Wound healing process

The healing process occurs in stages. Wound healing may not be complete for up to **two years.** Types of healing include primary intention, delayed primary intention and secondary intention

TYPES OF HEALING

PRIMARY INTENTION HEALING

Healing with minimal granulation tissue formation and minimal scarring e.g. healing following surgical closure of a wound by suturing or stapling

DELAYED PRIMARY INTENTION HEALING

Occurs when a wound is allowed to heal open for a few days and then closed as if primarily. Such wounds are left open initially because of gross contamination

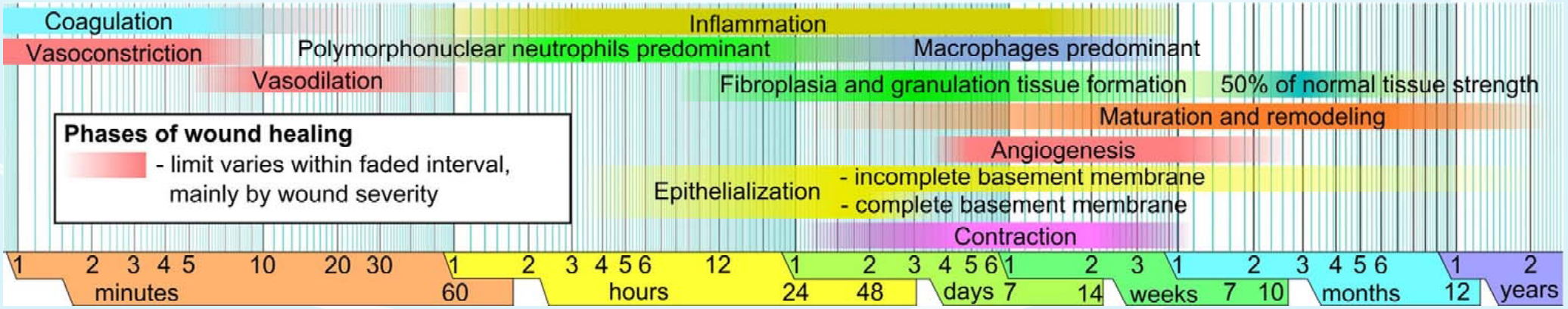
SECONDARY INTENTION HEALING

Wounds where the margins cannot be re-approximated. Healing occurs through granulation tissue, wound contraction and epithelialisation.

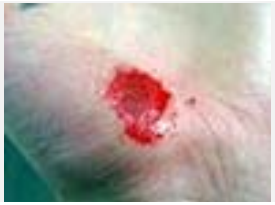
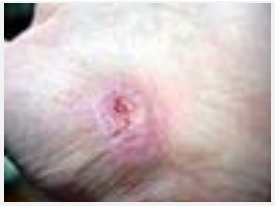
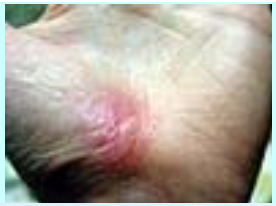
WOUND: is basically the loss of continuity of the skin. It is the visible result of external or internal damage.

HEALING: is a complex mechanism by which the body repairs damaged tissue through a series of cellular and biochemical events (a cascade). However this process is not only complex but fragile and susceptible to interruption or failure leading to the formation of chronic non-healing wounds (refer to chronic wounds guide).

Phases of wound healing



Wound healing

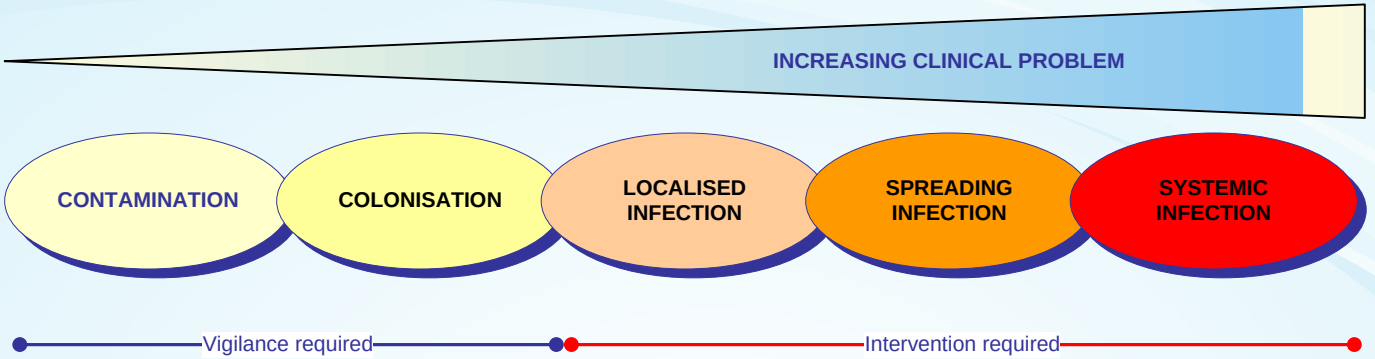
STAGE	IMAGE	PROCESS	CLINICAL EFFECTS
Immediate (8 – 10 minutes)		▪ Vasoconstriction and activation of endothelial cells, platelets and clotting cascade ▪ Biochemical response: release of prostaglandins, serotonin & clotting factors ▪ Haemostasis occurs with clot formation	▪ Haemorrhage is controlled or reduced
Inflammatory (0 – 3 days)		▪ Histamine is released ▪ Increased blood supply to the area ▪ Accumulation fluid in soft tissues ▪ Pressure is exerted on sensory nerve endings ▪ Neutrophils and macrophages begin phagocytosis	▪ Erythema ▪ Heat ▪ Oedema ▪ Discomfort ▪ Impaired function ▪ Cleansing of the wound bed and formation of crust or pus
Proliferation (3 – 24 days)		▪ New blood vessels infiltrate the wound ▪ Fibroblasts migrate to the scene and produce collagen ▪ Fibroblasts transform into either myofibroblasts or fibrocytes ▪ Myofibroblasts pull the wound edges together ▪ As new capillaries form the clot is removed ▪ Tissue appears granular (granulation tissue) ▪ Epithelial cells multiply and migrate over the surface ▪ In wounds that heal by primary intention stimulation of epithelial growth occurs concurrently with collagen synthesis ▪ In wounds healing by secondary intention epithelialisation will begin when the wound is filled with granulation tissue	▪ Red, granular and slightly uneven tissue ▪ Collagen is a basic building protein and is a major component of granulation tissue ▪ Fibroblasts require an acid environment to manufacture collagen thus Vitamin C is essential at this stage of healing ▪ The size of the defect is reduced ▪ A smooth marginal zone or islands of epithelium are seen in the wound
Maturation (up to 2 years) Collagen remodelling		▪ Collagen increases in strength ▪ There is a reduction in the number of vessels so blood flow is reduced	▪ Scar flattens and softens ▪ The scar pales and itching subsides

TIME

	Tissue	Inflammation /infection	Moisture	Edge of wound
Presentation	Necrotic tissue or slough present.	Increased exudate Increased odour Friable tissue Bridging and pocketing tissue	Moderate-high exudate ***or*** Dry wound bed	Non-advancing wound edges less than 20-40% in 2- 4weeks Reduction in size
Patho physiology	Defective matrix and cell debris impair healing. Establish vascular supply.	High bacterial count Prolonged inflammation Increased inflammatory cytokines Increased protease activity Low growth factor activity	Desiccation slows epithelial cell migration. Excessive fluid causes maceration of wound margin.	Non migrating keratinocytes. Non responsive wound cells and abnormalities in extracellular matrix or abnormal protease activity.
Aim	**Remove defective tissue**	**Remove or reduce bacterial load**	**Restore moisture balance**	**Edge of wound advancement**
Clinical action	Debridement *(once vascular supply established)* ▪Autolytic ▪Sharp surgical ▪Enzymatic ▪Mechanical ▪Biological	Remove infected foci Topical/systemic ▪Antimicrobials ▪Anti-inflammatory ▪Protease inhibitors	Apply moisture balancing dressing. Compression therapy. Negative pressure therapy.	Re-assess cause, consider corrective therapies: ▪Debridement ▪Skin grafts ▪Biological agents ▪Adjunctive therapies
Suggested products	•Hydrogel •Hydrocolloid •Cadexomer iodine	•Silver •Honey •Cadexomer iodine •Chlorhexidine •PHMB	▪ Hydrogel ▪ Hydrocolloid ▪ Alginate ▪ Hydrofibre ▪ Hydrofoam ▪ Compression bandaging ▪ Negative pressure therapy	•Measuring devices •Photographic devices
Clinical outcomes	Restoration of wound base and functional extracellular matrix proteins. Viable wound bed.	Low bacterial count or controlled inflammation. Decreased inflammatory cytokines. Decreased protease activity. Increased growth factor.	Restore epithelial cell migration. Oedema and excessive fluid controlled. Maceration avoided.	Migrating keratinocytes. Responsive wound cells. Restoration of appropriate protease profile.

Wound assessment - infection

BACTERIA are inevitably present in most wounds, often without detrimental effect. The presence of bacteria in a wound may result in: **CONTAMINATION** – the bacteria do not increase in number or cause clinical problems. **COLONISATION** – the bacteria multiply but wound tissues are not damaged. **INFECTION** – the bacteria multiply, healing is disrupted and wound tissues are damaged (local infection – the term has also been used to describe local infection). Bacteria may produce problems nearby (spreading infection) or cause systematic illness (systematic infection) (WUWHS 2008).



ACUTE WOUNDS

Localised infection:

- New or increased pain
- Erythema
- Local warmth
- Swelling
- Purulent discharge
- Pyrexia 5-7 days post surgery
- Delayed or stalled healing
- Abscess
- malodour

Spreading infection:

As for localised infection PLUS:

- **Erythema extending >2 cm**
- **Lymphangitis**
- **Crepitus in soft tissue**
- **Wound breakdown / dehiscence**

NOTES:
Failed grafts and full thickness burns – pain is not always present
Deep wounds – induration, extension of wound, higher WBC unexplained, signs of sepsis
Immunocompromised – signs and symptoms may be modified and less obvious

CHRONIC WOUNDS

Localised infection:

As for **acute** PLUS

- Peri-wound oedema
- Bleeding or friable tissue
- Discolouration of wound bed
- Pocketing: smooth non granulating areas in base of wound
- Bridging: infection may result in incomplete wound epithelialisation with strands/patches tissue forming “bridges” across the wound. Can occur in both acute/chronic healing by secondary intention

Spreading infection:

As for **acute** PLUS

Malais or other non specific deterioration in patient’s general condition

NOTES:
Immunocompromised/motor sensory neuropathies – signs and symptoms may be modified and less obvious e.g. diabetic foot
Arterial – previously dry ulcers may become wet when infeciton present

SIGNS OF SYSTEMIC INFECTION

Sepsis

Clinical infection with pyrexia or hypothermia, tachycardia, tachypnoea, raised or depressed WBC count

Severe sepsis

As above PLUS multiple organ dysfunction

Septic shock

As above PLUS hypotension despite adequate volume resuscitation

NOTES:
Other sites of infection should be excluded before assuming that systemic infection is related to the wound

World Union of Wound Healing Societies (WUWHS) 2008. Principles of best practice: Wound Infection in clinical practice. A consensus document, London: MEP Ltd

Wound infections - investigations

The diagnosis of wound infection is based mainly on clinical judgement – appropriate investigations e.g. microbiology of wound samples, can support and guide management.

Microbiology

Wound swabbing

Levine technique is internationally recognised as the most useful:

After cleansing the wound moisten swab tip with culture medium or normal saline then swab wound over clean exposed granulation tissue covering 1 cm^2 . Apply sufficient pressure to express fluid from within the wound tissue

Needle aspiration

Cellulitic areas can be sampled when use of wound swabs is not possible e.g. in the presence of adhered slough

Wound biopsy

Provides the most accurate information for type and quantity of pathogenic bacteria

Effective management of wound infection

Optimise host response

Manage co-morbidities e.g. glycaemic control, enhance tissue perfusion/oxygenation:

- Minimise or elimination risk factors for infection where feasible
- Optimise nutritional status and hydration
- Seek and treat other sites of infection e.g. UTI

Reduce bacterial load

- Prevent further wound contamination e.g. use of infection control procedures, protect wound with appropriate dressing
- Facilitate wound drainage as appropriate
- Optimise wound bed e.g. TIME principles
- Antimicrobial therapy – antimicrobial dressing +/- systemic antibiotics

General measures

- Manage any systemic symptoms e.g. pain, pyrexia
- Provide patient and carer education
- Optimise patient concurrence with management plan
- Ensure psychosocial support

NOTE:

- **Infection control procedures should be followed to prevent further contamination of the wound and cross-contamination**
- **Good hygiene practice includes paying particular attentionto thorough hand cleansing, suitable protective working clothes including gloves**

EVALUATION – initial and ongoing evaluation to include:

- **Systematic monitoring and recording**
- **Serial clinical photographs**
- **Tracing or tracking / wound mapping**
- **Assessment tool for antibacterial barrier dressings**

Reference: Levine Technique.