyribonucleoprotein, serous exudates, leukocytes and bacteria
Methods of debridement of non-viable tissue:

- surgical
- mechanical
- autolytic
- chemical
- biological
- enzymatic
Sharp debridement and wound excision must be thorough
Beware of the closed degloving wound
Methods of debridement of non-viable tissue:

- surgical
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- chemical
- biological
- enzymatic
Methods of debridement of non-viable tissue:

- Surgical
- Mechanical
- Autolytic
- Chemical
- Biological
- Enzymatic

**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
Methods of debridement of non-viable tissue:

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

Hypochlorite solution
Aserbine
Methods of debridement of non-viable tissue:

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

Larvae of Lucilia sericata (greenbottle fly) digest necrotic tissue and pathogens.
Methods of debridement of non-viable tissue:

- Surgical
- Mechanical
- Autolytic
- Chemical
- Biological
- Enzymatic

Varidase
Iruxol
Bacterial Invasion: A Continuum

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

Increasing severity

Infection

↑ Critical Colonization

Colonization

Contamination

Positive Bacterial balance

Negative Bacterial balance

Sibbald RG, et al. 2000
Slide – adapted from Smith &Nephew TIME: to Prepare
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

<table>
<thead>
<tr>
<th>Dressing</th>
<th>Water Absorbed (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gauze 6” x 9” 4-ply</td>
<td>28.0</td>
</tr>
<tr>
<td>ABD Pad 5” x 9”</td>
<td>111.9</td>
</tr>
<tr>
<td>EXU-DRY 6” x 9”</td>
<td>214.8</td>
</tr>
</tbody>
</table>

### Absorbency

- **Gauze**
- **Films**
- **Replicare / hydrocolloids**
- **AlgiSite* M / alginates**
- **Allevyn and other Foams**
- **EXU-DRY**
• 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