

3 Wound healing and tissue repair

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ABNORMAL WOUND HEALING

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Various factors can adversely affect wound healing (Summary box 3.1). Some wounds fail to heal in a timely and orderly manner, resulting in chronic non-healing wounds, significant morbidity and poor cosmesis. On the other hand, Summary box 3.1 Local and systemic factors influencing wound healing /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF , described these nodes in 1878.

Local Skin tension Hypoxia and ischaemia Vascular insufficiency Lymphoedema Contamination Infection Presence of foreign bodies Radiotherapy Systemic Advancing age Obesity Malnutrition . Then, Smoking Diseases (e.g. diabetes mellitus, connective tissue diseases)

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inflammation can result in excessive scar tissue, for example hypertrophic and keloid scars. These abnormal scars contain excess collagen, which is arranged in a disorganised pattern in keloid scars as opposed to a parallel pattern in hypertrophic scars. Hypertrophic scars do not extend beyond the boundary of the original incision or wound and eventually regress. They are more common in areas of increased tension, wounds crossing tension lines, deep dermal burns and wounds left to heal by secondary intention (longer than 3 weeks). Keloid scars extend beyond the boundaries of the original incision or wound (Figure 3.5), do not spontaneously regress and are difficult to treat. The aetiology is unknown but genetic predisposition is implicated. They often occur as a result of relatively minor trauma and mainly in those with darker skin pigmentation.

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ACUTE WOUNDS Bites

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Most bites involve either puncture wounds or avulsions. Wounds over the metacarpophalangeal joint should be treated as a human bite following a punch to the mouth until proven Victor-Auguste-François Morel-Lavallée , 1811–1865, surgeon who first described this lesion in 1863. Summary box 3.4 Reconstruction options for wound closure /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF otherwise. Joint infections are a surgical emergency as they can result in articular cartilage destruction. They present as hot, swollen and tender joints with a limited range of motion.

Figure 3.11 Degloving buttock injury. Primary closure Full-thickness skin graft Secondary closure Dermal matrices 78 : Tertiary (delayed primary) Local, regional or pedicled closure /f_l ap NPWT Tissue expansion Split-thickness skin graft Free /f_l ap

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Acute compartment syndrome

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Acute compartment syndrome occurs when there is increased interstitial pressure within a closed osteofascial compartment, which results in microvascular compromise. It is a surgical emergency as delayed treatment may lead to irreversible muscle ischaemia and significant long-term morbidity. Compartment syndrome most commonly occurs after lower limb fractures, both open and closed (see Chapter 32). It also occurs in the upper limb, buttock and abdomen. Other causes include soft-tissue trauma, arterial injuries, burns and prolonged compression. It is characterised by pain out of proportion to the injury, particularly with passive movement of the affected compartment muscles. Paraesthesia is another early sign. Absent pulses are uncommon and suggest the possibility of vascular injury. Compartment syndrome is generally a clinical diagnosis. It can be difficult to diagnose in the presence of impaired consciousness, in children and in patients with regional nerve blocks. Monitoring intracompartment pressures (ICPs) can sometimes help to guide management. A pressure of ≤ 30 mmHg between the diastolic pressure and ICP has been recommended as the threshold for fasciotomy. Fasciotomy involves incising the skin and deep fascia with long axial incisions (Figure 3.13). If the compartment pressure was high, the muscle will then be seen bulging out through the fasciotomy opening. The lower limb is reliably decompressed via two incisions. A medial longitudinal incision 1–2 cm posterior to the medial border of the tibia decompresses the superficial and deep posterior compartments. A lateral longitudinal incision 2 cm lateral to the anterior tibial border decompresses the peroneal and anterior compartments. Late diagnosis of compartment syndrome is a management dilemma as a late fasciotomy may result in rhabdomyolysis, infection, need for amputation and even death.

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Figure 3.15 Pressure ulcer.

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Figure 3.5 Multiple keloid scars. TABLE 3.1 US Centers for Disease Prevention and Control surgical wound classification. Class I Uninfected operative wounds No inflammation is encountered Clean Respiratory, alimentary, genital or uninfected urinary tracts are not entered Primarily closed and, if necessary, drained using a closed system Respiratory, alimentary, genital or urinary Class II tracts are entered under controlled conditions Clean- and without unusual contamination contaminated No evidence of infection or major break in technique is encountered Class III Open, fresh, accidental wounds Operations with major breaks in sterile Contaminated technique (e.g. open cardiac massage) or gross spillage from the gastrointestinal tract Incisions in which acute, non-purulent inflammation is encountered Class IV Old traumatic wounds with retained devitalised tissue and those that involve Dirty existing clinical infection or perforated viscera

American Society of Anesthesiologists score ≥ 3 ; operative time longer than the expected duration for similar procedures (>75th percentile). Summary box 3.3 Synopsis of wound classification systems

Aetiology Complexity Clean, surgical Simple Shearing or degloving Complex Crush Significant soft-tissue loss Blast Open fracture or joint Burn (thermal, electrical, chemical, radiation, Visceral involvement mechanical) Complicated Cold injury Infection Avulsion or traction Necrosis Low or high energy Haematoma Bite Gas gangrene Depth Compartment syndrome Epidermal Dermal (superficial or Chronic deep) Vascular ulcers (venous or arterial) Full thickness Contamination Pressure ulcers Clean Diabetic ulcers Clean-contaminated Contaminated Dirty Implant or non-implant

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Contractures

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Scar contractures can cause severe functional, psychological and aesthetic problems (Figure 3.19). Contractures across joints may restrict the range of movement, leading to deformity , - impairment and disability . Contractures may also result from the differential growth pattern between scar and surrounding tissues. - Surgical contracture release and reconstruction can be an effective treatment option. A key principle is the replacement of scar tissue with healthy tissue. A wide range of reconstructions are described (Summary box 3.4) and typically involve 14 skin grafts or flaps (more information can be found in Part 6). - Local flaps such as Z-plasty (Figure 3.20) and its variants can be used to lengthen and transpose the scar. Many other local flaps have been described, including Y-V , V-Y and W-plasty . Free flaps may be required for resurfacing severe contractures. In general, flaps are preferable to skin grafts because of - graft contracture. When skin grafts are used, full thickness is preferred to split thickness as they have a better texture and contract less during healing.

(red/raised) (dark/raised) Intralesional corticosteroids Silicone gel/sheeting + a intralesional corticosteroids 5-FU + intralesional corticosteroids b Fractional or pulsed-dye laser therapy Patient counselling regarding recurrence rate and expectations Surgical excision with adjuvant • Silicone gel or sheeting or intralesional corticosteroids or both • Radiotherapy • Alternative therapies (bleomycin, mitomycin C, imiquimod) Figure 3.18 Management algorithm for keloids. Light grey indicates initial management strategies; dark grey indicates secondary management options. Cryotherapy may be used in conjunction with intralesional corticosteroids, depending on physician experience and comfort with its application. Ablative fractional lasers are the preferred initial laser therapy option for patients with minor keloids. 5-FU, 5-fluorouracil. (Redrawn with permission from Gold MH, McGuire M, Mustoe TA et al . Updated international clinical recommendations on scar management: part 2-algorithms for scar prevention and treatment. Dermatol Surg 2014; 40 (8): 825-31.)

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Degloving

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Degloving is the avulsion of skin and subcutaneous fat from the underlying fascia, muscle or bone. A degloving injury may be open or closed. An example of an open degloving is a finger avulsion injury with loss of skin (Figure 3.10). Closed degloving injuries result from shearing forces, which may occur with motor vehicle collisions. The extent of these injuries is often underappreciated and much of the skin perforating may be non-viable (Figure 3.11). Disruption of vascular and lymphatic vessels may result in a characteristic - haemolymphatic collection between the fascial planes called a Morel-Lavallée lesion (Figure 3.12). Although these lesions were originally described as occurring over the greater trochanter, the term is also used now for similar lesions in other anatomical locations. Assessing the viability of degloved tissue can be difficult and may therefore require more than one surgical exploration before definitive reconstruction. Non-viable and debrided skin may show fixed staining and thrombosis of subcutaneous skin may show fixous veins. Most surgeons serially excise the degloved skin until punctate dermal bleeding is seen from viable tissue. Intravenous fluorescein may also help delineate it requires specialist equipment and there is a small risk of anaphylaxis. More recently, the use of indocyanine green fluorescence has been reported. A useful classification system to help guide management describes four patterns of degloving (Summary box 3.5 severe multiplanar degloving. Summary box 3.5 9 Classification of degloving injuries in limb trauma

Subcutaneous fat Superficial fascia Deep fascia Muscle Bone Figure 3.12 Mechanism of injury for Morel-Lavallée lesions. Cross-sectional illustrations of the layers of tissue from the skin to the bone demonstrate how a shearing force can cause the comparatively mobile subcutaneous tissues to move relative to the comparatively fixed underlying deep fascia, causing shearing of perforating arteries (red), veins (blue) and lymphatics (green) and ultimately leading to the formation of a haemolymphatic collection in this potential space. (Adapted with permission from Bonilla-Yoon I, Masih S, Patel DB et al. The Morel-Lavallée lesion: pathophysiology, clinical presentation, imaging features, and treatment options. Emerg Radiol 2014; 35-43.) Figure 3.13 Fasciotomy of the leg. 1 Limited degloving with abrasion or avulsion 2 Non-circumferential degloving 3 Circumferential single plane degloving 4 Circumferential multiplanar degloving

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Degloving is the avulsion of skin and subcutaneous fat from the underlying fascia, muscle or bone. A degloving injury may be open or closed. An example of an open degloving is a finger avulsion injury with loss of skin (Figure 3.10). Closed degloving injuries result from shearing forces, which may occur with motor vehicle collisions. The extent of these injuries is often underappreciated and much of the skin perforating may be non-viable (Figure 3.11). Disruption of vascular and lymphatic vessels may result in a characteristic haemolymphatic collection between the fascial planes called a Morel-Lavallée lesion (Figure 3.12). Although these lesions were originally described as occurring on the hand, the term is also used now for similar lesions in other anatomical locations. Assessing the viability of degloved tissue can be difficult and may therefore require more than one surgical exploration or definitive reconstruction. Non-viable and debridement bed stained and thrombosis of subcutaneous skin may show fixous veins. Most surgeons serially excise the degloved skin until punctate dermal bleeding is seen from viable tissue. Intravenous fluorescein may also help delineate it requires specialist equipment and there is a small risk of anaphylaxis. More recently, the use of indocyanine green fluorescence has been reported. A useful classification system to help guide management describes four patterns of degloving (Summary box 3.5 severe multiplanar degloving. Summary box 3.5 9 Classification of degloving injuries in limb trauma

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Haemostasis

Haemostasis

Disruption of the vascular endothelium following injury causes vasoconstriction and exposure of the subendothelial extracellular matrix. This encourages platelets to adhere, activate and aggregate, resulting in a platelet plug, which also helps limit further blood loss. Platelet adhesion results in their activation and release of granules. Alpha granules contain hundreds of proteins, including cytokines and growth factors; for example, transforming growth factor beta, platelet-derived growth factor, fibroblast growth factor, epidermal growth factor and vascular endothelial growth factor. These are involved in the deposition of extracellular matrix, chemotaxis, epithelialisation and the formation of new blood vessels (angiogenesis). Platelet aggregation occurs once platelets become activated. At the same time, tissue factor at the site of injury initiates the coagulation cascade (Figure 3.2), resulting in the formation of thrombin. Thrombin performs various functions, including fibrin generation, which helps to stabilise the platelet plug and form a scaffold for infiltrating cells .

The principles of wound management • The principles of scar management •

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Introduction

INTRODUCTION

Wound healing is a complex and dynamic biological process. In human adults, the normal response to injury across all organ systems typically results in fibrosis and scar formation. Fibrotic healing causes tissue dysfunction and its potential impact on patients is often underappreciated. This contrasts with early gestation when fetal tissues can remarkably heal without fibrosis.

Regenerative medicine is therefore an exciting field of research. A better understanding of the mechanisms involved can potentially help reduce the global burden of disease associated with wound healing. This chapter describes the pathophysiology of wound healing, the types of healing and how to classify wounds. Clinical judgement is crucial in managing wounds. A framework is provided to better understand the key principles of wound and scar management.

Inflammation

Inflammation

- ◦ In the early inflammatory phase (days 1–2), platelet activation causes an influx of inflammatory cells led by polymorpho - nuclear leukocytes, particularly neutrophils. The latter are important for minimising bacterial contamination of the wound. Platelets and the local injured tissue also release vasoactive amines such as histamine and serotonin, which increase vascular permeability , thereby aiding infiltration of inflammatory cells. During the late inflammatory phase (days 2–3) monocytes appear in the wound and di ff erentiate into macrophages. Macrophages play a vital role in wound healing. They func - tion as phagocytic cells and release proteolytic enzymes to help debride the wound. T hey are also the primary producer of cytokines and growth factors promoting fibroblast proliferation and angiogenesis. Historically , this phase has been described by rubor (red - ness), tumor (swelling), calor (heat) and dolor (pain). - Inflammation
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Learning objectives

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To understand: Normal wound healing and how it can be adversely • affected Types of healing and how to classify wounds • Learning objectives

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Leg ulcers

Leg ulcers

12 In developed 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 centre of the ulcer, is called slough. The more common aetiologies are listed in Summary box 3.7. A chronic ulcer that is 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. Summary box 3.7 Aetiology of leg ulcers

Vascular (venous, arterial, mixed) Trauma (bites, self-inflicted, burns) Infection (bacterial, fungal, mycobacterial, syphilis) Metabolic disorders (diabetes mellitus, gout, calciphylaxis) Autoimmune disorders (vasculitis, systemic sclerosis, rheumatoid arthritis) Neoplastic (squamous cell carcinoma, basal cell carcinoma)

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NORMAL HEALING IN OTHER SPECIFIC TISSUES Bone

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Bone healing occurs in similar phases to those for skin but with some differences (Figure 3.3). Most fractures heal by callus formation, which involves intramembranous and endo - chondral ossification. This is known as indirect or secondary bone healing and typically occurs in non-operative fracture management. A haematoma forms at the fracture site and - there is an inflammatory response. The fracture haematoma is gradually replaced by a soft callus. This fibrocartilage callus then undergoes endochondral ossification to form hard callus, which is woven bone helping to stabilise the fracture. Intramembranous ossification also occurs directly adjacent to the distal and proximal fracture ends. Hard callus formation is

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Schematic representation of the
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Necrotising fasciitis

Necrotising fasciitis

This is a severe, rapidly progressing infection of the soft tissue and fascia associated with significant morbidity and mortality (Figure 3.14). Reported mortality rates vary widely but a Danish nationwide cohort study including over 1500 patients found 30-day and 1-year mortality rates of up to 26% and 10–40%, respectively . The infection is commonly polymicrobial but monomicrobial presentation with *Streptococcus pyogenes* (group A streptococcus) is also frequent. Examples of other organisms include *Staphylococcus aureus* , *Escherichia coli* , *Pseudomonas* , *Clostridium* and *Bacteroides* . There is usually a history of trauma or surgery with wound contamination. Diabetes mellitus is the most common comorbidity , although up to 30% of patients may not have any comorbidities. Clinical features are shown in Summary box 3.6 . A scoring system to aid clinical decision making has been developed, but its performance remains questionable. remains primarily a clinical diagnosis and surgical treatment should not be delayed if suspicion is high. Treatment consists of appropriate intravenous antibiotics with urgent radical surgical debridement. A second look operation is usually planned in 24–48 hours depending on clinical response. Multiple debridements may be required. Summary box 3.6 Signs and symptoms of necrotising fasciitis

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Figure 3.14 Necrotising fasciitis of the anterior abdominal wall. Local Unusual pain Erythema, oedema, warmth Crepitus Blisters, bullae Greyish drainage ('dishwater pus') Fixed staining Necrosis, gangrene Systemic Fever, tachycardia, tachypnoea Shock Coagulopathy Multiorgan failure

Necrotising fasciitis

This is a severe, rapidly progressing infection of the soft tissue and fascia associated with significant morbidity and mortality (Figure 3.14). Reported mortality rates vary widely but a Danish nationwide cohort study including over 1500 patients found 30-day and 1-year mortality rates of up to 26% and 10–40%, respectively . The infection is commonly polymicrobial but monomicrobial presentation with *Streptococcus pyogenes* (group A streptococcus) is also frequent. Examples of other organisms include *Staphylococcus aureus* , *Escherichia coli* , *Pseudomonas* , *Clostridium* and *Bacteroides* . There is usually a history of trauma or surgery with wound contamination. Diabetes mellitus is the most common comorbidity , although up to 30% of patients may not have any comorbidities. Clinical features are shown in Summary box 3.6 . A scoring system to aid clinical decision making has been developed, but its performance remains questionable. remains primarily a clinical diagnosis and surgical treatment should not be delayed if suspicion is high. Treatment consists of appropriate intravenous antibiotics with urgent radical surgical debridement. A second look operation is usually planned in 24–48 hours depending on clinical response. Multiple debridements may be required. Summary box 3.6 Signs and symptoms of necrotising fasciitis

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Post exposure management for Tetanus Prone Wounds

Post exposure management for Tetanus Prone Wounds

underappreciated and is not as simple as is often perceived. The end points of surgical debridement can sometimes be difficult to determine. Healthy subcutaneous fat is yellow and soft. Muscle viability is judged by its colour, capacity to bleed and contractility. Contaminated, complex and complicated wounds often require more than one surgical debridement before definitive repair and closure; for example, blast injuries and necrotising fasciitis (see Part 4). Other types of debridement are summarised in Table 3.4. All wounds should be irrigated at the first available opportunity to reduce bacterial contamination. This also allows better visualisation for wound assessment. Warm normal saline

Immunisation Status Clean wound

Those aged 11 years and over ,
who have received 1 with an
adequate priming course of
tetanus vaccine the last dose
within 10 years None required

Children aged 5-10 years who have received priming course and pre-school booster Children under

5 years who have received an adequate priming course
Received adequate priming course of tetanus 3 vaccine but last dose more than 10 years ago
None required
Children aged 5-10 years who have received an adequate priming course but no pre-school booster (Includes UK born after 1961 with history of accepting vaccinations)
Immediate
Not received adequate priming course 3 of tetanus vaccine reinforcing (Includes uncertain immunisation status and/or born before 1961)
dose of vaccine 1
Clean wounds

are defined as wounds less than six 3 At least three doses of tetanus vaccine at Patients who are severely hours old, non-penetrating with negligible tissue appropriate intervals. This definition of “adequate immunosuppressed may not be adequately damage. course” is for risk assessment of tetanus-prone protected against tetanus, despite having wounds only. The full UK schedule is five doses been fully immunised and additional booster 2 If TIG is not available, HNIG may be used as an of tetanus

containing vaccine. alternative.

Figure 3.6 Postexposure management for tetanus-prone wounds. (Redrawn with permission from

<https://www.gov.uk/government/publications/tetanus-prone-wounds-posters>. © Crown copyright 2019. 2019TET02 10K OCT 2019 (APS).

TABLE 3.4 Types of debridement. Excision of non-viable tissue using surgical Surgical instruments such as a scalpel, curette, scissors or rongeur until healthy bleeding occurs at the wound edges Non-selective debridement such as

using irrigation, Mechanical wet-to-dry dressings and hydrotherapy. Both non viable and viable tissue may be removed Using dressings such as hydrocolloids or Autolytic transparent /f_i lms to retain moisture and allow wound enzymes to selectively liquefy non-viable tissue Chemically liquefy necrotic tissue with enzymes Enzymatic using topical agents such as collagenase or papain-urea Medical-grade larvae of *Lucilia sericata* r elease Biological proteolytic and antimicr obial substances to remove necr otic

tissue. They also directly promote wound healing

	Immediate treatment	Later treatment
Tetanus prone	High risk tetanus prone	None required
None required	None required	Further doses as required to complete the recommended schedule
Immediate	One dose of Immediate reinforcing schedule (to reinforcing human tetanus vaccine ensure future dose of immunoglobulin immunity) in a different site	vaccine
Immediate	One dose of reinforcing human tetanus	reinforcing human tetanus
2	2	2

dose of immunoglobulin dose of immunoglobulin in a different site vaccine in a different site vaccine doses or treatment may be required.

•
Figure 3.7 Meshing a split-thickness skin graft. (Reproduced with permission of MA Healthcare Limited from Hili S, Wong KY, Stephens /uni00A0P. Pretibial lacerations. Br J Hosp Med 2017; 78 : C162-6.)

such as water and antiseptic solutions. Irrigation can also be performed with a soft brush or sponge to clear particulate matter prior to preoperative application of skin antiseptic preparation. Wounds should be explored to determine the extent of injury, including any damage to underlying neurovascular structures, tendons, joints and bones. Careful tissue handling and meticulous technique are important throughout. Repair of all damaged structures may be attempted once the wounds are clean. Repair of nerves and vessels should be performed under magnification using loupes or a microscope. Skin closure should always be without tension. Direct closure is not always possible and other reconstruction methods should be considered. Historically, the reconstructive ladder and its variants such as the reconstructive elevator have been used as a framework to consider the simplest means to achieve wound closure for the desired goal. Advances in technology and surgical techniques have led to ongoing adjustments of these frameworks. Although these frameworks do not guide the useful reminder of the options available (Summary box 3.4). - A skin graft has no inherent blood supply and is dependent - on a well-vascularised recipient site for survival and wound healing. Split-thickness skin grafts (Figure 3.7) consist of the epidermis and a small portion of dermis whereas full-thickness skin grafts consist of the epidermis and the majority of the dermis. A flap contains tissue with its intrinsic blood supply that is transferred from one part of the body (donor) to another (recipient). The blood supply of the flap therefore does not rely on the recipient site like a skin graft. A free flap contains - tissue with its vascular pedicle that is surgically detached and transferred from its original location to a distant recipient site (Figure 3.8). A microscope is used to perform microvascular anastomoses to connect the blood vessels in the free flap to blood vessels close to the recipient site. Since its development in the 1990s, negative-pressure wound therapy (NPWT) is now widely used. It is not a replacement for definitive wound closure but is a useful adjunct

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(Figure 3.9). Negative pressure helps draw the wound edges together, remove exudate, reduce oedema and promote granulation tissue formation. NPWT is not recommended in the setting of exposed vessels, malignancy, untreated osteomyelitis, necrotic tissue or non-enteric and unexplored fistulae.

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Post exposure management for Tetanus Prone Wounds

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Immunisation Status Clean wound

Those aged 11 years and over ,

who have received 1 with an

adequate priming course of

tetanus vaccine the last dose

within 10 years None required

Children aged 5-10 years who

have received priming course and

pre-school booster Children under 5 years who have received an adequate priming course Received adequate priming course of tetanus 3 vaccine but last dose more than 10 years ago None required Children aged 5-10 years who have received an adequate priming course but no pre-school booster (Includes UK born after 1961 with history of accepting vaccinations) Immediate Not received adequate priming course 3 of tetanus vaccine reinforcing (Includes uncertain immunisation status and/or born before 1961)

dose of vaccine 1 Clean wounds are defined as wounds less than six 3 At least three doses of tetanus vaccine at Patients who are severely hours old, non-penetrating with negligible tissue appropriate intervals. This definition of “adequate immunosuppressed may not be adequately damage. course” is for risk assessment of tetanus-prone protected against tetanus, despite having wounds only. The full UK schedule is five doses been fully immunised and additional booster 2 If TIG is not available, HNIG may

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substances to remove necrotic tissue. They also directly promote wound healing

Immediate treatment

Later treatment 1

Tetanus prone High risk tetanus prone

None required None

required Further doses as required to complete the recommended

Immediate One dose of Immediate reinforcing schedule (to reinforcing human tetanus 2 dose of vaccine ensure future dose of

immunoglobulin immunity) in a different site vaccine

Immediate

One dose of Immediate One dose of reinforcing human tetanus

reinforcing human tetanus 2 2
dose of immunoglobulin dose of
immunoglobulin in a different site
vaccine in a different site vaccine
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•
Figure 3.7 Meshing a split-thickness skin graft. (Reproduced with permission of MA Healthcare Limited from Hili S, Wong KY, Stephens P. Pretibial lacerations. Br J Hosp Med 2017; 78 : C162-6.)

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Immunisation Status Clean wound

Those aged 11 years and over ,
who have received 1 with an
adequate priming course of
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within 10 years None required
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have received priming course and pre-school booster Children under 5 years who have received an adequate priming course Received adequate priming course of tetanus 3 vaccine but last dose more than 10 years ago None required Children aged 5-10 years who have received an adequate priming course but no pre-school booster (Includes UK born after 1961 with history of accepting vaccinations) Immediate Not received adequate priming course 3 of tetanus vaccine reinforcing (Includes uncertain immunisation

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are de /f_i ned as wounds less than
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immunosuppressed may not be
adequately damage. course” is for
risk assessment of tetanus-prone
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immunised and additional booster

2 If TIG is not available, HNIG may be used as an alternative of tetanus containing vaccine.

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	Immediate treatment	Later treatment
Tetanus prone	High risk	Tetanus prone
None required	None required	Further doses as required to complete the recommended schedule
Immediate	One dose of Immediate reinforcing schedule (to reinforcing human tetanus 2 dose of vaccine ensure future dose of immunoglobulin immunity) in a different site	vaccine Immediate One dose of Immediate One dose

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vaccine in a different site vaccine
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Pressure ulcers

Pressure ulcers

Pressure ulcers occur over a bony prominence or under a medical or other device (Figure 3.15) . A number of similar classifications exist. The US National Pressure Injury Advisory injury' in its staging system (Table 3.5) to provide a more accurate description of injuries to both intact and ulcerated 13 skin. Pressure injuries should be regarded as preventable. There is a higher incidence in those who are severely ill, those who have impaired mobility or those with a significant loss of sensation. The most common sites are listed in Summary box 3.8 Summary box 3.8 Common sites for pressure injuries and ulcers /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Prevention starts with assessing risk using a validated score to support clinical judgement such as the Braden scale, Waterlow score or Norton risk assessment scale. Patients at risk of developing pressure injuries should have a skin assessment, regular repositioning every 2–4 hours and the use of pressure- redistributing devices as appropriate. Patients should receive education on self-care and risk factors need to be addressed, such as providing nutritional support for any deficiencies. The treatment of pressure ulcers should focus on patient optimisation especially any aspects of poor nutrition and ongoing poorly managed medical problems to address any risk factors. Preventative measures are used and debridement may be appropriate (Table 3.4) . Dressings should be chosen to create an optimum wound-healing environment and appropriate antibiotics given if there are signs of infection. Surgery is not first-line treatment and is only considered when the above measures have been fully implemented. Patients must also be well motivated and able to fully comply with postoperative measures. Surgical management of pressure sores follows some of the same principles described for wound management in Table 3.2 Barbara Braden , contemporary , developed the Braden scale with Nancy Bergstrom in 1987. Judy Waterlow , contemporary , developed the Waterlow score in 1985. Doreen Norton , 1922–2007, nurse, developed the Norton risk assessment scale in 1962. are likely to fail. In suitable patients, successful reconstruction options include the use of large fasciocutaneous or musculocutaneous flaps. 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. - .

TABLE 3.5 US National Pressure Injury Advisory Panel 13 staging of pressure injuries. Stage Description 1 Non-blanchable erythema of intact skin 2 Partial-thickness skin loss with exposed dermis 3 Full-thickness skin loss 4 Full-thickness skin and tissue loss Obscured full-thickness skin and tissue Unstageable full-thickness pressure injury Deep tissue pressure Persistent non-blanchable, deep red, injury maroon or purple discoloration Ischium Heel Greater trochanter Malleolus Sacrum Occiput

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Principles

Principles

Clinical judgement is crucial in managing wounds. Some general principles of wound management are summarised in Table 3.2. Antibiotic prophylaxis is needed for clean-contaminated, contaminated and dirty wounds. It may also be used in clean wounds when there is a high risk of infection or when the sequelae of infection are potentially disastrous. Tetanus prophylaxis should be given based on the type of wound (Table 3.3) and immunisation status (Figure 3.6). Debridement is essential to remove any devitalised tissue and foreign material from the wound. Non-viable tissue must be excised until healthy bleeding occurs at the wound edges. The importance of thorough debridement is often

TABLE 3.2 Principles of wound management.

Preparation	
Antibiotic prophylaxis	Tetanus prophylaxis
Adequate analgesia/anaesthesia	Wound irrigation
Wound Early debridement and irrigation	
Exploration	Repair structures
Haemostasis	Closure
Skin closure	

without tension Consider
reconstruction options Suture
choice Consider drains Optimal
dressings Follow-up Removal of
sutures/splints Physiotherapy
Monitoring for complications Scar
management 7 TABLE 3.3 Tetanus-
prone wounds. Tetanus-prone
wounds High-risk tetanus-prone
wounds Any tetanus-prone wound
with: Puncture-type injuries in a
contaminated environment Heavy
contamination, e.g. soil or manure
Bites Compound fractures Wound
requiring surgery with >6-hour
delay Containing foreign bodies

Wounds or burns with Extensive devitalised tissue systemic sepsis

Adapted from

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Principles

Clinical judgement is crucial in managing wounds. Some general principles of wound management are summarised in Table 3.2. Antibiotic prophylaxis is needed for clean-contaminated, contaminated and dirty wounds. It may also be used in clean wounds when there is a high risk of infection or when the sequelae of infection are potentially disastrous. Tetanus prophylaxis should be given based on the type of wound (Table 3.3) and immunisation status (Figure 3.6). Debridement is essential to remove any devitalised tissue and foreign material from the wound. Non-viable tissue must be excised until healthy bleeding occurs at the wound edges. The importance of thorough debridement is often

TABLE 3.2 Principles of wound
management. Preparation
Antibiotic prophylaxis Tetanus

prophylaxis Adequate
analgesia/anaesthesia Wound
irrigation Wound Early
debridement and irrigation
Exploration Repair structures
Haemostasis Closure Skin closure
without tension Consider
reconstruction options Suture
choice Consider drains Optimal
dressings Follow-up Removal of
sutures/splints Physiotherapy
Monitoring for complications Scar
management 7 TABLE 3.3 Tetanus-
prone wounds. Tetanus-prone
wounds High-risk tetanus-prone
wounds Any tetanus-prone wound

with: Puncture-type injuries in a
contaminated environment Heavy
contamination, e.g. soil or manure
Bites Compound fractures Wound
requiring surgery with >6-hour
delay Containing foreign bodies
Wounds or burns with Extensive
devitalised tissue systemic sepsis
Adapted from
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Proliferation

Proliferation

The proliferative phase starts around day 3 and lasts for 2–4 weeks. It consists mainly of fibroblast activity with the production of ground substance (glycosaminoglycans and - proteoglycans), collagen, angiogenesis and re-epithelialisation of the wound. The wound tissue formed in the early part of this phase is - called granulation tissue. It has a pink and granular appearance. In the later part of this phase, there is an increase in the tensile strength of the wound as a result of increased collagen synthesised by fibroblasts. Some fibroblasts differentiate into myofibroblasts, which are contractile cells. These play an important role in contraction to bring the edges of the wound together .

(a) Epithelial cell Sebaceous gland Fibrin clot Sweat duct gland Platelet Neutrophil Fibroblast Eschar
(b) Macrophage New blood vessel Granulation tissue Monocyte Figure 3.1 Classic stages of wound healing. (a) Inflammation. G, Werner S, Barrandon Y et al . Wound repair and regeneration.

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contracture from a chainsaw

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Figure 3.20 Multiple Z-plasty release of finger contracture.

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Remodelling

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The remodelling phase begins 2–3 weeks after injury and lasts for a year or more. This phase is characterised by maturation of collagen. Type III collagen, which is prevalent during proliferation, is replaced by stronger type I collagen until the normal skin ratio of 4:1 type I to type III collagen is re-established. The collagen becomes more cross-linked and uniformly aligned. This maturation of collagen leads to increased tensile strength in the wound, which is maximal 12 weeks post injury and represents approximately 80% of the uninjured skin strength. -

Hair Bacteria Epidermis Oxygen Dermis Subcutaneous layer Collagen Capillary (c) Collagen (b) Proliferation. (c) Remodelling. (Adapted by permission from Springer: Gurtner Nature 2008; 453 : 314–21. 2008).

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SCAR MANAGEMENT

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The remodelling and maturation phase of wound healing results in scar formation. The immature scar is at first pink, hard, raised and often itchy. As the collagen matures, 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 3 months but a scar will continue to mature over 1–2 years, and sometimes more. Tensile strength will continue to increase but would not be expected to exceed around 80% that of normal skin. There is well-established evidence for managing scars with pressure/compression therapy, silicone sheets and gels, intralesional corticosteroid injection and surgery. Other treatment modalities include massage therapy, psychological counselling, laser therapy, radiotherapy, cryosurgery and intralesional injection of other products. Prevention of adverse scar formation is better than treatment, so it is important to correctly manage wounds (Table 3.2). Optimal surgical management starts with careful planning, tissue handling and meticulous technique. For example, placing incisions along relaxed skin tension lesions where possible, and avoiding straight-line incisions across flexion creases. Early debridement reduces the risk of infection and allows for earlier wound closure. Dirt-ingrained (tattooed) scars are usually preventable by proper initial scrubbing and cleansing of the wound (Figure 3.16). It is important to recognise normal scar.

Figure 3.16 Dirt-ingrained scar.

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(red, slightly raised) (red/raised, itchy) Silicone gel or sheeting
Apply prevention algorithm (i.e. silicone gel or sheeting, hypoallergenic paper tape or onion extract cream) corticosteroid injection (repeat monthly) PDL If persists for >1 month, treat as a linear hypertrophic scar Pressure therapy a PDL or fractional laser therapy Surgical excision + postoperative silicone gel or sheeting Figure 3.17 Management algorithm for hypertrophic scars.

Light grey indicates initial management strategies; dark grey indicates secondary a b management options. Preferred initial option. 2.5–20 mg/mL (face); 20–40 mg/mL (body). include bleomycin, mitomycin C, laser therapy and cryotherapy. e stabilisation. Combination and alternative therapies include massage, physical therapy, corticosteroids, tension-relieving surgical intervention, excision, grafting or /f_l ap coverage, hydrocolloid dressings, antihistamines and laser therapy.

5-FU, 5-fluorouracil; PDL, pulsed-dye laser. (Redrawn with permission from Gold MH, McGuire M, Mustoe TA part 2-algorithms for scar prevention and treatment.

Dermatol Surg hypertrophic (red/raised) Admission to a specialty burn unit (2 months) Intralesional Silicone gel or sheeting, b pressure garments and/or onion extract cream a or fractional Fractional laser therapy laser therapy Combination or alternative e therapies Severe scars Surgical excision + corticosteroids 5-FU +

corticosteroids c or alternative therapies c Alternative therapy options for severe lesions d Scar prevention and treatment should not begin before epithelium and wound

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TYPES OF WOUND HEALING

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There are different types of healing (Summary box 3.2 Primary healing is also known as healing by first intention. This occurs when there is direct approximation of the wound edges and is the aim of treatment. When there are no adverse influences, these wounds heal well and leave the best scar. Delayed primary healing occurs when the wound edges are not opposed immediately , which may be necessary in contaminated or untidy wounds. After debridement of non-viable tissue and when the wound is clean, the wound edges may be surgically approximated. This is also called healing by tertiary intention. Secondary healing or healing by secondary intention occurs in wounds that are left open and allowed to heal by granulation, contraction and re-epithelialisation. Summary box 3.2 Classification of wound closure and healing /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Primary	Wound edges apposed	Normal healing	Minimal scar	Secondary	Wound left open	Heals by granulation, contraction and re-epithelialisation
		Increased in /f_I	ammation and proliferation	Poor		scar
Tertiary (delayed primary)	Wound initially left open	Edges apposed later when healing conditions favourable				

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		Increased in /f_I	ammation and proliferation	Poor		scar
Tertiary (delayed primary)	Wound initially left open	Edges apposed later when healing conditions favourable				

TYPES OF WOUND HEALING

There are different types of healing (Summary box 3.2 Primary healing is also known as healing by first intention. This occurs when there is direct approximation of the wound edges and is the aim of treatment. When there are no adverse influences, these wounds heal well and leave the best

scar. Delayed primary healing occurs when the wound edges are not opposed immediately, which may be necessary in contaminated or untidy wounds. After debridement of non-viable tissue and when the wound is clean, the wound edges may be surgically approximated. This is also called healing by tertiary intention. Secondary healing or healing by secondary intention occurs in wounds that are left open and allowed to heal by granulation, contraction and re-epithelialisation. Summary box 3.2 Classification of wound closure and healing /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Primary	Wound edges apposed	Normal healing	Minimal scar	Secondary	Wound left open	Heals by granulation, contraction and re-epithelialisation	Increased in /f_I ammation and proliferation	Poor scar	Tertiary (delayed primary)	Wound initially left open	Edges apposed later when healing conditions favourable
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Tendon

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. Intrinsic healing consists of vincular blood flow and synovial diffusion. Extrinsic healing depends on the formation of fibrous adhesions between the tendon and the tendon sheath. Early active mobilisation following tendon repairs prevents adhesions limiting the range of motion and therefore promotes the more desired intrinsic healing. At the same time, tendon repairs must be protected by splintage to avoid rupture. Tendon

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WOUND MANAGEMENT

Assessment

WOUND MANAGEMENT Assessment

Wound management is guided by the timing and mechanism of injury as well as factors affecting healing (Summary box 3.1 It is also important to assess the patient's ideas, concerns and expectations. Patient outcomes also rely on good postoperative compliance. Assess the patient using Advanced Trauma Life Support principles to first identify and treat life- and then limb-threatening conditions. Some wounds require a multidisciplinary approach; for example, the involvement of orthopaedic surgeons and plastic surgeons in managing complex open lower limb fractures. Assess the site, size, geometry and nature of any wounds. Look for signs of contamination, infection, swelling and pulsatile bleeding. Deformities may suggest underlying fractures or dislocations. Has there been skin loss or degloving? What structures are visible? Thorough irrigation of wounds will allow better visualisation. It is important to correlate the clinical examination with the mechanism of injury as seemingly innocuous wounds can lead to underestimation of tissue damage. For example, high-pressure injection injuries of the hand can cause of amputation. The injected substances can track proximally into the forearm. Urgent surgical exploration and debridement - is required. Before palpation, ensure that the patient has adequate analgesia or a local anaesthetic block. When possible, it is important to assess motor and sensory function before any local anaesthesia. Unless there are obvious muscle injuries, the purpose of testing specific muscle groups is to evaluate potential nerve or tendon injuries. Imaging is useful to exclude foreign bodies, fractures or dislocations where appropriate. WOUND MANAGEMENT Assessment

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