Chapter 3

Wounds, healing and tissue repair

Learning objectives

To understand:

- Normal healing and how it can be adversely affected
- How to manage wounds of different types, of different structures and at different sites
- Aspects of disordered healing that lead to chronic wounds
- The variety of scars and their treatment
- How to differentiate between acute and chronic wounds

INTRODUCTION

Wound healing is a mechanism whereby the body attempts to restore the integrity of the injured part. This falls far short of tissue regeneration by pluripotent cells, seen in some amphibians, and is often detrimental, as seen in the problems created by scarring, such as adhesions, keloids, contractures and cirrhosis of the liver. Several factors may influence healing. However, a clean incised wound in a healthy person where there is no skin loss will follow a set pattern as outlined below.

Summary box 3.1

Factors influencing healing of a wound

- Site of the wound
- Structures involved
- Mechanism of wounding
  - Incision
  - Crush
  - Crush avulsion
- Contamination (foreign bodies/bacteria)[a]
- 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

[a] In explosions, the contamination may consist of tissue such as bone from another individual.

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 (**Figure 3.1**).

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. 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). These attract inflammatory cells such as polymorphonuclear leukocytes (PMN) and macrophages. Platelets and the local injured tissue release vasoactive amines, such as histamine, serotonin and prostaglandins, which increase vascular permeability, thereby aiding infiltration of these inflammatory cells. Macrophages remove devitalised tissue and microorganisms while regulating fibroblast activity in the proliferative phase of healing. The initial framework for structural support of cells is provided by fibrin produced by fibrinogen. A more historical (Latin) description of this phase is described in four words: *rubor* (redness), *tumor* (swelling), *calor* (heat) and *dolor* (pain).

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 (glycosaminogly-

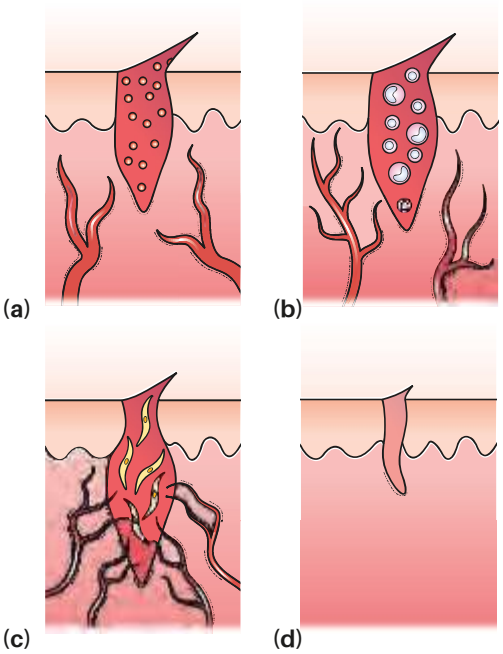


Figure 3.1 The phases of healing. **(a)** Early inflammatory phase with platelet-enriched blood clot and dilated vessels. **(b)** Late inflammatory phase with increased vascularity and increase in polymorphonuclear leukocytes and lymphocytes (round cells). **(c)** Proliferative phase with capillary buds and fibroblasts. **(d)** Mature contracted scar.

cans 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. This proliferative phase with its increase of collagen deposition is associated with wound contraction, which can considerably reduce the surface area of a wound over the first 3 weeks of healing.

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. This maturation of collagen leads to increased tensile strength in the wound which is maximal at the 12th week post injury and represents approximately 80% of the uninjured skin strength.

NORMAL HEALING IN SPECIFIC TISSUES

Bone

The phases are as above, but periosteal and endosteal proliferation leads to the formation of callus, which is immature bone consisting of osteoid (mineralised by hydroxyapatite and laid down by osteoblasts). In the remodelling phase, cortical structure and the medullary cavity are restored. If fracture ends are accurately opposed and rigidly fixed, callus formation is minimal and primary healing occurs. If a gap exists, then secondary healing may lead to delayed union, non-union or malunion.

Nerve

Distal to the wound, Wallerian degeneration occurs. Proximally, the nerve suffers traumatic degeneration as far as the last node of Ranvier. The regenerating nerve fibres are attracted to their receptors by neurotrophism, which is mediated by growth factors, hormones and other extracellular matrix trophins. Nerve regeneration is characterised by profuse growth of new nerve fibres which sprout from the cut proximal end. Overgrowth of these, coupled with poor approximation, may lead to neuroma formation.

Tendon

Although repair follows the normal pattern of wound healing, there are two main mechanisms whereby nutrients, cells and new vessels reach the severed tendon. These are intrinsic, which consists of vincular blood flow and synovial diffusion, and extrinsic, which depends on the formation of fibrous adhesions between the tendon and the tendon sheath. The random nature of the initial collagen produced means that the tendon lacks tensile strength for the first 3–6 weeks. Active mobilisation prevents adhesions limiting range of motion, but the tendon must be protected by splintage in order to avoid rupture of the repair.

ABNORMAL HEALING

Some of the adverse influences on wound healing are listed in *Summary box 3.1*. 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.

Summary box 3.2

Classification of wound closure and healing

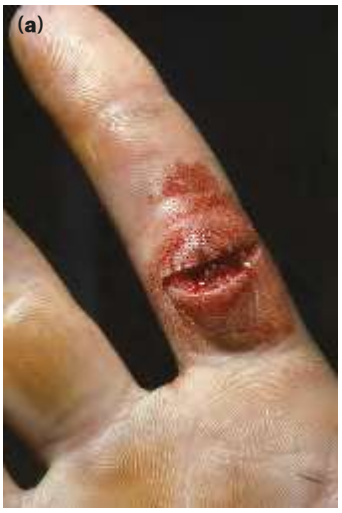
- Primary intention
  - Wound edges opposed
  - Normal healing
  - Minimal scar
- Secondary intention
  - Wound left open
  - Heals by granulation, contraction and epithelialisation
  - Increased inflammation and proliferation
  - Poor scar
- Tertiary intention (also called delayed primary intention)
  - Wound initially left open
  - Edges later opposed when healing conditions favourable

Augustus Volney Waller, 1816–1870, general practitioner of Kensington, London, UK (1842–1851), subsequently worked as a physiologist in Bonn, Germany; Paris, France; Birmingham, UK; and Geneva, Switzerland.
Louis Antoine Ranvier, 1835–1922, physician and histologist who was a professor in the College of France, Paris, France, described these nodes in 1878.

Healing by primary intention is also known as healing by first intention. This occurs when there is apposition of the wound edges and minimal surrounding tissue trauma that causes least inflammation and leaves the best scar. 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. This is also called healing by tertiary intention in some texts and will result in a less satisfactory scar than would result after healing by primary intention. Secondary healing or healing by secondary intention occurs in wounds that are left open and allowed to heal by granulation, contraction and epithelialisation.

TYPES OF WOUNDS – TIDY VERSUS UNTIDY

The site injured, the structures involved in the injury and the mechanism of injury (e.g. incision or explosion) all influence healing and recovery of function. This has led to the management of wounds based upon their classification into tidy and untidy (*Table 3.1* and **Figure 3.2**).



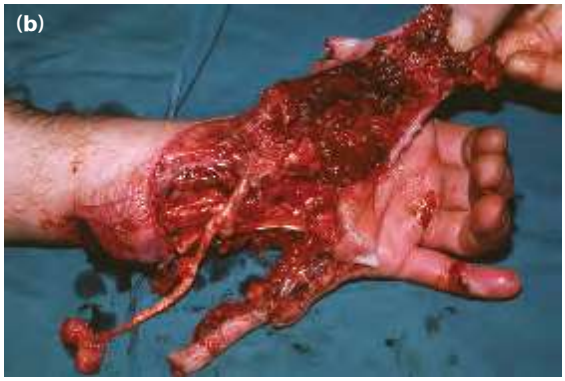


Figure 3.2 **(a)** Tidy incised wound on the finger. **(b)** Untidy avulsed wound on the hand.

The surgeon's aim is to convert untidy to tidy by removing all contaminated and devitalised tissue.

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). This is especially true in injuries caused by explosions, bullets or other missiles, where the external wound itself may appear much smaller than the wider extent of the injured tissues deep to the surface. 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 (see Compartment syndromes below). Any explosion where there are multiple victims at the same site or where there has been a suicide-related explosion will carry the risk of tissue and viral contamination. Appropriate tests for hepatitis viruses and human immunodeficiency virus (HIV) are required.

TABLE 3.1 Tidy versus untidy wounds.

Tidy	Untidy
Incised	Crushed or avulsed
Clean	Contaminated
Healthy tissues	Devitalised tissues
Seldom tissue loss	Often tissue loss

MANAGING THE ACUTE WOUND

The surgeon must remember to examine the whole patient according to acute trauma life support (ATLS) principles. A stab wound in the back can be missed just as easily in the reality of the accident and emergency room as in a fictitious detective novel. The wound itself should be examined, taking into consideration the site and the possible structures damaged (**Figure 3.3**). It is essential to assess movement and sensation while

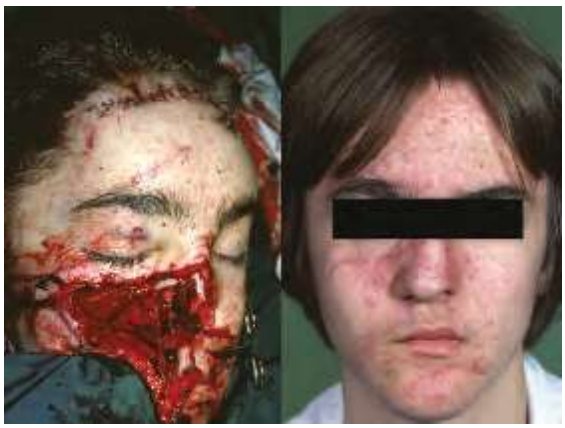


Figure 3.3 Facial trauma – apparent tissue loss but none found after careful matching.

The term '**debridement**' was introduced by the great French surgeon in Napoleon's army, Dominique Jean Larrey (1766–1842). He used it to describe the removal of bullets, bits of cloth, loose bits of bone and soft tissue.

watching for pain and listening to the patient. Tetanus cover should be noted and appropriate treatment carried out.

A bleeding wound should be elevated and a pressure pad applied. Clamps should not be put on vessels blindly because nerve damage is likely and vascular anastomosis is rendered impossible.

In order to facilitate examination, adequate analgesia and/or anaesthesia (local, regional or general) are required. General anaesthesia is often needed in children. With limb injuries, particularly those of the hand, a tourniquet should be used in order to facilitate visualisation of all structures. Due care should be taken with tourniquet application, avoiding uneven pressure and noting the duration of tourniquet time.

After assessment, a thorough debridement is essential. Abrasions, 'road rash' (following a fall from a motorbike) and explosions all cause dirt tattooing and require the use of a scrubbing brush or even excision under magnification. A wound should be explored and debrided to the limit of blood staining. Devitalised tissue must be excised until bleeding occurs, with the obvious exceptions of nerves, vessels and tendons. These may survive with adequate revascularisation subsequently or after being covered with viable tissue such as that brought in by skin or muscle flaps.

The use of copious saline irrigation or pulsed jet lavage (where the instrumentation is available) can be less destructive than knife or scissors when debriding. However, it has been suggested that pulsed jet lavage can implant dirt into a deeper plane and care should be taken to avoid this complication. Muscle viability is judged by the colour, bleeding pattern and contractility. In a tidy wound, repair of all damaged structures may be attempted. Repair of nerves under magnification (loupes or microscope) using 8/0 or 10/0 monofilament nylon is usual. Vessels such as the radial or ulnar artery may be repaired using similar techniques. Tendon repairs, particularly those in the hand, benefit from early active mobilisation because this minimises adhesions between the tendon and the tendon sheath (see above under **Tendon** for extrinsic tendon healing mechanism).

Skin cover by flap or graft may be required as skin closure should always be without tension and should allow for the oedema typically associated with injury and the inflammatory phase of healing. A flap brings in a new blood supply and can be used to cover tendon, nerve, bone and other structures that would not provide a suitable vascular base for a skin graft. A skin graft has no inherent blood supply and is dependent on the recipient site for nutrition.

SOME SPECIFIC WOUNDS

Bites

Most bites involve either puncture wounds or avulsions. Bites from small animals are common in children (**Figure 3.4**) and require cleansing and treatment according to the principles outlined in *Summary box 3.3*, usually under general anaesthetic.

Injuries to the ear, tip of nose and lower lip are most usually seen in victims of human bites. A boxing-type injury of the metacarpophalangeal joint may result from a perforating contact with the teeth of a victim. Anaerobic and aerobic organism prophylaxis is required as bite wounds typically have high virulent bacterial counts.

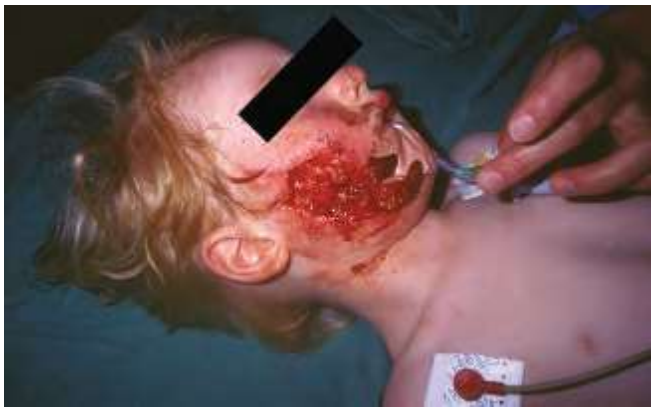


Figure 3.4 Dog bite in a child.

Summary box 3.3

Managing the acute wound

- Cleansing
- Exploration and diagnosis
- Debridement
- Repair of structures
- Replacement of lost tissues where indicated
- Skin cover if required
- Skin closure without tension
- All of the above with careful tissue handling and meticulous technique

Puncture wounds

Wounds caused by sharp objects should be explored to the limit of tissue blood staining. Needle-stick injuries should be treated according to the well-published protocols because of hepatitis and 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. In the gluteal or thigh region, there may be an associated disruption of fat in the form of a fat fracture, which results in an unsightly groove but intact skin. An untreated haematoma may also calcify and therefore require surgical exploration if symptomatic.

Degloving

Degloving occurs when the skin and subcutaneous fat are stripped by avulsion from the underlying fascia, leaving neurovascular structures, tendon or bone exposed. A degloving injury may be open or closed. An obvious example of an open degloving is a ring avulsion injury with loss of finger

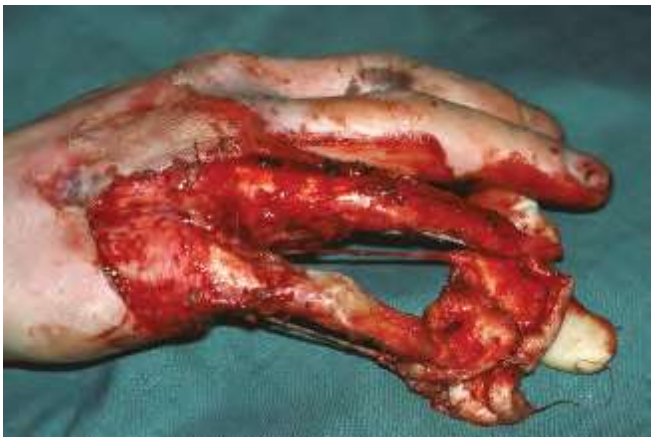


Figure 3.5 Degloving hand injury.

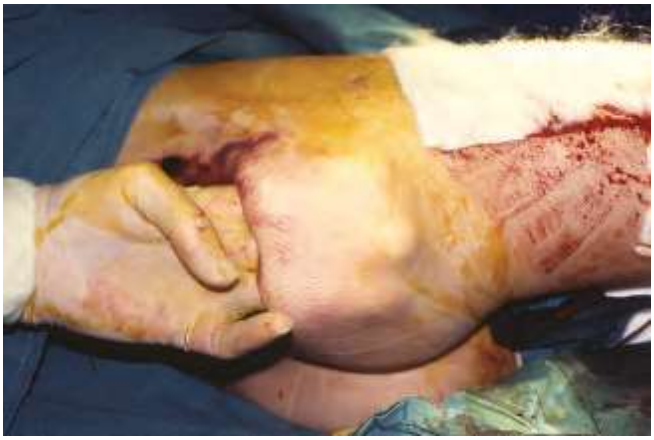


Figure 3.6 Degloving buttock injury.

skin (**Figure 3.5**). A closed degloving may be a rollover injury, typically caused by a motor vehicle over a limb. Such an injury will extend far further than expected, and much of the limb skin may be non-viable (**Figure 3.6**). Examination under anaesthetic is required with a radical excision of all non-bleeding skin, as judged by bleeding dermis. Fluoroscein can be administered intravenously while the patient is anaesthetised. Under ultraviolet light, viable (perfused) skin will show up as a fluorescent yellowish green colour, and the non-viable skin for excision is clearly mapped out. However, the main objection to this method is that of possible anaphylactic shock due to fluoroscein sensitivity. Most surgeons therefore rely upon serial excision until punctate dermal bleeding is obvious. Split-skin grafts can be harvested from the degloved non-viable skin and meshed (**Figure 3.7**) to cover the raw areas resulting from debridement.

Compartment syndromes

Compartment syndromes typically occur in closed lower limb injuries. They are characterised by severe pain, pain on passive movement of the affected compartment muscles, distal sensory disturbance and, finally, by the absence of pulses distally (a late sign). They can occur with an open injury if the wound does not extend into the affected compartment.

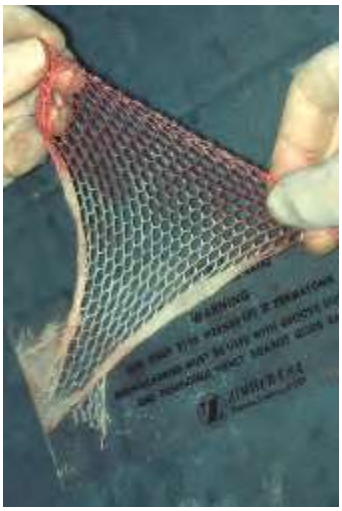


Figure 3.7 Meshed split-skin graft.

Compartment pressures can be measured using a pressure monitor and a catheter placed in the muscle compartment. If pressures are constantly greater than 30 mmHg or if the above clinical signs are present, then fasciotomy should be performed. Fasciotomy involves incising the deep muscle fascia and is best carried out via longitudinal incisions of skin, fat and fascia (**Figure 3.8**). The muscle will then be seen bulging out through the fasciotomy opening. The lower limb can be decompressed via two incisions, each being lateral to the subcutaneous border of the tibia. This gives access to the two posterior compartments and to the peroneal and anterior compartments of the leg. In crush injuries that present several days after the event, a late fasciotomy can be dangerous because dead muscle produces myoglobin which, if suddenly released into the blood stream, causes myoglobinuria with glomerular blockage and renal failure. In the late treatment of lower limb injuries, therefore, it may be safer to amputate the limb once viable and non-viable tissues have been demarcated.

High-pressure injection injuries

The use of high-pressure devices in cleaning, degreasing and painting can cause extensive closed injuries through

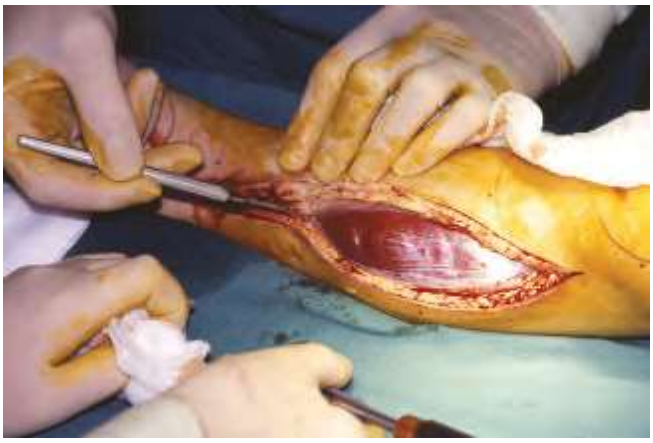


Figure 3.8 Fasciotomy of the lower leg.

small entry wounds. The liquid injected spreads along fascial planes, a common site being from finger to forearm. The tissue damage is dependent upon the toxicity of the substance and the injection pressure. Treatment is surgical, with wide exposure, removal of the toxic substance and thorough debridement. Preoperative x-rays may be helpful where air or lead-based paints can be seen. It should be noted that amputation rates following high-pressure injection injuries are reported as being over 45%. Delayed or conservative treatment is therefore inappropriate.

CHRONIC WOUNDS

A chronic wound may be defined as one that fails to heal in the expected time for a wound of that type, which is usually less than 3 weeks. Delays in healing can occur at any phase but most often occur in the inflammatory phase.

Leg ulcers

In resource-rich countries, the most common chronic wounds are leg ulcers. An ulcer can be defined as a break in the epithelial continuity. A prolonged inflammatory phase leads to overgrowth of granulation tissue, and attempts to heal by scarring leave a fibrotic margin. Necrotic tissue, often at the ulcer centre, is called slough. The more common aetiologies are listed in *Summary box 3.4*.

A chronic ulcer, unresponsive to dressings and simple treatments, should be biopsied to rule out neoplastic change, a squamous cell carcinoma known as a Marjolin's ulcer being the most common. Effective treatment of any leg ulcer depends on treating the underlying cause, and diagnosis is therefore vital. Arterial and venous circulation should be assessed, as should sensation throughout the lower limb. Surgical treatment is only indicated if non-operative treatment has failed or if the patient suffers from intractable pain. Meshed skin grafts (**Figure 3.7**) are more successful than sheet grafts and have the advantage of allowing mobilisation, as any tissue exudate can escape through the mesh. It should be stressed that the recurrence rate is high in venous ulceration, and patient compliance with a regime of hygiene, elevation and elastic compression is essential.

Summary box 3.4

Aetiology of leg ulcers

- Venous disease leading to local venous hypertension (e.g. varicose veins)
- Arterial disease, either large vessel (atherosclerosis) or small vessel (diabetes)
- Arteritis associated with autoimmune disease (rheumatoid arthritis, lupus, etc.)
- Trauma – could be self-inflicted
- Chronic infection – tuberculosis/syphilis
- Neoplastic – squamous or basal cell carcinoma, sarcoma

Pressure sores

These can be defined as tissue necrosis with ulceration due to prolonged pressure. Less preferable terms are bed sores, pressure ulcers and decubitus ulcers. They should be regarded as preventable but occur in approximately 5% of all hospitalised patients (range 3–12% in published literature). There is a higher incidence in paraplegic patients, in the elderly and in the severely ill patient. The most common sites are listed in *Summary box 3.5*.

A staging system for description of pressure sores devised by the American National Pressure Ulcer Advisory Panel is shown in *Table 3.2*.

Summary box 3.5

Pressure sore frequency in descending order

- Ischium
- Greater trochanter
- Sacrum
- Heel
- Malleolus (lateral then medial)
- Occiput

TABLE 3.2 Staging of pressure sores.

Stage	Description
1	Non-blanchable erythema without a breach in the epidermis
2	Partial-thickness skin loss involving the epidermis and dermis
3	Full-thickness skin loss extending into the subcutaneous tissue but not through underlying fascia
4	Full-thickness skin loss through fascia with extensive tissue destruction, maybe involving muscle, bone, tendon or joint

If external pressure exceeds the capillary occlusive pressure (over 30 mmHg), blood flow to the skin ceases, leading to tissue anoxia, necrosis and ulceration (**Figure 3.9**). Prevention

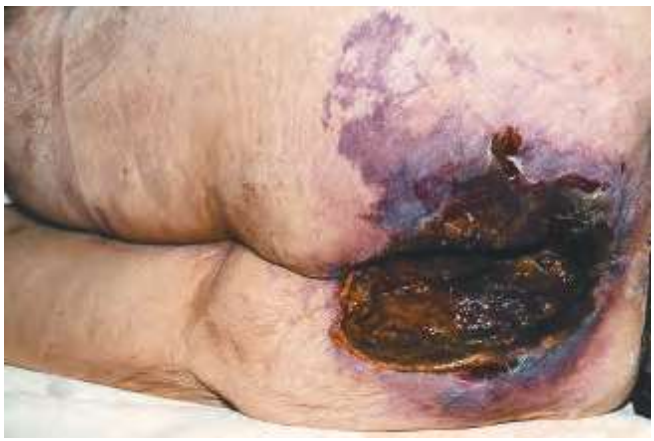


Figure 3.9 Pressure ulcer.

Jean-Nicholas Marjolin, 1780–1850, surgeon, Paris, France, described the development of carcinomatous ulcers in scars in 1828.

is obviously the best treatment, with good skin care, special pressure dispersion cushions or foams, the use of low air loss and air-fluidised beds and urinary or faecal diversion in selected cases. Pressure sore awareness is vital, and the bed-bound patient should be turned at least every 2 hours, with the wheelchair-bound patient being taught to lift themselves off their seat for 10 seconds every 10 minutes. It should be stressed that the most important treatment is to treat the cause of the pressure sore and that surgical treatment is a last resort often doomed to failure if the cause persists.

Surgical management of pressure sores follows the same principles involved in acute wound treatment (*Summary box 3.4*). The patient must be well motivated, clinically stable with good nutrition and adhere to the preventative measures advised postoperatively. Preoperative management of the pressure sore involves adequate debridement, and the use of vacuum-assisted closure (VAC) may help to provide a suitable wound for surgical closure (see below). The aim is to fill the dead space and to provide durable sensate skin. Large skin flaps that include muscle are best and, occasionally, an intact sensory innervated area can be included (e.g. extensor fascia lata flap with lateral cutaneous nerve of the thigh). If possible, use a flap that can be advanced further if there is recurrence and that does not interfere with the planning of neighbouring flaps that may be used in the future.

Vacuum-assisted closure

This is now more correctly known as negative pressure wound closure. Applying intermittent negative pressure of approximately −125 mmHg appears to hasten debridement and the formation of granulation tissue in chronic wounds and ulcers. A foam dressing is cut to size to fit the wound. A perforated wound drain is placed over the foam, and the wound is sealed with a transparent adhesive film. A vacuum is then applied to the drain (**Figure 3.10**). Negative pressure may act by decreasing oedema, by removing interstitial fluid and by increasing blood flow. As a result, bacterial counts decrease and cell proliferation increases, thereby creating a suitable bed for graft or flap cover.

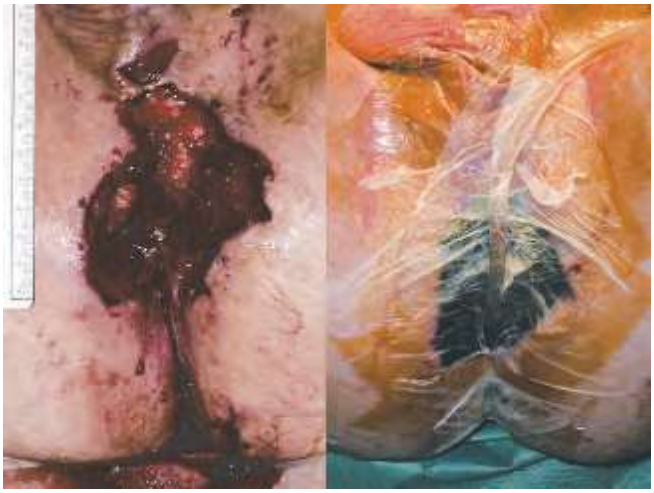


Figure 3.10 Vacuum-assisted closure dressing of a large wound.

NECROTISING SOFT-TISSUE INFECTIONS

These are rare but often fatal. They are most commonly polymicrobial infections with Gram-positive aerobes (*Staphylococcus aureus*, *S. pyogenes*), Gram-negative anaerobes (*Escherichia coli*, *Pseudomonas*, *Clostridium*, *Bacteroides*) and beta-haemolytic *Streptococcus*. There is usually a history of trauma or surgery with wound contamination. Sometimes, the patient's own defence mechanisms may be deficient. These infections are characterised by sudden presentation and rapid progression. The fact that deeper tissues are involved often leads to a late or missed diagnosis (**Figure 3.11**). Clinical signs are shown in *Summary box 3.6*.

There are two main types of necrotising infections: clostridial (gas gangrene) and non-clostridial (streptococcal gangrene and necrotising fasciitis). The variant of necrotising fasciitis with toxic shock syndrome results from *Streptococcus pyogenes* and is often called the 'flesh-eating bug' in this situation. Treatment consists of appropriate antibiotics with wide surgical excision. Tissue biopsies are essential for histological

Summary box 3.6

Signs and symptoms of necrotising infections

- Unusual pain
- Oedema beyond area of erythema
- Crepitus
- Skin blistering
- Fever (often absent)
- Greyish drainage ('dishwater pus')
- Pink/orange skin staining
- Focal skin gangrene (late sign)
- Shock, coagulopathy and multiorgan failure

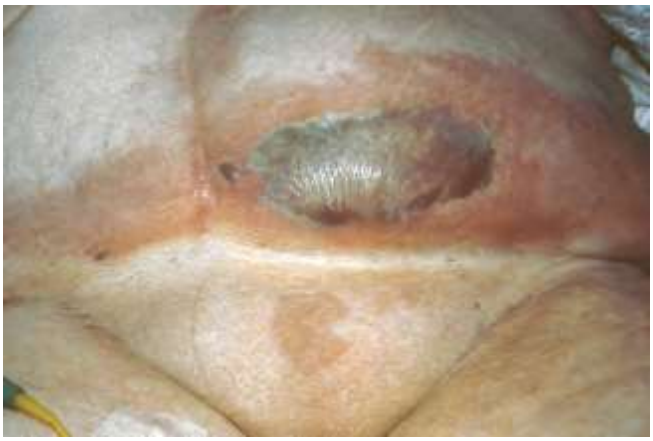


Figure 3.11 Necrotising fasciitis of the anterior abdominal wall.

Hans Christian Joachim Gram, 1853–1938, Professor of Pharmacology (1891–1900) and of Medicine (1900–1923), Copenhagen, Denmark, described this method of staining bacteria in 1884.

diagnosis and culture to obtain appropriate antibiotic sensitivity information. The raw areas resulting from excision often require skin grafting. Treatment is surgical excision, with tissue biopsies being sent for culture and diagnosis. Wide raw areas requiring skin grafting often result.

SCARS

The maturation phase of wound healing has been discussed above and represents the formation of what is described as a scar. The immature scar becomes mature over a period lasting a year or more, but it is at first pink, hard, raised and often itchy. The disorganised collagen fibres become aligned along stress lines with their strength being in their weave rather than in their amount (this has been compared with steel wool being slowly woven into a cable). As the collagen matures and becomes denser, the scar becomes almost acellular as the fibroblasts and blood vessels reduce. The external appearance of the scar becomes paler, while the scar becomes softer, flattens and its itchiness diminishes. Most of these changes occur over the first three months but a scar will continue to mature for one to two years. Tensile strength will continue to increase but would not be expected to exceed 60–80% that of normal skin.

Scars are often described as being atrophic, hypertrophic and keloid. An atrophic scar is pale, flat and stretched in appearance, often appearing on the back and in areas of tension. It is easily traumatised as the epidermis and dermis are thinned. Excision and resuturing may only rarely improve such a scar.

A hypertrophic scar is defined as excessive scar tissue that does not extend beyond the boundary of the original incision or wound. It results from a prolonged inflammatory phase of wound healing and from unfavourable scar siting (i.e. across the lines of skin tension). In the face, these are known as the lines of facial expression.

A keloid scar is defined as excessive scar tissue that extends beyond the boundaries of the original incision or wound **(Figure 3.12)**. Its aetiology is unknown, but it is associated with elevated levels of growth factor, deeply pigmented skin, an inherited tendency and certain areas of the body (e.g. a triangle whose points are the xiphisternum and each shoulder tip).

The histology of both hypertrophic and keloid scars shows excess collagen with hypervascularity, but this is more marked in keloids where there is more type III collagen.

The treatment of both hypertrophic and keloid scars is difficult and is summarised in *Summary box 3.7*.

Hypertrophic scars improve spontaneously with time, whereas keloid scars do not.

Summary box 3.7

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[a]
- Excision and postoperative radiation (external beam or brachytherapy)[a]
- Intralesional excision (keloids only)
- Laser – to reduce redness (which may resolve in any event)
- Vitamin E or palm oil massage (unproven)

[a]All excisions are associated with high rates of recurrence.

AVOIDABLE SCARRING

If an acute wound has been managed correctly (see *Summary box 3.3*), most of the problems described above should not occur. However, the surgeon should always stress to the patient that there will be a scar of some description after wounding, be it planned or accidental. A dirt-ingrained (tattooed) scar is usually preventable by proper initial scrubbing and cleansing of the wound **(Figure 3.13)**. Late treatment may require excision of the scar or pigment destruction by laser.

Mismatched or misaligned scars result from a failure to recognise normal landmarks, such as the lip vermilion/white roll interface, eyelid and nostril free margins and hair lines such as

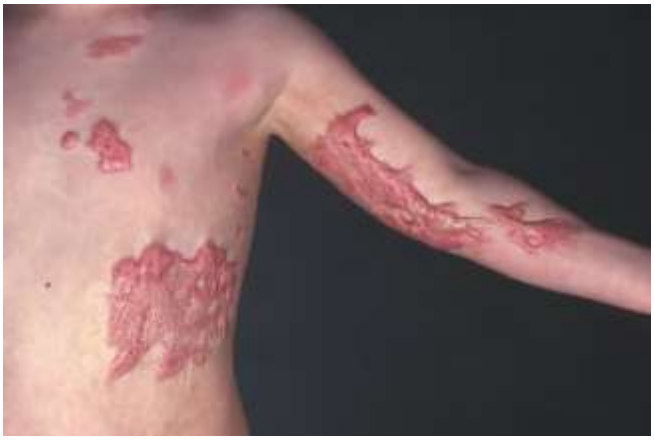


Figure 3.12 Multiple keloid scars.

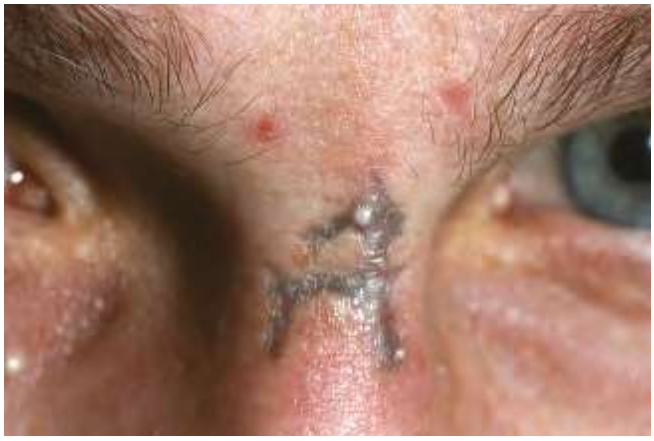


Figure 3.13 Dirt-ingrained scar.

Laser is an acronym for **L**ight **A**mplification by **S**timulated **E**mission of **R**adiation. A laser is an intense beam of monochromatic light.

those relating to eyebrows and moustache. Treatment consists of excision and resuturing.

Poorly contoured scars can be stepped, grooved or pincushioned. Most are caused by poor alignment of deep structures such as muscle or fat, but trapdoor or pincushioned scars are often unavoidable unless the almost circumferential wound can be excised initially. Late treatment consists of scar excision and correct alignment of deeper structures or, as in the case of a trapdoor scar, an excision of the scar margins and repair using W or Z-plasty techniques.

Suture marks may be minimised by using monofilament sutures that are removed early (3–5 days). Sutures inserted under tension will leave marks. Wounds can be strengthened post suture removal by the use of sticky strips. Fine sutures (6/0 or smaller) placed close to the wound margins tend to leave less scarring. Subcuticular suturing avoids suture marks either side of the wound or incision.

CONTRACTURES

Where scars cross joints or flexion creases, a tight web may form restricting the range of movement at the joint. This may be referred to as a contracture and can cause hyperextension or hyperflexion deformity (**Figure 3.14**). In the neck, it may interfere with head extension (**Figure 3.15**). Treatment may be simple involving, multiple Z-plasties (**Figure 3.16**), or more complex, requiring the inset of grafts or flaps. Splintage and intensive physiotherapy are often required postoperatively.

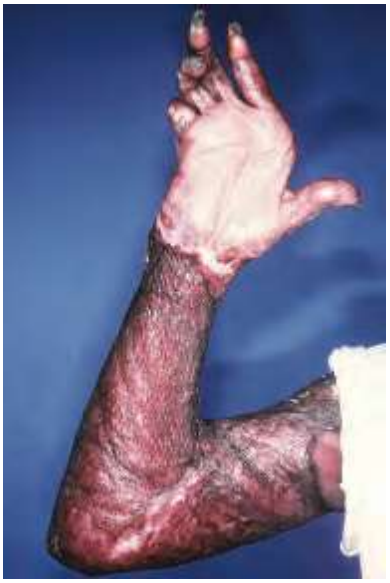


Figure 3.14 Burn contractures showing hyperextended fingers and hyperflexed elbow.

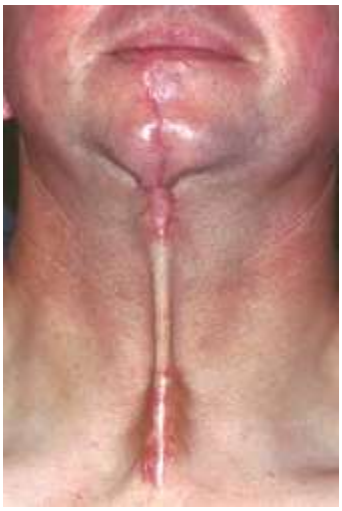


Figure 3.15 Post-traumatic (chainsaw) midline neck contracture.

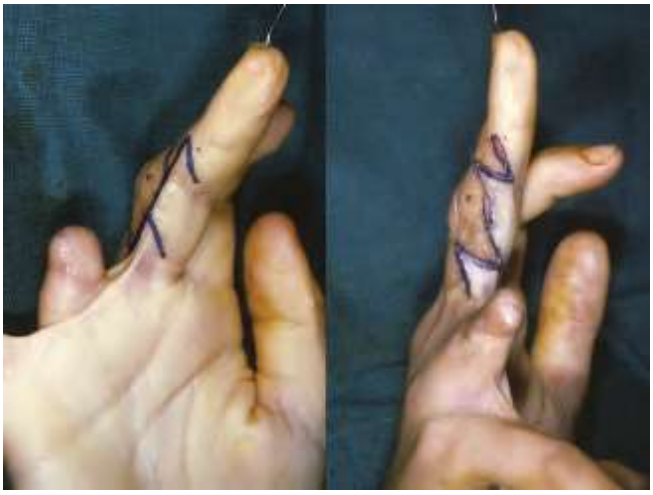


Figure 3.16 Multiple Z-plasty release of finger contracture.

FURTHER READING

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