Wounds

Normal healing
The four stages of wound healing
When specialized tissue is destroyed it cannot be replaced, and a stereotyped response called repair then follows in four stages.

- **Haemostasis: immediate.** In response to exposed collagen, platelets aggregate at the wound and degranulate, releasing inflammatory mediators. Clotting and complement cascades activated. Thrombus formation and reactive vasospasm achieve haemostasis
- **Inflammation: 0-3 days.** Vasodilatation and increased capillary permeability allow inflammatory cells to enter wound, and cause swelling. Neutrophils amplify inflammatory response by release of cytokines; reduce infection by bacterial killing; and debride damaged tissue. Macrophages follow and secrete cytokines, growth factors, and collagenases. They phagocytose bacteria and dead tissue and orchestrate fibroblast migration, proliferation, and collagen production
- **Proliferation: 3 days-3 weeks.** Fibroblasts migrate into the wound and synthesize collagen. Specialized myofibroblasts containing actin cause wound contraction. Angiogenesis is stimulated by hypoxia and cytokines and granulation tissue forms
- **Remodelling: 3 weeks-1 year.** Re-orientation and maturation of collagen fibres increases wound strength.

Types of wound closure
If wounds are closed by means of sutures, then healing is said to occur by primary intention.
1. Primary intention
1. Primary closure. If a wound is closed at the time of surgery, or following trauma, this is called primary closure. This must only be performed on wounds that are clean and healthy. Unhealthy tissue at a wound edge, whether it is devitalized following trauma, or old scar tissue from previous surgery, must be debrided.
2. Delayed primary closure. Sometimes a wound is contaminated and yet controlled closure by sutures is desirable. The wound may be treated with antiseptic dressing and sutures placed after 3 or 4 days, by which time the wound should be free of potentially infecting organisms. This is delayed primary closure.
2. Secondary intention. If wounds are left alone to heal, healing is said to occur by granulation or secondary intention. Grossly contaminated or infected wounds are debrided and left to granulate. The process is that of normal wound healing, but this takes longer than in wounds that have been closed. Prominent scars may result.

Delayed Wound Healing

This problem can be anticipated in certain categories of patients whose tissue repair process may be compromised.

Causes of delayed wound healing

Intrinsic or local factors:

These include abnormalities within the wound that prevent normal wound healing. These factors include:

1) Ischemia and hypoxia. Atherosclerosis or local damage to vessels in the form of trauma or vasculitis causes ischemia and subsequent hypoxia in the wound. Hypoxia leads to impaired collagen synthesis, prevents fibroblast migration, and increases the susceptibility of the wound to infection.

2) Infection. Infection is considered to be present when the bacterial count of a quantitative tissue culture >10^5 organisms per gram of tissue.

3) The presence of foreign bodies and necrotic tissue .Their presence prolongs the inflammatory phase of wound healing until they are removed. Such factors also predispose a wound to infection.

4) Chronic venous insufficiency leads to persistent venous hypertension and chronic edema in the lower extremities. These factors in turn lead to pericapillary fibrosis, tissue ischemia, and the liberation of superoxide radicals, which are thought to result in delayed wound healing in extremities with chronic venous insufficiency.

5) Ionizing radiation to the wound leads to abnormal wound healing. Early manifestations include erythema, edema, and hyperpigmentation, but the chronic effects of tissue ischemia, atrophy, and fibrosis cause radiation-exposed wounds to enter a chronic course.

6) Edema. Acute swelling, especially around joints, can lead to skin breakdown and full-thickness skin loss.

7) The microenvironment of the chronic wound has been shown to be different from that of the acute wound in investigational settings. Studies have implicated a decrease in the endogenous levels of certain growth factors in wounds with impaired healing. Other studies have established that an imbalance in the synthesis and degradation of extracellular matrix proteins is central to the establishment and maintenance of chronic wounds. This occurs through inadequate synthesis of extracellular matrix proteins, increased degradative enzymes, decreased regulation of degradative enzymes, or a combination of these causes.

Extrinsic or systemic factors:

These also contribute to abnormal wound healing. These factors are primarily linked to the underlying general health of the patient.

1) Malnutrition alters normal healing through the indirect and the direct effects of vitamin and mineral deficiency.

2) Diabetes mellitus The lack of insulin (and its poorly understood trophic effects on healing tissues), hyperglycemia (by adversely affecting the migratory and phagocytic functions of inflammatory cells and the proliferation of fibroblasts and endothelial cells), neuropathy, and the vascular disease that occurs in diabetic patients all contribute to poor healing.

3) Steroids and antineoplastic drugs can markedly diminish the speed and quality of the healing process. The exact effects of steroids are not yet understood. Chemotherapeutic agents alter wound healing by decreasing mesenchymal cell proliferation and inducing a leukopenic state that reduces the inflammatory cells available for wound healing. Immunosuppression from AIDS or other diseases may also affect various phases of wound healing.

4) Smoking contributes to delayed wound healing by causing cutaneous vasoconstriction, decreasing the oxygen-carrying capacity of hemoglobin, and contributing to atherosclerosis.

5) Collagen vascular diseases The medicines that are used to treat collagen vascular diseases may impair cell migration and collagen deposition. Adjustment of dosage may lead to improved wound healing.

6) Cleansing agents [such as chlorhexidine gluconate or povidone-iodine or chemicals may impair wound healing by affecting cell migration.

7) Repetitive trauma, intentional or otherwise, from shearing or pressure forces often leads to a failure in healing.

8) Renal disease and liver diseases patients with renal and/or liver disease often heal their wounds more slowly.

Scars and adverse scar

The most superficial wounds such as superficial burns and abrasions will heal by epithelialisation alone without scar formation. In these circumstances adnexal structures are preserved and the epithelium regenerates from these structures. This may leave alterations in keratinisation, texture or pigmentation of the healed area, but not scarring as such.
A scar is the inevitable consequence of wound repair. The final phase of wound repair is the process of remodelling and scar maturation. The fibroblasts, capillaries, glycosaminoglycans, and immature collagen of granulation tissue and the newly healed wound are replaced by relatively acellular, avascular scar tissue composed of mature collagen with scattered

fibroblasts. This biological process is manifested by a change in appearance of the scar from a red, raised, firm, contracting, perhaps itchy nodule to a pale, flat, softer, static, symptomless plaque of mature scar. The rate at which any given scar passes through this process can vary widely depending on the age of the individual, the site of the wound, the time the wound took to heal, the direction of the scar and the tension across it. In general, scars in younger patients with wounds on the trunk that heal slowly, perhaps with infection or dehiscence, and scars that have a lot of tension across them will take much longer to mature than scars in older people, in thin-skinned areas, that heal rapidly by first intention and that have minimal tension across them.

Adverse scars
There are many types of adverse scar, many of which can be avoided or prevented by correct incision planning and adequate wound management. Some types, however, cannot be prevented and are unpredictable in their occurrence. The appearance of some scars can be improved by surgical or other means, but scars can never be removed totally.

Types of adverse scars
Wrong direction
Incisions that pass along ideal lines are more likely to leave acceptable scars. There are many types of 'lines of election' for incisions, most of which pass along skin wrinkles or along relaxed skin tension lines (that is a line along which maximal skin tension passes when the part is in a relaxed position). These lines have minimal tension across the wound edges. A scar which crosses these lines will have a greater tendency to stretch or become hypertrophic, and even if not hypertrophic will usually appear more conspicuous than one which follows a relaxed skin tension line. Other ideal positions for scars are at junctions between anatomical areas such as the nose and the cheek, the cheek and the ear or the junction between a hairy and hairless area

Poor alignment of features
Where a scar crosses the junction between distinct anatomical features, such as the vermillion of the lip, it is essential that these features are accurately realigned. Such misalignments result in conspicuous adverse scars.

Stretched scar
Scars from excisional wounds on the trunk and limbs often stretch. It has been shown that the width of a scar depends on the tension across the wound at the time of wound closure. Where tension cannot be avoided there is evidence that prolonged wound support with buried nonabsorbable or long-term absorbable sutures can minimise scar stretching.

Contracted scar
The process of wound contraction continues in the remodeling phase of scar maturation such that a scar will always be shorter than the incision from which it results. Where a linear scar crosses a flexor surface this shortening may result in a scar contracture which may prevent full extension of that part. This will occur on the flexor surface of a finger if a straight-line incision is used.

Curved or zigzag incisions will avoid this problem. Where scarring is extensive such as burn scars then scar contractures may be inevitable.

Pigment alteration
The new epidermis of a scar will often not have the same degree of pigmentation as surrounding unscarred areas. Most scars are hypopigmented, but hyperpigmentation can also occur.

Contour deformity
Where wound edges are not anatomically aligned in the vertical plane or where a bevelled cut is not repaired accurately there is a risk of contour irregularity in the healed scar. This can usually be avoided by accurate wound repair, if necessary excising bevelled edges to restore even vertical edges for repair.

Tattooing
In traumatic wounds it is possible for particles of grit, dirt or soot to become implanted in the wound as it heals. This results in tattooed scars where the particles of foreign material show through as blue or black discoloration of the scar. Adequate primary wound management can avoid this. Abrasions with ingrained dirt should be scrubbed with a stiff brush; more deeply tattooed wounds should be excised. Late correction of tattooed scars can be very difficult.

Stitch marks
If skin sutures are left in place for more than 7 days then scars from the stitch marks will usually result. This problem can be avoided by using subcuticular sutures wherever possible, removing skin sutures before 7 days and, where prolonged wound support is needed, supplementing skin sutures with subcuticular sutures allowing early removal of the skin sutures.

Hypertrophic scars
In some circumstances scars remain in the remodelling phase for longer than is usual. These hypertrophic scars are more cellular and more vascular than mature scars, there is increased collagen production and collagen breakdown, but the balance is such that excess collagen is produced. Clinically these scars are red, raised, itchy and tender. Such scars will eventually mature to become pale and flat, and it is this spontaneous resolution which distinguishes hypertrophic scars from keloid scars. Hypertrophic scars typically occur in wounds where healing was delayed, perhaps where complications such as infection or dehiscence occurred. They are more common in children and where skin tension is high such as the tip of the shoulder or any scar that runs across relaxed skin tension lines. The risk of developing a hypertrophic scar can be minimised by ensuring quiet primary healing. Where hypertrophy does occur patience is usually rewarded by improvement with time. Massage of the scar with moisturising cream or the application of pressure to the remodelling scar can accelerate the natural process of maturation. Revision of hypertrophic scars is appropriate where they cross skin tension lines or where a specific wound healing complication

occurred. In the absence of these factors scar revision should be avoided as it will usually be met with recurrence.

Keloid scars
In some situations there is an extreme overgrowth of scar tissue that grows beyond the limits of the original wound and shows no tendency to resolve. Keloid scars are biologically identical to hypertrophic scars that in turn are an extension of normal scar behaviour. Whilst it is usually possible to make the distinction between these scar types, they are best regarded as a spectrum of scar behaviour. They often occur in wounds that healed perfectly without complications. They are more common in certain sites such as the central chest, the back and shoulders and the ear-lobes. Many keloid scars are untreatable and surgical treatment as a single modality will usually be met with recurrence. Some keloid scars will improve with the application of pressure. Intralesional injections of steroids such as triamcinolone can be helpful. The best cure rates are achieved with a combination of surgery and postoperative interstitial radiotherapy.

Treatment of hypertrophic and keloid scars
■ Pressure – local moulds or elasticated garments
■ Silicone gel sheeting (mechanism unknown)
■ Intralesional steroid injection (triamcinolone)
■ Excision and steroid injectiona
■ 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)

Avoid ugly scar by achieving as early closure as possible

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