WOUND CARE FOR MEDICAL STUDENTS

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1. **INTRODUCTION**

    The skin is the largest organ of the human body. Its function is to protect the internal milieu of the body from external forces that may injure and kill or destroy the organism. Any break in this protective organ is called a wound.

Figure 1: The wounded man

    Any acute wound will bleed, and control of the bleeding is the first priority in the management of any injury. All sorts of potions, herbs, plants and other methods have been used to cover wounds to stop bleeding and promote healing. The most important factor in the healing process is to prevent infection. If the wound is clean and kept clean, it will heal by primary intention. If the wound gets infected, it will be converted from an acute wound to a chronic wound. In the case of a chronic wound the healing process will change from healing by primary intention to secondary intention.

2. **HEALING OF WOUNDS**

    A fresh wound which is not contaminated or infected will heal by primary intention. The wound is cleaned, and the edges approximated with suture material to make the defect as small as possible without tension. Healing will then proceed over the next few days, and the suture material can usually be removed after 7 to 10 days when the adhesion of the wound is strong enough. The healing process takes much longer and a wound can only be considered to be completely healed after 6 to 9 months when the inflammatory and modulating processes have stabilised.

This process usually leaves a small, neat scar.

When a wound is closed with too much tension, it leads to necrosis of the tissue, or if it gets infected, healing will be delayed, and then it is known as healing by secondary intention. The wound in such a case first needs to be cleaned and the infection needs to be controlled before healing can take place. The process takes much longer and usually leaves a large scar when healed.

Wound healing takes place in phases but there is considerable overlapping of the phases. The following phases can be recognised:

a). The haemostatic phase, where bleeding is controlled by vasospasm and clot formation.

b). The inflammatory phase, with the release of inflammatory mediators and the development of the classic signs of rubor (redness), calor (warmth), dolor (pain), tumor (swelling) and loss of function.

c). The destructive phase, with the removal of injured and dead tissue from the injured site.

d). The proliferative phase, with neo-vascularisation and ingrowth of cells and collagen needed for healing.

e). Maturation of the scar.

From a practical point of view, only two phases of the healing process will be discussed, namely inflammation and repair.

3. THE PHYSIOLOGY OF WOUND HEALING: PART 1 – INFLAMMATION

The physiology of wound healing forms the basis on which wound care is based. A wound is a pathological state in which tissue becomes separated or destroyed. Wound healing is the complex sequence of events directed towards closure of this defect, usually by replacement with scar-forming connective tissue. The process of repair by replacement with scar tissue contrasts with the process of regeneration, where damaged tissue is replaced with identical cells. The many changes that have developed in this field in the past decade are the result of a greater understanding of the events occurring within minutes of wounding. Many experts believe that efforts aimed at improving the speed of wound healing, the nature of the healing process and even the eventual scar outcome, need to be concentrated on events that begin almost instantly when injury occurs. Wound healing is aimed at reversing the loss of structural integrity caused by injury to the tissue. This process of wound healing can be divided into a number of dynamic, overlapping phases:

a) The initial response is **vascular**. To prevent localised haemorrhage, the coagulation cascade is initiated. The damaged ends of the blood vessels immediately constrict. Platelets in the blood escaping from the injured vessels release thromboxane A2 to slow the loss of blood. During vasoconstriction, vessels turn inward and narrow. This vasoconstriction lasts only a few minutes, long enough for blood clots to seal the

leaking blood vessel. The coagulation cascade starts with the release of platelet factors (thromboplastins) and other substances from the damaged cells. The extrinsic and intrinsic pathways of the coagulation cascade are initiated, thromboplastins activate conversion of prothrombin to thrombin, and thrombin converts soluble fibrinogen into insoluble fibrin. Eventually an aggregate of fibrin, red blood cells and platelets grows large enough to plug the capillary and stop the flow of blood. As the clot dries it forms a scab, which protects the wound site from dehydration and pathogen invasion. Concurrently vasodilatation ensues – 10 to 30 minutes after injury, mast cells in connective tissue release serotonin and histamine, causing vessels to dilate and increase their permeability. This increased blood flow causes heat release and a temperature rise in the skin around the wound. The increased permeability results from separation of endothelial cells in the vessel walls induced by serotonin and histamine. Thus plasma migrates into the interstitial space, nourishing the wounded tissue and leukocytes leak into the extracellular spaces surrounding the wound.

b)   Tissue damage and the activation of clotting factors during the vascular phase stimulate the **release of inflammatory mediators** – bioactive substances – constituting the early aspect of wound healing known as the inflammatory phase. This reaction, which begins within seconds of wounding, is the same whether the cause is a surgical cut or a wound invaded by pathogenic bacteria. The qualitative nature and duration of this phase is critical in determining the eventual outcome of wound healing, from the successful closure of the defect to the quality of the resultant scar. The response occurs rapidly and can be detected by the presence of localised heat, swelling, erythema and discomfort, which usually restrict function. During this process, permeability of the intravascular space results in leakage of plasma and soluble components, and cellular constituents arriving in the following sequence: first platelets, then neutrophils, followed by monocytes and lymphocytes which differentiate into macrophages as they enter the connective tissue. The migration of epithelial cells then begins, resurfacing in the injured tissue. The macrophage is the key player in the degradation of injured tissue debris and in the reparative phase of wound healing, initiating the transition from initial inflammation to the early repair phase of wound healing.

c)   The inflammatory phase of wound healing is a complex, dynamic interaction of cellular proliferation, differentiation and specialised cytokine-induced changes resulting in control of bleeding, wound debridement and extracellular matrix preparation, setting the groundwork for the repair phase of wound healing and the ultimate closure of the defect. Interruption of any of these many intricate cellular interactions will result in changes varying from delayed or non-healing of the wound, to exaggerated scarring.

4.   **PHYSIOLOGY OF WOUND HEALING: PART 2 – REPAIR**

The inflammatory phase of wound repair prepares the groundwork for the formation of granulation tissue (the name is derived from the granular appearance of the newly forming blood vessels) and consists of a loose matrix of fibrin, fibronectin, collagen, and glycosaminoglycans (especially hyaluronic acid) containing macrophages, fibroblasts and developing blood vessels. This tissue formation occurs between 4 and 21 days following wounding and serves as a scaffolding for new tissue ingrowth

in deep wounds. Wound closure by contraction, the inward movement of the wound edges of the injured tissue, is a normal part of the healing process. Contraction begins 8 – 10 days following injury.

Figure 2: Macrophage-orchestrated inflammatory response

Fibroblasts and the extracellular matrix (ECM) orchestrate with fibroblasts applying tension to the surrounding matrix. Fibroblasts align themselves along the axis of the wound and form cell-to-cell links, which contribute to contraction of the wound. Production of collagen remains a major process in wound repair several weeks after wound closure, and the collagen continues to undergo remodelling for 2 years or more, until stability of the process occurs. Precise regulation of collagen metabolism during the repair process is exerted by cytokines and by the interaction of the ECM with fibroblasts. Collagen synthesis is maximal between 14 and 21 days. After 21 days the rate of synthesis and the volume density of collagen in the wound return to normal levels. However, the tensile strength of the tissue continues to increase for a considerable time, up to 60 days or even one year.

In the adult, the normal repair of wounds occurs by the formation of granulation tissue and its organisation to a scar. Scar is a dynamic, metabolically active tissue and tends to remain weaker than unwounded tissue. A scar tends to contract abnormally, and over-healing may lead to a hypertrophic scar or keloid. The scar appears reddish at first but as the connective tissue grows tauter and vascularisation slows, it gradually loses colour. Hair, sebaceous and sweat glands are also absent, as is the ridged pattern of the epidermis. Thus the new skin's appearance is unusually smooth. The rate at which wounds gain tensile strength is slow. For

example, wounds have gained only about 20% of their final strength by the third week. Wounded tissue fails to attain the same breaking strength as uninjured skin. At maximum strength a scar is only 70% as strong as intact skin.

5. WOUND CLASSIFICATION

Wounds are divided into acute and chronic. Acute wounds are freshly created, usually bleed initially and are not infected. They can be contaminated with organisms from the environment, but if managed appropriately, can heal with primary intention. Examples are surgical wounds, trauma and injuries. Chronic wounds are formed when something has interfered with the natural healing process as described above. This is mostly infection, which causes delayed wound healing.

6. ACUTE WOUNDS

Wounds may firstly be classified according to the mechanism of injury and secondly according to the amount of contamination (American Association for the Surgery of Trauma, and the US Center for Disease Control (CDC).

a) Mechanism of injury

i) Laceration, sharp penetration, surgical incisions.

A clean wound, mostly superficial with minimal tissue damage and contamination. It requires suturing and will heal by primary intention.

ii) Abrasion.

Involves the skin surface to varying depths, caused by friction which creates heat (burn) necrosis, and if contaminants (tar, soil) enter they may cause infection. Effective treatment requires vigorous cleaning and scrubbing, copious lavage and removal of tattooing by dermabrasion mostly under general anaesthesia. Occlusive dressings promote moist wound healing through secondary intention.

iii) Crushing, avulsion, degloving.

These wounds are often hidden. The appearance is deceptive and of a serious nature. Often present are underlying fractures and large spaces filled with blood. The potential for life-threatening infection is real. These wounds require careful diagnosis and sometimes complicated treatment, for example replantation.

iv) High-energy transfer/high-velocity wounds.

Motor vehicle accident (MVA) and missile injuries cause widespread devascularised tissue damage. The principles of treatment include multiple debridements, measures to prevent infection, fasciotomies, reconstruction and rehabilitation.

v) Burns.

Application of heat (or cold) to the skin will create a lesion called a burn. The depth will be determined by the intensity of the heat and the duration of exposure. Management consists of fluid resuscitation to replace the losses and protection of the injury to allow healing (if partial thickness) or skin grafting (if full thickness).

vi) Bites.

All bites are contaminated by micro-organisms living in the mouths of humans and animals. Besides the visible puncture wounds, there is a crushing-type injury beneath the skin that also needs management. Blood loss is a major cause of death in bites by wild animals and sharks.

b) **Degree of Contamination**

Table 1: Classification of acute wounds (CDC 1985)

Classification	Description	Procedure types
Clean	Surgical wound, no infection, no opening of respiratory, GIT, urinary tract. Closed primarily	Thyroidectomy, neck dissection, mastectomy, joint replacement, vascular surgery
Clean/contaminated	Respiratory, GIT, urinary tract opened, no contamination	Bronchoscopy, cholecystectomy, appendectomy, small bowel resection, TUR prostate, Whipple
Contaminated	Open fresh wounds. Break in sterile technique, spillage, inflammation	Appendectomy for inflamed appendix, cholecystectomy with bile spillage, diverticulitis
Dirty	Old wounds, devitalised tissue, open viscera, infection, organisms present	I and D abscess, myringotomy for otitis media, perforated bowel, peritonitis

The classification in Table 1 allows one to determine whether the patient may need antibiotic treatment as part of the surgical management. Clean wounds do not need antibiotics, while contaminated wounds need a single dose of prophylactic antibiotic given just before surgery. Depending on the degree of

contamination, a drain may have to be left in the wound to guard against wound infection. Patients with dirty wounds will need therapeutic antibiotic treatment from before till after the surgery, and the wound needs to be left open (i.e. unsutured) for the infection to be able to drain out.

c) **AAST adaptation for the management of fresh wounds**

i)   Clean wounds – incision, clean instrument

ii) Clean contaminated – domestic injury

iii) Grossly contaminated – agricultural injury

iv) Infected – contamination may happen primarily or secondarily due to inadequate or inappropriate treatment. Organisms flourish, causing suppuration.

d) **War wounds**

War wounds are a special group with specific guidelines to manage them correctly. These are mostly high-energy transfer wounds, and are severely contaminated. They should be managed as dirty wounds and treated accordingly. This implies therapeutic antibiotics and proper debridement in the operating theatre under general anaesthesia. All dead and contaminated tissue is removed, the wound is thoroughly rinsed, packed with swabs, and the skin left unsutured. It is covered with bulky dressings to absorb the wound fluid and serum, and should be left undisturbed until wound inspection after 3 - 5 days. This is done under general anaesthesia in the operating theatre, where all dressings are removed, and further debridement is done if necessary. If the wound is clean, does not smell and no pus is visible, the wound may be closed with delayed primary suturing, or a split thickness skin graft may be done if the defect is too large for suturing.

With the development of personal body protection (body armour or bullet-proof vests), soldiers often survive attacks but with more severe and devastating extremity injuries. Traumatic amputations are common, especially with landmine injuries to the lower extremities and improvised explosive devices (IEDs) injuring security personnel in mine-protected vehicles. All these injuries are managed by control of haemorrhage and management in a sterile environment (operating theatre) according to the principles mentioned above.

The International Committee of the Red Cross (ICRC) has vast experience of managing war wounds due to their humanitarian efforts with displaced persons in war zones. They use a classification system to grade the severity of wounds to enable effective management and compare outcomes. This is a practical way to grade the severity of injuries, and it also helps with the recording of these injuries in the clinical notes.

Table 2: ICRC grading of war wounds		
SYMBOL	DESCRIPTION	EXPLANATION AND SCORING
E	Entry wound (cm)	
X	Exit wound (cm)	(0 if none)
C	Cavity	Can it take 2 fingers? No=C0, yes=C1
F	Fractures	No=F0, simple = F1, comminuted =F2
V	Vital structure	Are vital structures injured? No=V0, VN=neurological, VT=thoracic, VA=abdomen, VH=haemorrhage from major vessel
M	Metallic body	Are bullets or fragments visible on X-ray? M0=none, M1=yes one, M2=multiple

Giannou, C., Baldan, M. ICRC War Surgery. ICRC 2009
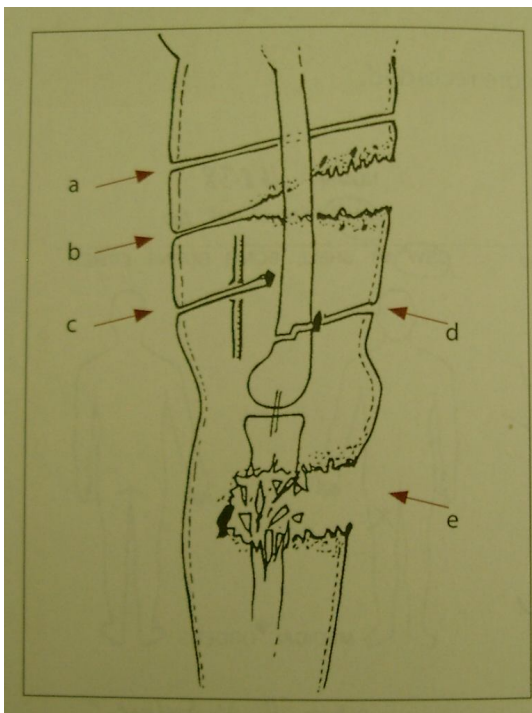
Figure 3: Diagram of different types of penetrating injuries

a) Perforating low-energy wound.
b) Perforating high-energy wound.
c) Penetrating wound with vascular injury, metallic fragment visible.
d) Penetrating low-energy injury with simple fracture of the femur, bullet visible.
e) Penetrating high-energy injury with large entrance wound, comminuted fracture of the tibia, metallic fragments visible.

7. CHRONIC WOUNDS

Acute wounds heal timely in an orderly fashion responsive to standard therapy. By contrast, the chronic healing process follows a delayed, incomplete, uncoordinated course. The distinction between acute and chronic relies on the timeliness of healing, but is arbitrary and varies with site, cause, age and physical condition. Although they are part of a heterogeneous group, venous ulcers, diabetic ulcers and pressure sores constitute 70% of cases.

a) Classification according to cause

i) Vascular insufficiency:
Poor arterial supply (atherosclerosis)
Poor venous drainage (venous ulcers)
Lymphoedema

ii) Traumatic:
Pressure ulcers (decubitus and neuropathic)
Burns, frostbite
Bites (insects, ticks, spiders, snakes)
Radiation injury

iii) Metabolic:
Diabetes
Gout
Calcinosis
Gaucher's disease

iv) Inflammatory disorders:
Pyoderma gangrenosum
Vasculitis – polyartheritis nodosa, granulomatoses
Panniculitis – necrobiosis lipoidica diabeticorum

v) Infections:
Bacterial – acute and chronic, TB, osteomyelitis
Other – fungal, treponemal, viral, protozoal

vi) Connective tissue disease:
SLE, rheumatoid arthritis, scleroderma, sclerosis
Sjögren's syndrome

vii) Haematologic:
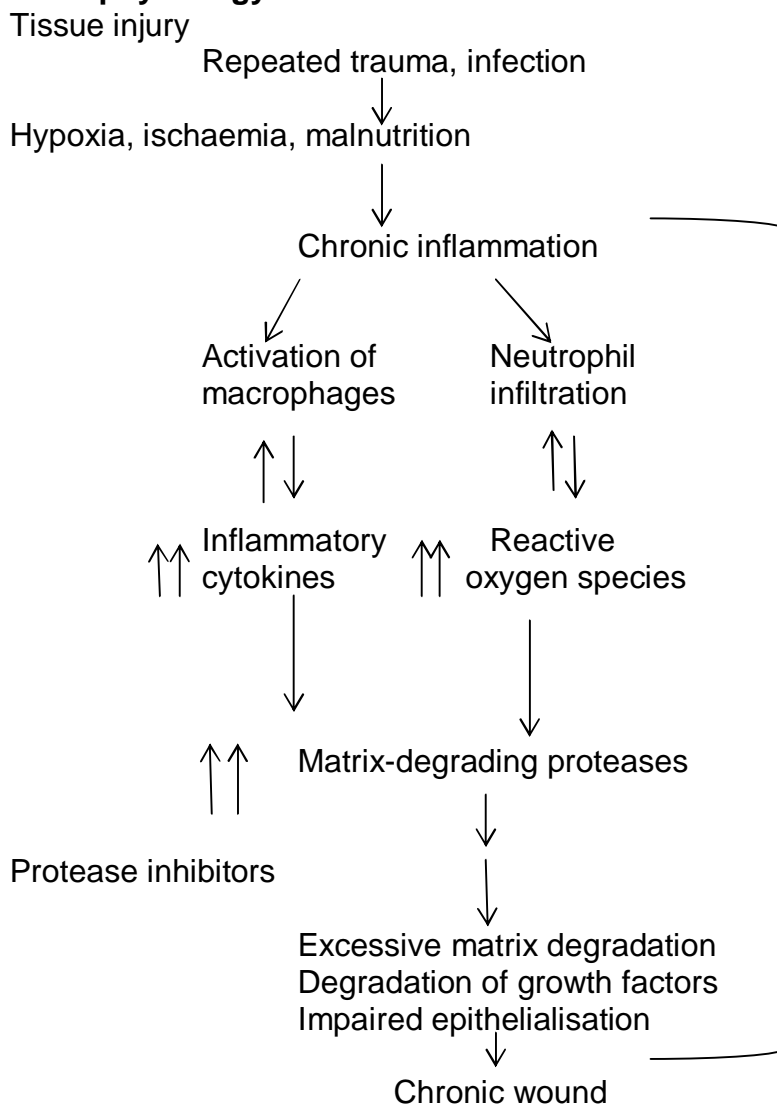RBC (sickle cell anaemia, thallassaemia, spherocytosis, polycythemiavera)

WBC (leukaemia)
Platelets (thrombocytosis, hypercoagulable states)
Protein C deficiency
Dysproteinaemia (amyloidosis, cryoglobulinemia)

viii)   Neoplastic:
Marjolin's ulcer
Primary and metastatic skin tumours
Lymphomas, sarcomas (Kaposi)
Haemangiomas, vascular and lymphatic malformations

ix)   Miscellaneous:
Drugs (Coumadin, steroids, aspirin, colchicines, NSAIDs, retinoids)
Fictitious (malingering)

x)   Local factors:
Foreign bodies, dead tissue, toxins, smoking.

b)   Pathophysiology
Tissue injury

Repeated trauma, infection

↓

Hypoxia, ischaemia, malnutrition

↓

Chronic inflammation

↙   ↘

Activation of   Neutrophil
macrophages   infiltration

↑↓   ↑↓

↑↑ Inflammatory   ↑↑ Reactive
   cytokines   oxygen species

↓   ↓

↑↑ Matrix-degrading proteases

↓

Protease inhibitors

↓

Excessive matrix degradation
Degradation of growth factors
Impaired epithelialisation

↓

Chronic wound

(From Nwomeh et al. 1998. Clin. Plast. Surg. 25:3, July)

c)   **Classification according to appearance**

There are different classifications for chronic wounds. The simplest one is used to assess a new chronic wound when first seen by the health care practitioner.
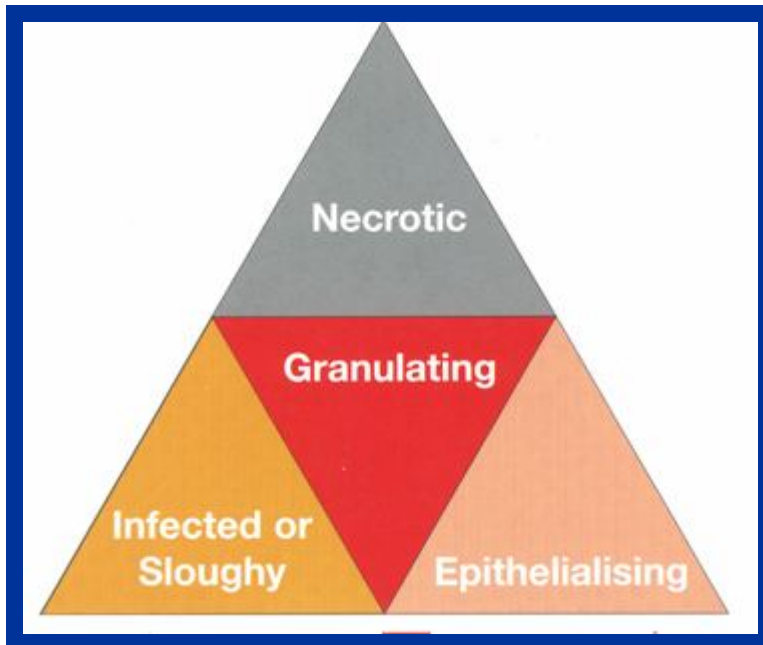
Figure 4: Classification of chronic wounds

According to this model wounds are classified into four groups, namely black or grey (necrotic tissue), yellow (infected or with slough), red (granulating) and pink (epithelialising and healing). The aim of wound care is to move away from the black and yellow towards the red and pink colour to achieve healing of the wound. Other models add a green colour for infection which also needs to be addressed.

Another model addresses the management of the different wounds. It is called the TIME OVS model.

T: Tissue viability. Dead tissue needs to be removed by debridement.
I: Infection. Bacterial load needs to be reduced by debridement, dressings, antibiotic treatment.
M: Moisture control. Excess fluid needs to be removed with appropriate dressings.
E: Edge of the wound. Dry or macerated by too much fluid.
O: Oedema. Needs to be controlled by compression and elevation.
V: Vascular blood supply. Assessment of patient and arterial supply to wounded area.
S: Skin surrounding wound. Signs of inflammation, oedema, infection, maceration, etc.

d) Wound assessment

i) A full assessment of a problem wound is critical in successful management and treatment, and facilitates the choice of wound dressing required to promote healing. This includes the history of the wound, the location, the condition of the surrounding skin and wound margins, and the extent, depth and condition of the wound bed.

ii) Physiological assessment is also essential. This includes the general health status of the patient, associated systemic and vascular disease, neurosensory and neuromuscular status, presence of foreign material, severity of the wound, and possible factors affecting treatment options.

iii) Acute and chronic wounds. Acute wounds usually occur without an underlying cause, usually trauma related. They are of short duration, eliciting a normal inflammatory response, usually followed by healing without subsequent breakdown. Chronic wounds usually result from underlying pathology, often vascular in origin. They are of prolonged duration with a delayed and ongoing inflammatory response, and liable to recurrence. Systemic factors encouraging chronicity of wounds include varied disorders, the commonest of which are the circulatory disorders. Other significant disorders that need to be excluded include respiratory disorders, malabsorption syndromes (Crohn's disease, etc.), and disorders of mobility and sensation (spinal injuries, diabetes, MS, etc.). Identifying local wound infection or systemic infection is of paramount importance.

iv) Wound assessment chart

This local assessment includes the following:

(1) Wound edge – viable, rolled, fibrotic or flat and closed (Rolled edges could be an important clue to biopsy the edge to exclude malignancy).

(2) Wound colour

- red (granulating, protect it)
- yellow (sloughy, cleanse it)
- black (necrotic, debride it)
- pink (epithelialising, protect it)

(3) Skin condition – macerated, intact, denuded, reddened.

(4) Peri-wound colour – normal, white, bright red, dark red/purple, black, light red or pink.

(5) Oedema – mild, moderate, severe.

(6) Size – length, width, depth. In diabetic wounds it is extremely important to assess whether the wound can be probed down to bone, signifying more serious

pathology and more aggressive management. Wound measurement may be done by ruler, tracing the surface on dressings, particularly grid dressings (Flexi grid S&N, etc.), or with probes or fingered gloves to document the depth of the wound.

(7)   Photographic documentation (especially digital) is being used more commonly, with its added advantage of transmission via computer, when necessary, for discussion.

(8)   Undermining – extent, range.

(9)   Sinus tract – extent, direction.

(10)   Drainage – absent, minimal, moderate, high.

(11)   Drainage type – serous, serosanguinous, purulent.

(12)   Odour – pungent, foul, faecal, musty.

(13)   Necrotic tissue – white macerated thick; yellow slough; black/brown Eschar.

(14)   Granulation tissue – red, grainy, friable.

(15)   Epithelial tissue – pink.

(16)   Pain – burning, boring, throbbing, dull, intermittent, constant.

(17)   Doppler waveform, where applicable.

Apart from these local descriptions of the wound, a fully documented medical history is noted, including allergies and sensitivities, previous treatments and questions related to the factors discussed above.

8   WOUND MANAGEMENT

The objectives of wound care are to remove the barriers to healing. This will include the following:

a)   Remove dead, necrotic, infected tissue, slough and pus. This is called debridement, which means cutting away the offending tissue.

b)   Control infection, with appropriate dressings, local antiseptics, and sometimes antibiotics.

c)   Control moisture. This may include adding moisture to a dry necrotic wound with some hydrogel ointments or absorbent dressings to remove excess fluid and pus.

d)   Prepare wound bed with the appropriate dressings for either healing by secondary intention, or skin grafting to accelerate healing.

e).   Improve blood supply and venous drainage if possible.

f).   Control oedema of the extremity, as this will impair healing.

g).   Control systemic diseases, such as diabetes, hypertension, etc.

9.   WOUND DEBRIDEMENT

There are different ways in which unwanted tissue can be removed from a wound. The most effective way is to take the patient to theatre and surgically remove it under anaesthesia with a scalpel and scissors. This also allows control of the bleeding with electrocautery. If a patient cannot be taken to the operating theatre, there are other ways to manage this problem. Rinsing with water or saline can be used, and rinsing with a handheld shower is also very effective for removing non-adherent slough from a wound. The VERSAJET™ is a mechanical instrument that uses a water current to remove non-adherent tissue from the wound bed, with very little bleeding.
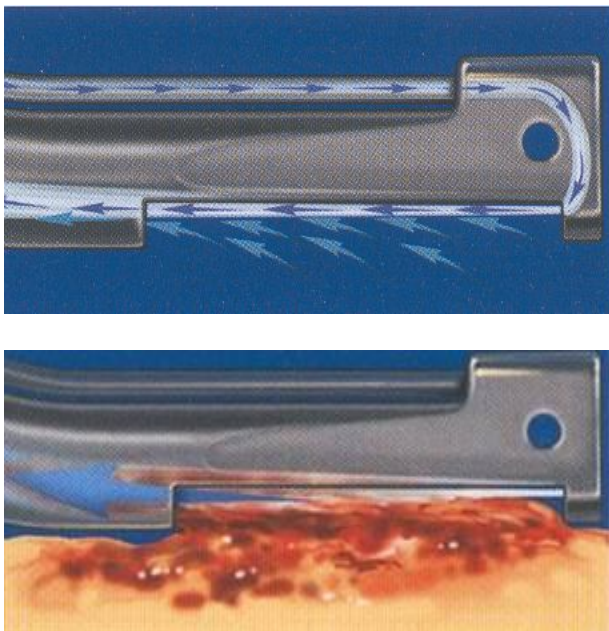
Figure 5: The VERSAJET™ method of action

If the area is small and the slough superficial, a chemical debridement can be done with ointments (such as Iruxol or Ascerbine) to liquefy the material and remove it with the dressings. Some institutions have access to maggots which do a very effective debridement of adherent dead tissue under controlled conditions.

Figure 6: A septic amputation wound with slough, before maggot debridement therapy

10.   **INFECTION AND COLONISATION**

All wounds are colonised with micro-organisms, and when a swab is taken of the wound for culture, it will grow organisms. That does not mean that there is infection in the wound. Infection occurs when the load of organisms becomes too much for the body to manage, usually more than 10^6 per gram of tissue. This phase of colonisation has been named the critical colonisation phase, where the volume of organisms causes local effects without the signs of infection, like delaying wound healing, and suppression of the inflammatory and immunological responses of the body. When the quantity of organisms exceeds 10^6 per gram of tissue, or the virulence changes, or a synergism develops between different organisms, invasion of tissue takes place and infection develops, with the classical clinical signs of tumor (swelling), calor (temperature), dolor (pain), and rubor (redness). These signs will initially be present at the wound edge and the surrounding skin, but may spread and cause systemic infection and sepsis. Usually the exudate (fluid) from the wound also increases in volume and may become offensive smelling. Other signs of infection are pain, pus formation, wound breakdown, oedema and erythema.

Figure 7: A mastectomy wound with surrounding cellulitis

In the management of wound infection, a swab is taken and sent to the laboratory for a wound culture. New dressings will have to be applied, and some antimicrobial dressings may be considered (see below). A course of systemic antibiotics may be necessary if there are signs of systemic infection. The choice of antibiotic will initially have to be empirical, and can be adjusted as soon as the microbiology report becomes available. The infection will mostly be caused by a Gram-positive organism (*Streptococcus* or *Staphylococcus*) that may be sensitive to penicillin, so our first-line treatment is with oral penicillin. If the infection develops in hospital the probability is large that the infection will be caused by a resistant *Staphylococcus* (MRSA) and a penicillinase-resistant drug will have to be used (such as Cloxacillin, Amoxil, Augmentin). Vancomycin or Linezolid may also be considered. When the exudates smells offensive, then Gram negatives or anaerobes must be considered and treated with the appropriate medication. In hospital an aminoglycoside can be added, sometimes with metronidazole, or a quinolone.

Any wound of the abdominal wall, pelvis, perineum and thighs down to the knees will be colonised with enteric-type micro-organisms, and the treatment to start with here will be the triple combination of penicillin, an aminoglycoside and metronidazole. This combination can be adjusted (de-escalation) as soon as the cultures are available.

The increase in exudates usually needs a very absorbent dressing and frequent dressing changes as part of the management of the patient. In some situations a vacuum-assisted dressing may be indicated, where an absorbent dressing is used in conjunction with continuous negative pressure suction on the wound to remove all the fluid secreted by the wound. This is often used in cases with the open abdomen.

11. POOR WOUND HEALING

In any patient with a chronic wound, the causes of poor wound healing must be looked for and managed if possible. Some of the causes of poor healing are the following:

a) **Malnutrition.** This is an important factor in a large percentage of hospitalised and institutionalised patients. They do not eat properly, and the underlying disease process also increases their metabolic rate and suppresses their immune system.

b) **Cancer.** Besides the fact that the cancer uses energy, the patient may have loss of appetite, and be kept nil per mouth for special examinations during the diagnostic work-up.

c) **Diabetes mellitus.** This disease leads to starvation amidst plenty. Even though the patient's blood sugar may be high, the cells are starving as the glucose does not enter the cell where it is needed for metabolic processes.

d) **Other infections.** Infections at other sites (such as tuberculosis) will also lead to poor reserves for healing.

e) **Immunosuppression.** HIV and AIDS are the most well-known problems leading to poor healing.

f) **Obesity.** Obese patients have poor healing capacity.

g) **Old age**. Geriatric patients have multiple reasons for poor healing besides age alone.

h) **Medication.** Drugs such as corticosteroids are well known for their immunosuppressive action. Other drugs have the same effect, such as chemotherapy and anti-rejection medication. Radiotherapy has the same effect at a local level as well.

12. WOUND DRESSINGS

A large variety of wound dressings is available on the market, and it is difficult to decide which is best to use in any given situation. A few guiding principles may be helpful in deciding which dressing will be the most appropriate for the patient.

In the previous century it was felt that a wound should form a crust to prevent

infection, and wounds were covered with antiseptics such as mercurochrome, Merthiolate, gentian violet and such colourful fluids to dry the wound. Over the past few decades it was found that moist wound healing was superior to dry healing as far as pain, prevention of infection and cosmetic result are concerned. Wounds are now covered to keep the fluid or moisture on the wound to accelerate the healing process. The fluid secreted by the wound contains inflammatory mediators, neutrophil leucocytes and antibodies to control infection, and later growth factors and fibroblasts to initiate the healing process of fibrosis and scar formation. Epithelialisation proceeds from the wound edges towards the middle – the new epithelium is very friable needing and needs protection from injury by gentle dressing changes.

The different types of dressings can be listed as follows:

a)   **Primary contact layer.** This layer protects the underlying friable tissue. It usually consists of a large mesh type covered with non-adherent ointment (e.g. tulle gras, Jelonet, Adaptic, etc.) or a silicone-based dressing (Mepitel) which is non-adherent.

b)   **Antimicrobials.** This layer contains some antimicrobial, such as topical antibiotics (in sofra tulle and bactroban), silver-based dressings(such as Acticoat, Contreet, Elta silver gel, Silvercel, Promogran prisma, Silvocure, Askina Calgitrol Ag, Allevyn Ag and Actisorb plus silver), iodine-based dressings (Inadine, Betadine, Iodosorb), chlorhexidinebased dressings (Bactigras and Hibiscrub), and medicinal honey ointments (L-mesitran, Melladerm, Activon, Algivon Actilite).

c)   **Moisture control dressings**. These can add moisture if the wound is too dry or has eschar formation, retain the moisture if there is enough, or absorb excess fluid secreted by the wound. Examples of each are the following:

i)   Adding moisture: Mostly the gels (Intrasite gel, Nu-gel, Cutimed gel, Granugel, Citrugel, Askinagel, Aquaclear, Hydroderm, L-mesitran Hydro and Novogel).

ii)   Retaining moisture: These are mainly the occlusive dressings to prevent evaporation of the fluid. The two types are permeable films (such as Opsite, Tegaderm, Leukomed, Biocclusive, Hydrofilm, Merfilm, Askinaderm, Episil and Spyroderm), and hydrocolloids (like Granuflex, Comfeel, Hydrocoil, 3M Tegaderm Hydrocolloid and Askina Hydro).

iii) Absorptive dressings: These are able to absorb large volumes of exudates secreted by the wound. Examples are the foam dressings (Allevyn, Biatain, Tegaderm Foam, Mepilex, Cutimed Siltec, and Cavity, Advazorb, Tielle, Askina foam, Permafoam and Ligazano), alginates (Kaltostat, Seasorb, Sorbagon, Melgisorb, Curasorb), capillary dressings (Drawtex, Advadraw), composite dressings (Combiderm, Alione, Eclypse) and others (Cerdac, Mellasorb Exu-dry, Mesorb).

iv)   Vacuum dressing: When the amount of fluid is too great for a dressing to manage, the continuous suction method can be used. VAC (vacuum assisted closure, KCI) and the Negative Pressure Wound therapy (Smith and Nephew)

are commercially available and can be used to control the exudates from the wound.

d) **Odour control**. When odour is a problem, dressings with activated charcoal are available to absorb the bad smell (Actisorb plus, Askina, Carbosorb, Sorusol). This is usually associated with infection with anaerobic organisms (Bacteroides spp and Peptostreptococci) that generate short-chain fatty acids (SCFAs) via anaerobic metabolism. These cause the smell as they are volatile, and also inhibit the growth of wound-healing cells such as keratinocytes, fibroblasts and endothelial cells. Treatment with antibiotics may be needed.

e) **Enzymatic debriding agents**. When slough needs to be removed, these agents can be used (Iruxol, Ascerbine, Hypergel).

f) **Pain reducing-dressings.** These have an anti-inflammatory drug impregnated in the dressing for slow release to the wound (Biatain Ibu, Phytopain).

g) **Scar management.** When the wound has healed, some dressings can improve the scar and make it less obvious (Advasil, Conform, Bio-Oil, Cica Care, Mepiform, Scarban, Moducoll).

13.   <u>SPECIAL TYPES OF WOUNDS</u>

a)   **Venous ulceration**

i)   Venous stasis ulcers make up 70% of vascular ulcers. They result from chronic venous insufficiency. It is critical to differentiate venous from arterial ulcers as the compression therapy used for venous ulceration could have dire consequences if used in a patient with an arterial ulcer.

Venous malfunction initiates a series of events that result in increased hydrostatic pressure, venous hypertension and ultimately skin ulceration.

ii)   Clinical presentation

Lower limb ulcers are extremely common. A past history of deep vein thrombosis (DVT) is a good predictor of venous incompetence leading to ulceration. A full clinical work-up of the patient's general health status and blood counts should therefore be taken and all systems should be examined with special emphasis on the appearance of the lower extremities. Ankle oedema is the earliest sign of chronic venous insufficiency (oedema is usually minimal or absent in arterial disease). Leg pain relieved by elevation is consistent with venous disease (in arterial disease pain is exacerbated with elevation) and the skin on the lower limbs is usually hard and fibrotic.

iii)   Other signs include dilated superficial tissue veins, dermatitis, pigmentation due to extravasation of red blood cells into the skin which looks purple in light-skinned people and dark brown or dark purple in dark-skinned people, and dry, flaky skin that has the appearance of fish scales. Venous ulcers are also characterised by irregular wound margins; they are non-tender, associated with eczema, covered with exudate and usually located on the medial aspect of the lower leg or ankles

(arterial ulcers occur on toes, feet and unusual locations, are tender, grey and may contain necrotic tissue). Palpation of peripheral pulses and temperature assessment of lower limbs and hands is essential. Cold skin with diminished pulses suggests arterial disease.

iv) Chronic non-healing ulcers should be biopsied to exclude carcinoma or vasculitis. Underlying osteomyelitis (X-ray confirmation), contact dermatitis and soft tissue infections should be excluded or identified and treated. Once diagnostic tests have ruled out arterial disease, then compression therapy and moist wound environment are the mainstays of venous ulcer management. High compression (>35 mmHg) is more effective than low compression but should only be used in the absence of arterial disease. Non-compliance with compression is, however, often a problem, hence the importance of patient education. In post-thrombotic syndrome or if venous insufficiency is still present, ulcers tend to recur. General or systemic risk factors facilitating recurrence would be obesity, inadequate nutrition, lack of exercise and smoking.

b) **Arterial ulcers**

i) Peripheral ischaemia and reduced skin blood flow may lead to arterial ulcers. Arterial insufficiency and occlusive disease is usually caused by atherosclerosis of the extremities. The incidence of arterial insufficiency and ulceration increases with age – the sixth and seventh decades account for the highest incidence. Risk factors also include male patients, and those who have diabetes mellitus, hypertension, Reynaud's Disease, sickle cell anaemia, hypercholesterolemia, a sedentary lifestyle, obesity and smoking.

ii) It is imperative to differentiate between venous ulceration and arterial ulcers, as treating these two disease processes are vastly different. The diagnosis of each is vital to avoid inappropriate and dangerous treatment. Venous ulcers occur slowly and are associated with oedema. Arterial ulcers occur in patients with the risk factors mentioned above and are usually located on the lower leg over the toes, between the toes or on the tips, on the heels or bony prominences of the foot and rarely over the medial malleolus. They have deep pale wound beds with even wound margins. The surrounding atrophic skin changes described previously are present as well as the symptoms and signs of arterial insufficiency. A history of intermittent claudication, coldness and numbness in the toes and feet is typical. Resting pain in the lower limbs which improves when the limb is dependent, is a sign of advanced atherosclerotic vascular disease.

iii) Examination reveals decreased or absent pulses, rubor with the foot in a dependent position, pallor when elevated, cool dry skin and bruits over the narrowed artery. As the disease progresses trophic skin changes are seen: cyanosis, thin shiny skin, loss of hair and thickened deformed toenails. Diagnosis is critical and this is usually apparent on history and examination. Doppler flow studies provide useful additional information.

iv) Arterial ulcers are particularly difficult to treat as the cause, arterial insufficiency, is progressive and in many cases irreversible with conservative means. The goal for treatment of patients with arterial ulcers is to increase the blood supply to the

affected area. Proper maintenance of the feet is important by protecting bony prominences, hygiene and properly fitting shoes and socks. Elevating the head of the bed promotes blood flow to the feet. Local treatment involves adequate debridement and moist occlusive dressings once or twice a week. Operative intervention such as aorta-bifemoral bypass, may be necessary in cases resistant to conservative therapy. While the local treatment of the ulcer is important, it is futile if the risk factors and underlying disease are not controlled. These include cessation of smoking, control of diabetes, obesity, and cholesterol, exercising and use of anti-coagulants where necessary.

c)   **Wound healing in diabetes mellitus**

i)   Diabetic ulcers of the lower leg often present innocently but may progress rapidly to fulminating infection and amputation. They have the highest lower limb amputation rate of any chronic leg wound. Ulceration mainly affects the feet.

ii)   Factors contributing to altered healing:

(1)   Ischaemia: This is a combination of macrovascular atherosclerosis with emboli, and microvascular disease with a pathologically thickened perivascular capillary basement membrane. This abnormality may cause increased vascular permeability resulting in oedema and extracellular matrix depositional "trapping" of inflammatory cells and suppression of leukocyte function.

(2)   Neuropathy: This affects all nerve types with resultant loss of pain protection and protective reflexes. The foot muscles loose their motor feedback causing the arch of the foot to collapse resulting in deformed metatarsals and clawing of the toes. Painless repetitive injuries from minor wounds such as trimming of toe nails lead to chronic ulceration, and ill-fitting shoes lead to pressure necrosis on the plantar surface and tips of the toes. Protective calluses increase the chance of pressure. Loss of sympathetic nerve supply leads to a non-sweating foot, and the lack of moisture causes cracking of the skin, alteration of bacterial flora and invasion with colonisation.

(3)   Uraemia: Silent or overt renal failure causes loss of protein and oedema, but also independently alters wound healing.

(4)   Suppressed immunity: A reduced ability to deal with infection exists on a cellular and biochemical level. Not only is there a break in the physical barrier through skin cracking, but lack of glucose control impairs local leukocyte defences. There is a decreased production and an increased destruction of growth factors present (insulin-like GF I and II and keratinocyte GF). Hyperglycaemia contributes to the metabolic pathophysiology of diabetes-related complications (Greenhalgh). This occurs through intricate pathways at the molecular level.

(iii) Clinically the classic chronic diabetic ulcer presents as a small punctate wound over the plantar aspect of a deformed metatarsal head or toe tip. The rim has a raised epithelial edge with pale granulation in the centre. There may be surrounding cellulitis present and in a progressive fashion the infection may invade along plantar fascial planes or cause failed wound healing of gangrenous

toes, and metatarsal or eventually below-knee amputation. Ulcers secondary to necrobiosis lipoidica usually occur over the pretibial region.

iv)   Management of the diabetic foot:

(1)   Preventive foot maintenance includes careful nail clipping, selection of well-fitting shoes and routine daily foot inspection.

(2)   Evaluation of proximal vascular status and sensory function. Calcification makes the arteries inflexible and therefore palpable ankle pulses, toe blood pressure and ankle – brachial (AB) index above 0,5 as predictors of ulcer healing ability, is not reliable. Small ulcer size of short duration in non-white patients and transcutaneous oxygen levels above 25-30 mm Hg are reliable indicators of the ability to heal.

(3)   Tight control of blood sugar and infection is essential. Aggressive debridement, removal of surrounding callus and broad-spectrum antibiotics are indicated.

(4)   Dialysis for uraemia (or a kidney transplant) is beneficial.

(5)   Meticulous protection and wound care will heal superficial shallow ulcers. "Total contact" casts will "off-load" pressure and restricted activity should heal moderately deep ulcers.

(6)   Reconstructive surgery includes vascular bypass, skin grafts, pedicled flaps and free flaps for deep, extensive and bony defects. Limb salvage operations require careful planning with full patient co-operation to prevent early breakdown and failure.

(7)   Hyperbaric oxygen may theoretically be advantageous but lack of randomised, prospective research evidence makes it controversial.

(8)   Natural or engineered growth factors show potential promise. The only commercially available growth factor in the USA is for diabetic ulcer treatment.

d)   **Radiated wounds**

i)   The effects of surgery are permanent and immediately obvious, while the effects of radiation are permanent, continuous and progressive and continue throughout the patient's entire life (Anon).

ii)   The exact incidence of clinical complications due to radiotherapy is not known because they may develop many years after original exposure. In the treatment of head and neck cancer it may be as high as 65%, (Harson). In our efforts to achieve maximum long-term cure for certain cancers with combination therapy, we sometimes cause not only severe physical deformity but also complicate difficult reconstructions due to radiation sequelae. Radiation as a primary single modality of treatment has as one of its aims the preservation of important functional and aesthetic structures. An example is the intra-oral reconstruction of a functional tongue or soft palate after total surgical ablation for cancer. As with

breast conservation surgery and pelvi-perineal resection for cancer, it is still not possible to perform a fully functional reconstruction. At some centres radiation is favoured as primary treatment for these conditions. Regrettably, fibrotic "frozen" tissue may result that could be painful and ulcerating, with the potential of adversely affecting quality of life.

iii) Pathophysiology: The sequence of events following radiation continues to worsen long after the noxious agent has been removed. Basically the gradual and progressive obliterative endarteritis and cellular dysfunction leads to hypoxic, hypo-vascular and hypo-cellular tissue (Marx). The radiated area is characterised by loss of collagen and increased fibrosis with contraction. Revascularisation cannot occur in collagen-deficient tissue because budding capillaries do not have a soft collagenous matrix to invade. There is a progressive loss of vascularity in a nearly linear fashion over time. This creates a hypoxic wound bed. In radiated wounds, the oxygen gradient decreases from the wound edge to the centre of the wound at such a gradual rate that the hypoxic stimulus for angiogenesis is not initiated. This results in the formation of poor granulation tissue and a wound of inferior quality that will normally not heal on its own. Irradiation causes quantitative and qualitative suppression of fibroblasts and formation of inferior quality collagen. Decreased mitotic activity and reduced numbers of epithelial cells are also present, causing thinning and complete destruction of the epithelium.

iv) Management: Basic management principles are used, e.g. nutritional support, elimination of causative agents, control of infection, debridement, pressure irrigation, occlusive dressings and eventual reconstructive procedures. Promising options for the future remain the therapeutic use of growth factors and cytokines (transforming GF-beta, platelet-derived GF, interleukin–3 and granulocyte-macrophage GF). Infection is often a difficult problem to handle and local antiseptics supported by systemic antibiotics is often necessary for control. Complicated flap reconstructions after repeated wide excisions can heal these often extensive wounds.

v) When operating in irradiated tissue, some basic principles should be adhered to:

(1) Primary closure of oedematous wounds should be resisted.

(2) Skin or muscle flaps must be used for tension-free closure to prevent exposure of vital structures.

(3) Split skin grafts should be used preferably on immobile areas.

(4) Incisions over vital structures, e.g. carotid vessels in the neck, should be avoided. With breakdown of tissue, exposure of vessels creates the life-threatening complication of a vascular "blow-out".

(5) Free grafts (e.g. bone) are rarely successful.

e) **Control of oedema**

Some chronic wounds such as varicose ulcers of the lower extremities are caused by poor venous return and oedema of the limb. In these cases wound care is as important as control of the oedema of the limb by using compression dressings and bandages, and even compression stockings. These wounds will not heal if the oedema is uncontrolled, and if healed, will usually break down again after treatment if swelling develops again. These patients will have to wear pressure garments for the rest of their lives, and may also have an ulcer for life if not cared for properly. This is a large drain on the resources of any modern health care system.

14. MAGGOT THERAPY

Maggot therapy has been known for a very long time. In the 1500s it was recorded that wounds infested with maggots were healthy looking and healed well. Maggots were used as therapy in the American Civil War in the 1860s. Further research was done early in the 20th century, but with the development of antibiotics from the 1930s maggot therapy became obsolete.

Interest in maggot therapy emerged again in the 1960s when resistance to antibiotics started developing, and currently a few centres around the world are using it regularly. The most common larvae used are those of the green bottle blowfly, *Lucilia sericata*, which only feed on dead tissue. The flies are kept in controlled conditions, and lays eggs which hatch in 18 to 24 hours. The eggs are sterilised to produce sterile larvae, which are then placed on the wound that needs treatment. Here they feed for 3 to 7 days, before they develop into pupae, which are removed from the wound and discarded with the used dressings.
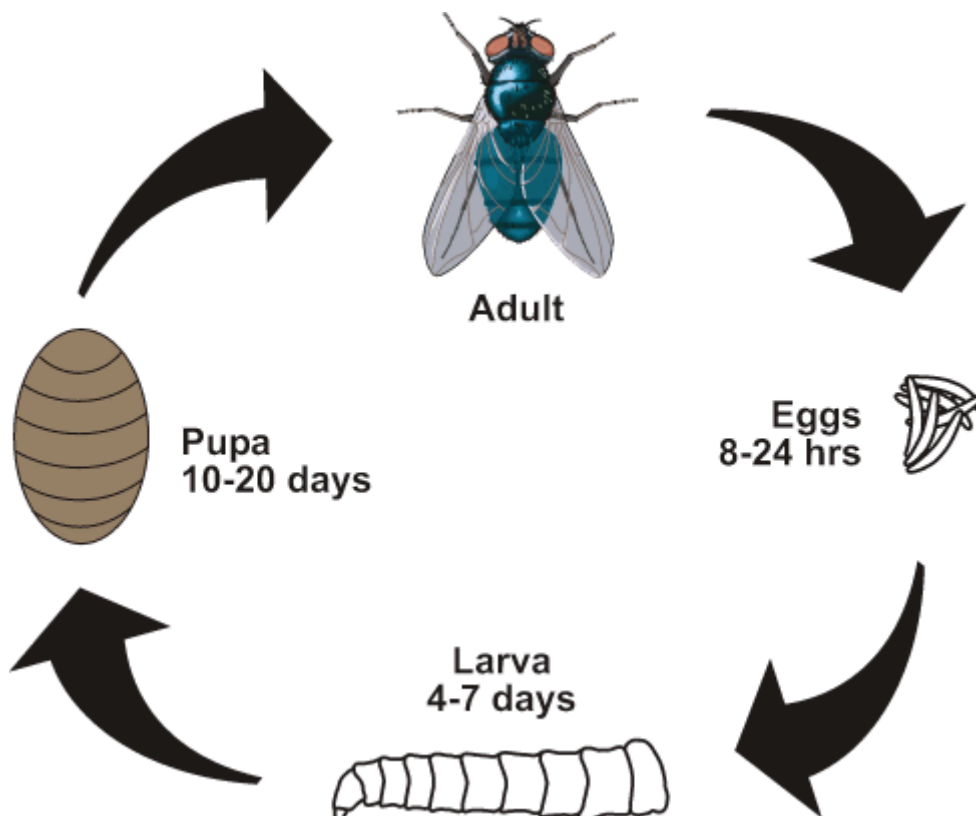
Adult

Eggs
8-24 hrs

Larva
4-7 days

Pupa
10-20 days

Figure 8: Life cycle of the blowfly, *Lucilia sericata*

The maggots debride and disinfect the wound, and stimulate tissue growth. The debridement is an extracorporeal digestion of necrotic tissue by proteolytic enzymes secreted by the maggots. They also secrete antimicrobial factors that kill micro-organisms and disinfect the wound, and cytokines and growth factors that stimulate and promote healing. A secreted substance also causes vasodilatation, leading to the development of granulation tissue in the wound.

In practice the wound is cleaned with saline, maggots are applied and covered with a dressing permeable to air which allows oxygen to get to the maggots. This is left in place for 3-5 days before opening of the dressing and removal of the maggots. This process can be repeated until the wound is clean of slough and necrotic tissue, when standard wound care can be commenced.

15. HYPERBARIC OXYGEN (HBO) THERAPY

a) The principles of managing problem wounds include correction of perfusion and oxygenation insufficiencies, debridement, control of infection, wound care and surgical reconstruction. If a patient suffers from deficient tissue oxygenation due to non-reconstructable vascular disease, HBO as an adjunctive therapy may be indicated. The best tool available to evaluate tissue hypoxia is transcutaneous oxygen tension ($TcPO_2$). A value greater than 50 mmHg indicates spontaneous healing, whereas a value between 30-50 mmHg is marginal and below 30 mmHg HBO therapy is required. HBO therapy delivers 100% oxygen to the patient at greater than two times the normal atmospheric pressure at sea level. This increases the partial pressure of oxygen in plasma resulting in "hyperoxic plasma" and tissue PO_2 levels exceeding 600 mmHg. HBO creates a steep tissue oxygenation gradient, forcing oxygen into injured and healing tissues by diffusion. It is accomplished via different chambers accommodating single or multiple patients. HBO therapy enhances wound healing by increasing neutrophil bactericidal capacity (kills anaerobic bacteria and inhibits toxin formation), stimulating fibroblasts and promoting angiogenesis.

b) HBO is indicated for osteoradionecrosis, chronic osteomyelitis, necrotising infections, ischaemic reperfusion and thermal injuries. Although double-blinded prospective randomised research results are lacking, some treatment results of diabetic ulceration with HBO seem to be very promising. Problem wounds relate significantly to a patient's productivity, disability and premature death. Combined with other modalities, HBO treatment in selected patients does improve healing and may improve the outcome.

16. RECONSTRUCTION OF TISSUE DEFECTS

a) After assessment of a wounded patient and classification of the wounds, the most important action of the wound care team is to formulate and implement an appropriate treatment plan. General factors influencing this are the patient's age, occupation, mobility, psychological tenacity, expectations and co-operation. Added to this is the availability of expertise and logistical factors. Although the

famous Sir Harold Gilles coined the phrase "Never do today what can be better done tomorrow", his intention was never to advocate postponement of treatment for non-medical reasons. Indecisiveness and delegating important wound management decisions to the most junior members of the team is unethical and are a great injustice to the wounded patient.

b)　　Different options should follow the reconstructive ladder from simple to complex, but sometimes it will be necessary to travel by lift, by-passing simpler options and electing a more complicated but better one:

i)　　Non-surgical treatment with dressings (consult with experts)

ii)　　Primary/delayed primary/secondary suturing

iii)　　Excision of ulcer bed, debridement(s)

iv)　　Skin grafting (split or full-thickness)

v)　　Local/regional/distant pedicled flaps (refer to surgeon with the necessary expertise)

vi)　　Free microvascular tissue transfers

vii)　　Correction of arterial inflow and venous incompetence

c).　　The prerequisite for choosing a non-surgical option is the belief that the wounds will heal with conservative treatment in a reasonable time. Allowing full-thickness skin defects to heal by secondary intention leaves a poor quality scar and should only happen if dictated by the patient's general condition. Skin grafting requires an adequate bed for the graft to take.

d)　　Five common reasons why skin grafts fail are:

i)　　poor quality bed (old granulation tissue)

ii)　　haematoma/seroma (between graft and bed)

iii)　　infection (Streptococcus species)

iv)　　movement (apply post-op splints to extremities)

v)　　technical (thickness of graft)

e)　　Indications for flap reconstruction are:

i)　　inadequate bed (exposed bone, cartilage, neuro-vascular bundles, tendons)

ii)　　poor vascular bed

iii)　　exposed body cavities (chest wall and dura) and vital organs (heart, lungs, brain)

iv)   reconstruction of function and sensation (e.g. sole of foot)

17.   KELOIDS

a)   A keloid is descriptive of an uncontrollable growth of scar tissue. Keloids must be differentiated from hypertrophic scars. A hypertrophic scar is confined to the area of injury or incision and usually flattens with time. A keloid grows beyond the boundaries of the wound and does not usually improve with time. Keloids are unpredictable in their behaviour, as they sometimes occur only in certain wounds and not in others in the same individual. With multiple ear piercing in vogue at present, another intriguing phenomenon has become apparent. Keloid formation can occur at the second piercing but not at first piercing. There is also the phenomenon of spontaneous keloids. These are keloids that are usually multiple and occur on most parts of the body. Such patients have no prior history of wounds. Keloids do not form in animals, so they cannot be used as research models.

b)   The precise aetiology and pathogenesis of keloids is unknown. Several associated factors have been observed.

i)   Genetic: Although there have been sporadic reports of keloids in families, the majority of patients with keloids do not have a family history of the condition. No gene markers have yet been identified.

ii)   Race: Keloids are more common in Negroid and Asian population groups and less common in people of Caucasian descent. Melanocytes or melanin pigment may play a role in the aetiopathogenesis of keloids because none have been reported in albinos.

iii)   Anatomical sites: There are certain areas of the body that have a predilection for keloids. These include the ears, back, presternal, arm and shoulder regions. They are rarely found in the upper eyelids, palms, soles and genitalia.

iv)   Wound factors: Factors in wound closure influencing the likelihood of keloid formation are tension, suppuration and wound infection leading to healing by secondary intention, some growth factors and cytokines such as GF beta.

v)   Immune theory: Some have likened the occurrence of a keloid to that of a secondary immune response. The progression of an initial keloid may be slow (primary response), and the recurrence is more florid (secondary response).

vi)   Biochemistry:

Collagen type in normal skin – the dermis has approximately 85% collagen type I and 15% collagen type III, but in keloids collagen type III is increased. There is also an increase in chondroitin-4-sulphate, alpha-2-macroglobulin, alpha-1-antitripsin, and activity of collagenase.

c)   No single mode of therapy is effective for keloids. Because of the high

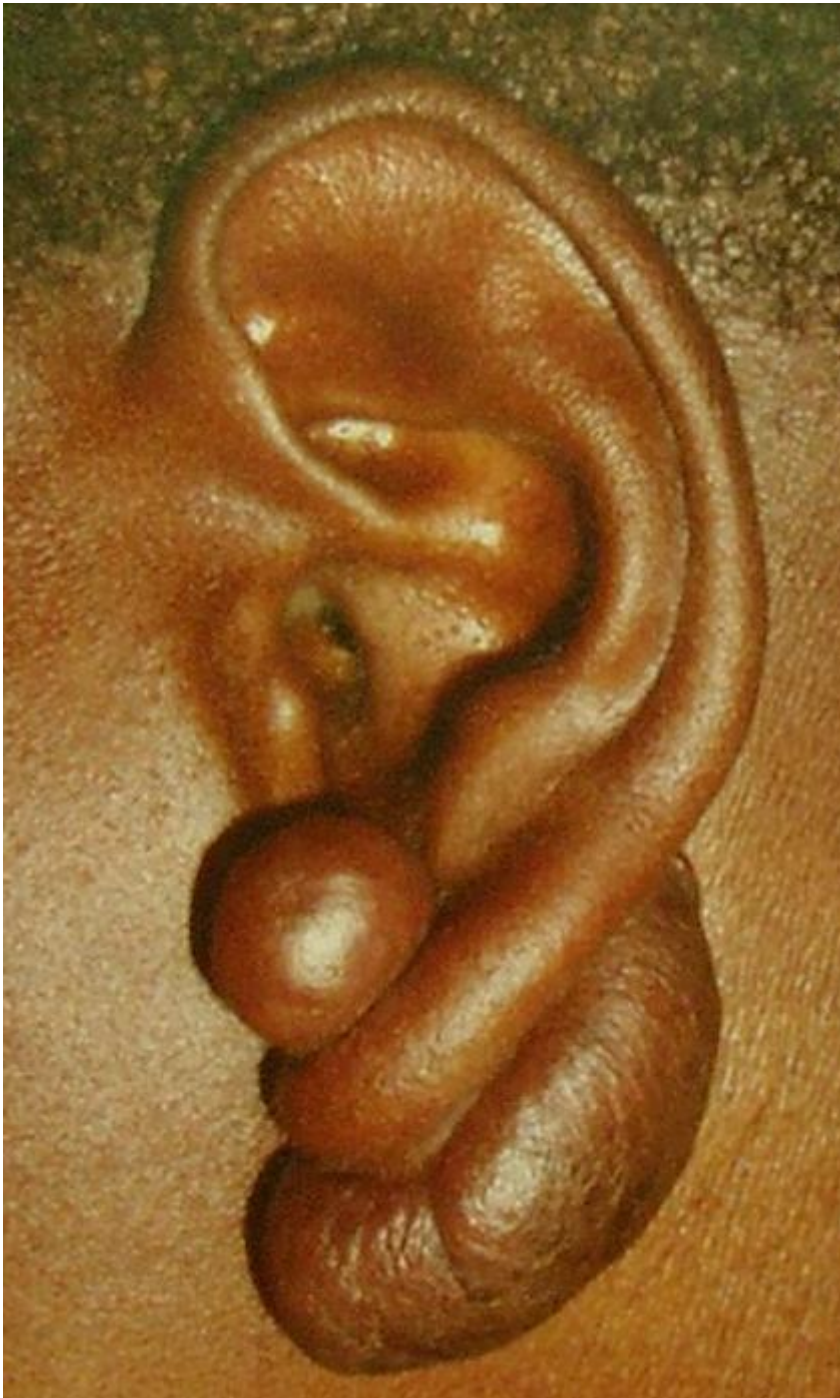occurrence rates, a multimode therapy approach is recommended.

Figure 9: Keloid formation after ear piercing

(From Swan, M. 2010. The Principles of managing acute wounds. Wounds (UK) 6(4):42-50)

i)   Pharmaceutical: Steroids. Intralesional injection of steroids can be used preoperatively to soften the keloid and postoperatively to prevent or treat early recurrences. Injections are repeated every 2 to 6 weeks. Bleomycin has also been used intralesionally as well as several other agents such as colchicine, D-

penicillinamine and beta amino-propionitile.

ii) Surgery: This is the most frequent mode of management and should be performed at the appropriate time. If performed on a floridly developing keloid, the chances of recurrence are much higher and the result may be more disfiguring than the original keloid. After keloid excision, one must be take care not to close the wound under tension.

iii) Postoperative radiation: Usually performed for 3 consecutive days immediately post-surgery.

iv) Pressure therapy and taping: Taping of a suture line certainly results in a superior scar outcome. Pressure therapy in the form of pressure garments, facial masks and clip-on earrings is recommended. This has to be used for a protracted period of not less than 12 months.

v) Silicone gel – sheets or topical ointment

vi) Immunotherapy – Intralesional interferon

18. **AMPUTATION**

When the blood supply to a wound is inadequate because of atherosclerosis, diabetes mellitus or other systemic diseases, a vascular consultation may be required for the possible improvement of the circulation. If this is not possible, an amputation of the limb may be necessary to control the progress of the disease process and prevent systemic sepsis. The decision as to the level of amputation may be a difficult in a patient who is a poor anaesthetic risk, has underlying vascular disease and infection in the wound. Too low an amputation level will not eliminate the infection, and may still be in the area if blood supply is critical. A higher amputation level will have a better blood supply to heal, but may be too high for rehabilitation with or without prosthesis.

When sepsis is the indication for amputation, the decision is between a guillotine amputation (cut through all elements of the limb at the same level) or fashioning a proper stump with the bone short with the muscle and skin flaps longer, but leaving it open for later closure when the infection is under control. Advice from a senior colleague or orthopaedic consultant may help in making the correct decision.

19. **CONCLUSION**

Wound care is one of the oldest treatments that physicians have carried out on patients in the history of medicine. With the developments in wound care during the past 50 years, and the new dressings that have become available, wound care is no longer a best-guess approach but a science. It is now known that there are basic principles that need to be followed, and a fair amount of knowledge is necessary for proper and efficient wound care.