Wound healing

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INTRODUCTION

Wound healing is a mechanism whereby the body attempts to restore the integrity of the injured part.
Factors influencing healing of a wound

- Site of the wound
- Structures involved
- Mechanism of wounding
- Incision
- Crush
- Contamination (foreign bodies/bacteria)
- Loss of tissue
Other local factors
Vascular insufficiency (arterial or venous)
Previous radiation
Pressure

Systemic factors
Malnutrition or vitamin and mineral deficiencies
Disease (e.g. diabetes mellitus)
Medications (e.g. steroids)
Immune deficiencies (e.g. chemotherapy, acquired immunodeficiency syndrome (AIDS))
Smoking
NORMAL WOUND HEALING
This is variously described as taking place in three or four phases,
the most commonly agreed being:
1 the inflammatory phase;
2 the proliferative phase;
3 the remodelling phase (maturing phase).
Occasionally, a **haemostatic phase** is referred to as occurring
before the inflammatory phase, or a destructive phase
following inflammation consisting of the cellular cleansing of
the wound by macrophages.
•The inflammatory phase begins immediately after wounding and lasts 2–3 days. Bleeding is followed by vasoconstriction and thrombus formation to limit blood loss. Platelets stick to the damaged endothelial lining of vessels, releasing adenosine diphosphate (ADP), which causes thrombocytic aggregates to fill the wound.
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• When bleeding stops, the platelets then release several cytokines from their alpha granules.
• These are platelet-derived growth factor (PDGF), platelet factor IV and transforming growth factor beta (TGFβ).
• Macrophages remove devitalised tissue and microorganisms while regulating fibroblast activity in the proliferative phase of healing.
The proliferative phase lasts from the third day to the third week, consisting mainly of fibroblast activity with the production of collagen and ground substance (glycosaminoglycans and proteoglycans), the growth of new blood vessels as capillary loops (angioneogenesis) and the re-epithelialisation of the wound surface.  

*Fibroblasts require vitamin C to produce collagen.* The wound tissue formed in the early part of this phase is called granulation tissue. In the latter part of this phase, there is an increase in the tensile strength of the wound due to increased collagen, which is at first deposited in a random fashion and consists of type III collagen.
The remodelling phase is characterised by maturation of collagen (type I replacing type III until a ratio of 4:1 is achieved). There is a realignment of collagen fibres along the lines of tension, decreased wound vascularity and wound contraction due to fibroblast and myofibroblast activity.
A more historical (Latin) description of this phase is described in four words: rubor (redness), tumour (swelling), calor (heat) and dolour (pain)
ABNORMAL HEALING

• Delayed healing may result in loss of function or poor cosmetic outcome.
• The aim of treatment is to achieve healing by primary intention and so reduce the inflammatory and proliferative responses.
• Delayed primary intention healing occurs when the wound edges are not opposed immediately, which may be necessary in contaminated or untidy wounds.
• The inflammatory and proliferative phases of healing are well established when delayed closure of the wound is carried out.
• Secondary healing or healing by secondary intention occurs in wounds that are left open and allowed to heal by granulation, contraction and epithelialisation.
Classification of wound closure and healing

__Primary intention__
Wound edges opposed
Normal healing
Minimal scar

__Secondary intention__
Wound left open
Heals by granulation, contraction and epithelialisation
Increased inflammation and proliferation
Poor scar

__Tertiary intention__ (also called delayed primary intention)
Wound initially left open
Edges later opposed when healing conditions favorable.
# Types of Wounds – Tidy versus Untidy

<table>
<thead>
<tr>
<th>Tidy Wounds</th>
<th>Untidy Wounds</th>
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</thead>
<tbody>
<tr>
<td>Incised</td>
<td>Crushed or avulsed</td>
</tr>
<tr>
<td>Clean</td>
<td>Contaminated</td>
</tr>
<tr>
<td>Healthy tissues</td>
<td>Devitalized tissues</td>
</tr>
<tr>
<td>Seldom tissue loss</td>
<td>Often tissue loss</td>
</tr>
</tbody>
</table>
• Primary repair of all structures (e.g. bone, tendon, vessel and nerve) may be possible in a tidy wound, but a contaminated wound with dead tissue requires debridement on one or several occasions before definitive repair can be carried out (the concept of ‘second look’ surgery).
• Multiple debridements are often required after crushing injuries in road traffic accidents or in natural disasters such as earthquakes, where fallen masonry causes widespread muscle damage and compartment syndromes.
The surgeon must remember to examine the whole patient according to acute trauma life support (ATLS) principles:
- Cleansing
- Exploration and diagnosis
- Debridement
- Repair of structures
- Replacement of lost tissues where indicated
- Skin cover if required
- Skin closure without tension
- Tetanus cover should be noted and appropriate treatment carried out.
SOME SPECIFIC WOUNDS

Bites
Most bites involve either puncture wounds or avulsions. Small animal bites are common in children and require cleansing and treatment usually under general anaesthetic.

Puncture wounds
Needle-stick injuries should be treated according to the well-published protocols because of hepatitis and human immunodeficiency virus (HIV) risks. X-ray examination should be carried out in order to rule out retained foreign bodies in the depth of the wound.
**Haematoma**
If large, painful or causing neural deficit, a haematoma may require release by incision or aspiration.

**Degloving**
Degloving occurs when the skin and subcutaneous fat are avulsion from its underlying fascia. Examination under anaesthetic is required with a radical excision of all non-bleeding skin.
SCARS

• An *atrophic scar* is pale, flat and stretched in appearance, often appearing on the back and areas of tension.

• A *hypertrophic scar* is defined as excessive scar tissue that does not extend beyond the boundary of the original incision or wound.

• A *keloid scar* is defined as excessive scar tissue that extends beyond the boundaries of the original incision or wound.
Treatment of hypertrophic and keloid scars
Pressure – local moulds or elasticated garments
Silicone gel sheeting (mechanism unknown)
Intralesional steroid injection (triamcinolone)
Excision and steroid injections
Excision and postoperative radiation
Intralesional excision (keloids only)
Laser – to reduce redness
Vitamin E or palm oil massage (unproven)
Types of adverse scars

Wrong direction
Poor alignment of features
Stretched scar
Contracted scar
Contour deformity
Pigment alteration
Tattooing
Stitch marks