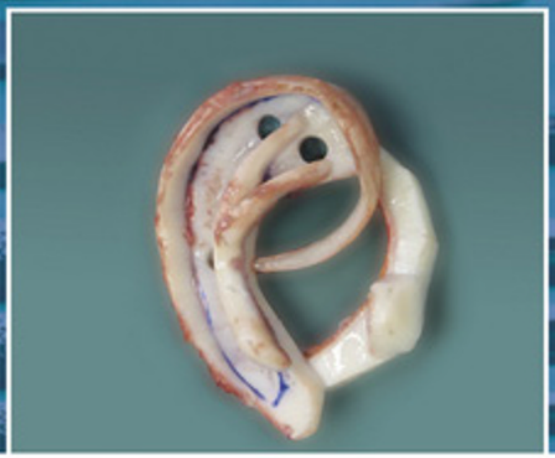
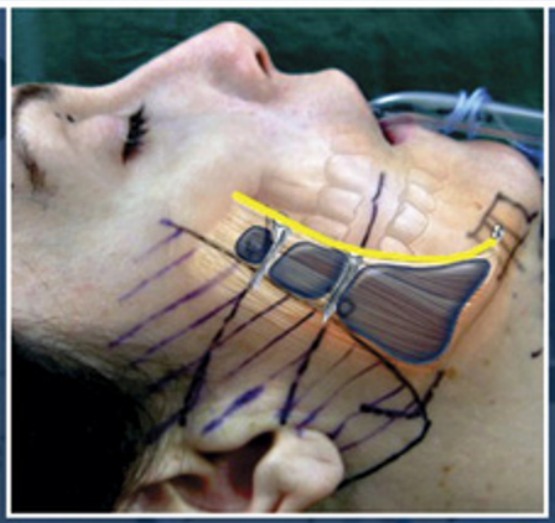


Plastic and Reconstructive Surgery

Approaches and Techniques



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Edited by
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Plastic and reconstructive surgery

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Approaches and techniques

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Foreword

Plastic surgery is a unique specialty, defined by concept rather than anatomical area. As such, it has grown enormously over the last 70 years and continues to evolve with changes in technology, improved understanding of anatomy and patient-centred outcomes. This progression leads to a vast and ever increasing array of new techniques and options for reconstruction, benefiting both our patients and the many other medical and surgical specialties that consult the plastic surgeon. With many tools at their disposal and the wide array of clinical maladies that they treat, the plastic surgeon has evolved into a problem solver. This synergy between plastic surgery and other surgical specialties has enabled these other specialties to utilize the problem-solving skills of the plastic surgeon to expand their therapeutic spectrum and tackle increasingly more difficult problems. However, to be an effective problem solver it is incumbent on the plastic surgeon to be familiar with the latest developments of our broad and expanding field. This highlights the necessity of a single-volume text that is comprehensive and practical, covering the full spectrum of plastic surgery and presenting the current state of the art of our specialty. This is exactly what the editors of *Plastic and reconstructive surgery: Approaches and techniques* set out to achieve in producing this excellent textbook.

It is truly an international effort at all levels, as the editors, from Australia (Ross D. Farhadieh), the UK (Neil W. Bulstrode) and Canada (Sabrina Cugno), have joined forces to recruit over 130 international contributors and produce a resource of over 1100 pages that provides a well-organized and thorough, yet succinct, text of the essentials of current plastic surgery. The editors are all highly qualified and accomplished young plastic surgeons, and they have been able to provide a global perspective of our specialty. Many of the contributors are world-renowned experts; however, there is also a new generation of young rising stars whose contributions are equally good, providing a new, fresh and contemporary feel.

Each chapter is clearly organized and provides an overview of the principles and the most recently described basic science essentials, as well as clinical applications and techniques, and pertinent bibliography for additional reading. The critical core information is provided for each topic, providing an excellent synopsis and reference for the student and practitioner.

Although aimed primarily at the trainee, I believe that it will also serve as an excellent and quick reference for the seasoned practising surgeon faced with complex problems requiring reconstruction throughout the body. It will also be an especially useful resource for senior plastic surgery trainees preparing to take their Board and Fellowship exams. The final chapter, dealing specifically with the certification process and fellowship exams, is interesting, and provides useful information and different perspectives of the qualifying processes in the British, European, Australian and North American systems.

It has been an honour and pleasure for me to have been asked to contribute the Foreword to this new textbook, which very nicely fulfills one of the traditions of surgery of passing down knowledge from one generation of problem solvers to the next. The new generation is likely to face even more complex problems and, using the latest techniques, solve them more elegantly than we can now. But the principles in plastic surgery never change, and this textbook provides the intrinsic fundamentals that all trainees must know, even as the field is ever expanding.

I congratulate the editors for their Herculean effort in recruiting an international cast of distinguished surgeons and thank the authors for flawlessly summarizing the huge avalanche of new information that has graced our specialty; finally, I thank the publishers, Wiley Blackwell, for producing this timely, impressive and comprehensive plastic surgery compendium.

Julian J. Pribaz
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Dedication

To inspirational mentors and dedicated apprentices everywhere.

Preface

During my plastic surgery training there appeared to be a plethora of summary and broad-stroke single-volume plastic surgery textbooks, all of which lacked adequate detail. Conversely I would encounter multivolume behemoths, detailed reference texts that always seemed leaden and difficult to digest. In my experience neither of these options fully addressed the needs of a trainee surgeon, or for that matter a more senior surgeon. Thus was born, on a long flight from Sydney to London, the notion of compiling a single-volume textbook that seeks to achieve the perfect balance of detail and palatability. To that end, in compiling this textbook we approached some of the world's leading authorities in the various fields of plastic surgery. This was with the belief that not only could readers benefit from such experts' enormous experience, but they could also gain practical insights from the ability of such experts to sift through the ever increasing volume of literature and distil what is relevant and applicable to everyday practice.

My co-conspirators in this endeavour, Mr Neil Bulstrode from Great Ormond Street and Dr Sabrina Cugno from Montreal Children's Hospital, had the perfect blend of enthusiasm and sense of humour to see this work through. I am grateful to them for their advice, time, effort and, most importantly, their friendship, all of which made this compendium possible. I also wish to thank all of our colleagues who took time out of their busy lives to make this volume possible. It has been an extraordinary experience for all of us to collaborate on this project. Our special thanks go to Professor Julian Pribaz for being kind enough to review the volume and write the Foreword. We also wish to thank Wiley Blackwell for their continued support for this project.

On a personal note I wish to thank my mother Tadjvar, my brother Arash and wife Yassi for enduring me during the last 2 years, and for their unwavering support. My parents have been my guiding light in demonstrating the importance of a strong work ethic and integrity. I also wish to thank Messrs Neil Bulstrode and Adriaan Grobbelaar for the extraordinary fellowship opportunities and friendship they have afforded me. I am very grateful to Messrs Ash Mosahebi and Jian Farhadi, who kindly accommodated me in their operating theatres and taught me a great deal about the art of plastic surgery. In Melbourne I wish to extend my sincere thanks to Mr Bryan Mendelson, who during my visits showed immense patience and generosity in teaching me the philosophy and techniques of facial aesthetic surgery. My gratitude also goes to Mr Stephen Flood, who illustrated by example a sensible approach to plastic surgery and kindness in mentorship; this continues to serve as an aspiration. As a plastic surgeon I am most indebted to Professor Wayne Morrison, who has curiosity, humility, dedication to teaching and generosity of will and spirit, tempered with a crisp sense of humour, in equal inspirational measure. It was truly an honour and a privilege to serve as his registrar.

Rostam D. Farhadieh
Sydney
January 2015

About the companion website

This book is accompanied by a companion website:

www.wiley.com/go/farhadieh/plasreconsurgery

The website includes:

- Powerpoints of all figures from the book for downloading

PART I

Basic science and principles

CHAPTER 1

Wound healing and scar formation

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TYPE I – CLASSICAL CUTANEOUS WOUND HEALING

A wound, in the context of skin, is a breach in the barrier that distinguishes an organism from its environment. The process through which the organism works in order to address this breach is 'wound healing' which, because of the important role the skin plays in the survival of the organism, is quite literally vital, and conserved through evolution. In the normal course of events, a lower species accepts tissue loss and heals a wound by exposure, licking, picking and, at the molecular level, scarring. The single most important impact on wound healing in humans is the early closure of wounds, by apposition with sutures in incisional wounds, and skin replacement in excisional wounds. Humans can deny significant skin and composite tissue loss by a 'like for like' replacement in the specialty of plastic and reconstructive surgery, and here we can boast a form of 'supranormal wound healing'.[1]

Wound healing classifications

There are many ways to classify wound healing. In simple terms, we can consider:

- four phases – coagulation, inflammation, fibroplasia and remodeling;
- four types – fetal, adult, acute and chronic;
- four ages – young, plateau, regressing, atrophic; and
- two systems of healing – epidermal and dermal.

We can also classify wound healing in terms of clinical features and their wound management.

Phases of wound healing

Wound healing can be considered a process of four sequential but overlapping phases by which the body closes a breach in tissue continuity. There are many ways to define such a complex process. Key to understanding the standard classification of the process is a consideration of: the timing, the cellular activation or influx and the chemical mediators.[2]

Coagulation

This *immediate* response to cutaneous injury involves two cascades: the clotting cascade, with the formation of platelet clot which adheres to the collagen exposed following endothelial disruption; and the complement cascade and complement-mediated vasodilatation. Histamine (released by mast cell degranulation) and kinins contribute to vasodilatation and increased vascular permeability. The first cells involved then are platelets and mast cells, and the first mediators histamine, kinins, platelet-derived growth factor (PDGF) and transforming growth factor beta (TGF-β). The increasing interest in platelet-rich plasma (PRP) clinically in regenerative medicine is based on these early cascades.

Inflammation

Between the time of injury and around 4 days post injury, the clinical signs of inflammation develop. Classically these are redness, swelling, pain and loss of function. These result from inflammatory mediators and the capillary leak into the extracellular space that they coordinate. The next inflammatory cell type to arrive at the wound is the macrophage, followed by neutrophils and then lymphocytes. Keratinocytes in the wound edge and follicle remnants migrate and proliferate, and fibroblasts are chemo-attracted, and become activated.

Fibroplasia

From day 4, and for 2–4 weeks, the wound bed becomes vascularized, and type III collagen is laid down by fibroblasts to replace any dermal loss. In the absence of epidermal cover, this appears clinically as granulation tissue. Closed wounds become red and raised for a while. Hydrated glycosaminoglycans form a ground substance for the collagen fibrils. This phase is characterized by fibroblast proliferation, but also by keratinocyte proliferation.

Plastic and Reconstructive Surgery: Approaches and Techniques, First Edition. Edited by Ross D. Farhadieh, Neil W. Bulstrode and Sabrina Cugno.

Remodelling

From a few weeks to 18 months or more, the wound goes through a long phase of remodeling. Fibroblasts mature into myofibroblasts to contract the wound. Type III collagen is gradually replaced by type I collagen. Disorganized collagen becomes lamellar.

Repair versus regeneration

There are significant differences between the wound healing seen in fetal life and that seen in postnatal life. So-called scarless healing occurs for a period in the fetus, however this is not absolute but dependent on gestational age and wound size.[3] In late fetal life scarring does occur, and before this point in time, a large enough wound will still result in scarring. In postnatal life, scarring is the inevitable and permanent consequence of wounding beyond the epidermal basement membrane. An adult lower vertebrate such as a salamander can regenerate an amputated limb but will not aspire to climb Mount Everest or graduate from Oxford! Although regenerative medicine attempts to harness the same plasticity seen in lower vertebrate regeneration, what is generally achieved is only 'partial regeneration', and not replacement of like with like. The ongoing concern in work to manipulate adult/somatic stem cells to achieve true regeneration is around a loss of control and the risk of carcinogenesis.

Acute versus chronic wound healing

Acute wound healing is hard to distinguish absolutely from chronic wound healing, and the processes at a cell and molecular level may be similar.[4] Chronic wound healing occurs when healing takes longer than might be anticipated in a fit, healthy person, and is often associated with comorbidity. Such wounds seem to become stuck in the inflammatory or proliferative/fibroplastic phases. Local wound management can only usefully begin following an assessment and optimization of systemic comorbid conditions.

Epithelial/epidermal versus mesenchymal/dermal wound healing

The epidermis provides the ultimate barrier between the body and environment. The main cell type is the keratinocyte. Regeneration occurs from a population of follicle stem cells.[5] The dermis provides the structural support to the epidermis and other related adnexae. The main dermal cell type is the fibroblast. As in embryogenesis, there is ongoing 'cross-talk' between the epidermis and dermis in somatic cutaneous wound healing, the epidermal element of which has been relatively underplayed. Even less consideration has been given to the contribution of subcutaneous fat to cross-talk.

Wound healing and scarring

In many ways, wound healing and scarring are inseparable – the one leads to the other at some level and over time. When does a wound become a scar? That will probably depend at a molecular level on very early wounding and wound management events. Although a scar is the inevitable and permanent consequence of postnatal wounding beyond the basement membrane and a compromise between regeneration and repair, at a clinical level, its significance is largely patient related and subjective. This can now be captured clinically by the Patient and Observer Scar Assessment Scale (POSAS), which draws patients into their own management.[6]

Most patients would perceive a wound as a scar from the time that the wound is no longer open, and often that equates to the absence of exudate and any requirement for dressing care. Clinically, even a normotrophic scar will go through a natural progression to maturity. When young, it will be active. Most young scars exhibit features of hypertrophy. Most scars then go through a plateau period of relatively little clinical change in the absence of treatment. Most scars then go through a period of regression of inflammatory signs and symptoms, and eventually settle to a mature state. Some scars, after many months or years, and sometimes because of treatment, become very thin, pale and atrophic.

When then does a normotrophic scar become a pathological scar? A normotrophic scar results from uneventful primary healing, but there is wide variation with age, site and skin type, and a period of hypertrophy is not unusual. Hypertrophic scarring is classically seen in paediatric burn wounds that have struggled to heal.[7] The scar becomes red, raised, painful and itchy around 2–3 months following wound closure, particularly in wounds that have taken more than three weeks to heal. The scar settles over 18 months to 2 years, but often incompletely. Hypertrophic scars occur particularly in extreme Fitzpatrick skin types. Presumably, there is a bell curve distribution within the population, where those at the extreme end of hypertrophic scarring could be termed pathological. Although a keloid scar shares many of the features of a hypertrophic scar, and may represent an extreme example of the same, it is defined by extension beyond the confines of the injury, by almost inevitable progression beyond 2 years, seldom regressing; by being refractory to most treatments and recurring within 4–6 months of cessation of most treatments; and by behaving pathologically like a benign tumour. Understanding such an extreme phenotype may prove key to the effective management of more normotrophic scarring.

Epidermal wound healing

Adult epidermal stem cell biology is relatively well understood. Keratinocyte stem cells reside primarily in the bulge region of the hair follicle and, by asymmetric division, populate the interfollicular basement membrane with transit amplifying cells.[8] These divide a number of times to provide the differentiating cells of the stratifying epidermis. A huge array of small

peptides are involved in coordinating the response to epidermal wounding by autocrine, paracrine and juxtacrine signaling.[9] In a human model of epidermal healing, a number of phases of keratinocyte activity are suggested, as shown in Figure 1.1.[10]

Acute activation

Almost immediately following wounding, the epidermis expresses interleukin 1β (IL-1β) and interleukin 6 (IL-6) and the dermis TGF-β1, committing transit amplifying cells to mitosis. Although TGB-β1 is antiproliferative, it is pro-migratory to keratinocytes.[11]

Early activation

Towards 24 h following wounding, keratinocyte proliferation and migration are clear. Epidermal expression of TGF-α and IL-6 is accompanied by dermal fibroblast keratinocyte growth factor (KGF) and IL-6 expression. A paracrine loop of epidermal IL-1β induction of the potent keratinocyte mitogen KGF from dermal fibroblasts seems likely.[12] TGF-α both is a keratinocyte mitogen and induces the migratory K6/K16 keratinocyte cytoskeletal phenotype in the suprabasal compartment.[13] It may also recruit nearby follicles by juxtacrine signalling.[14]

Restitution

Over weeks, homeostasis is restored, with relatively late activation of the bulge to restore the transit amplifying population.

Dermal wound healing

Wound healing studies have concentrated far more on the dermis than epidermis, and particularly on macrophage production of TGF-β isoforms. This multifunctional growth factor appears to play a key role in dermal healing and scarring.[15] Although the TGF-β1 isoform promotes scarring, the TGF-β3 isoform appears to have the opposite effect.[16] Juvista (Avotermin) was developed to improve the quality of normotrophic scarring, but failed in a European phase 3 clinical trial.[17] TGF-β, however, remains a key pharmaceutical target.

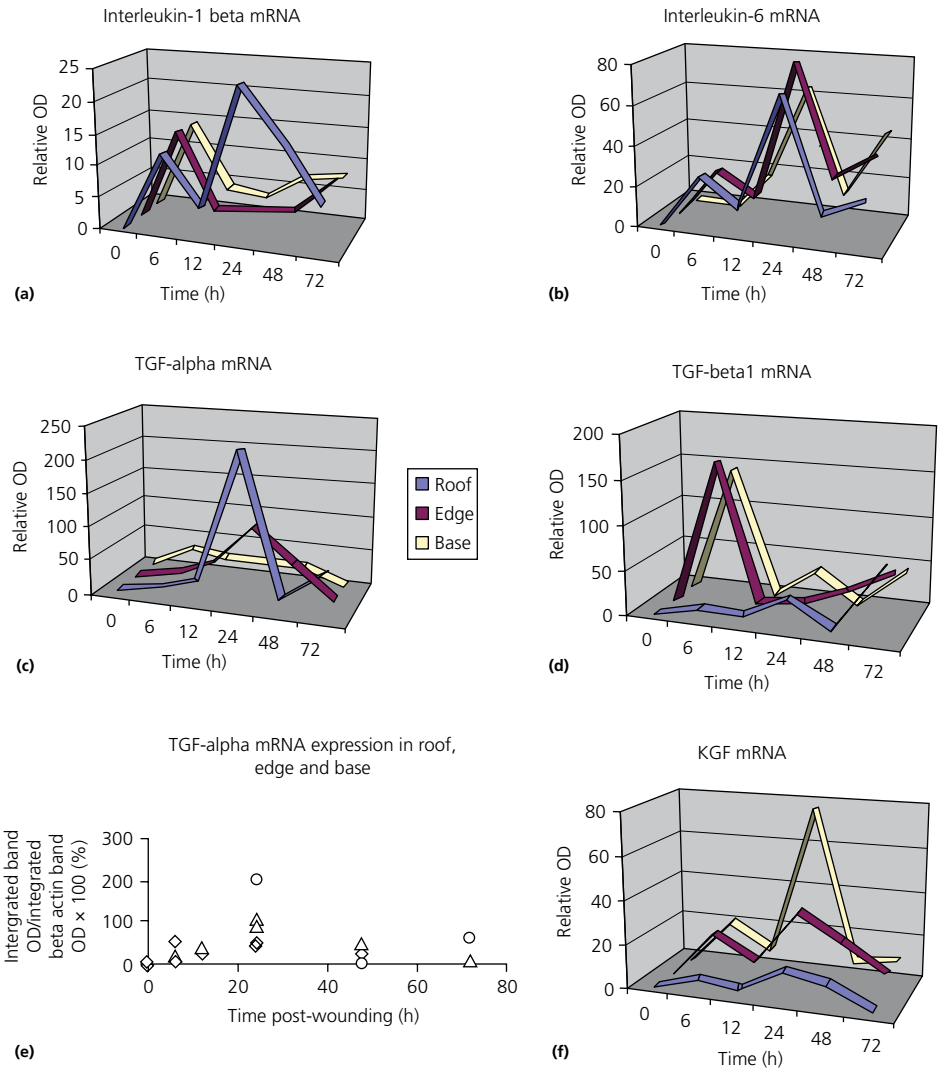

Figure 1.1 Temporo-spatial cytokine and growth factor gene expression in human suction blister wound healing. TGF, transforming growth factor; KGF, keratinocyte growth factor; relative OD, relative optical density.

A preoccupation with one growth factor, albeit a powerful, multifunctional factor with isoforms of different action, and for each a dose-dependent heterogeneity of responses, is arguably to ignore the complexity of wound healing cascades. There are many factors at play, often pleiotropic, and there is significant redundancy – so that many factors can contribute to the same outcome (i.e. rapid closure by scar). The growth factor that has shown most promise when delivered alone is PDGF.[18]

The dermis is far more than an extracellular matrix populated by fibroblasts. It hosts a vascular network (arterial, venous and lymphatic) and a neural network, and supports the follicle and other adnexal stem cell niches. It must also, it seems, interact with the subcutaneous fat. One approach to tissue engineering to provide for wound tissue loss is to synthesize an appropriate nanotechnology scaffold for key cellular elements to populate and develop. This can be *biofunctionalized* by incorporating a latent growth factor.[19]

Wounds can be classified based on clinical management and outcome as shown in Table 1.1.

'Normal'/primary incisional wound healing (type Ia)

Incisional wound healing occurs following surgical access, or 'incisional' or lacerating trauma.[20] In the former, the wound will begin sterile, and in the latter some degree of contamination is usual. In neither instance is there significant tissue loss. Classically, in modern medical practice, these wounds are formally closed, although the timing of that closure will vary, affecting the quality of the healing processes and the scar that results.

Early closure (type Iai)

If an incisional wound is closed directly, the healing will tend to be optimal. Elective surgical wounds are closed at the end of the procedure performed under sterile conditions. Traumatic lacerating wounds will generally be cleaned and closed the same day, and before significant bacterial colonization occurs. A relatively arbitrary time limit to early closure has evolved in practice of 48 h from injury. Beyond this, it is considered likely that colonization may be significant enough to deleteriously affect healing, and as a consequence the quality of scarring, which may become hypertrophic.

Late closure (type Iaii)

If a type Ia wound is sutured after 48 h, wound colonization may result in infection, dehiscence and delayed healing. Counterintuitively, according to current dogma if the wound is left open until day 4 or 5, as in 'delayed primary closure', satisfactory results can be achieved at a stage when the inflammatory response has become established – although a recent Cochrane review found no evidence for this.[21]

No closure (type Iaiii)

An incisional wound beyond the epidermal basement membrane that is left unclosed will gape and behave much like a deep excisional wound, healing by classical 'secondary intent'.[22] The time

Table 1.1 A revised wound classification based on clinical management and outcome

Type I: Classical	Wound ± intervention	Character of healing
(a) 'Normal'/primary incisional – incisional, no tissue loss	(i) Closed early (<48 h)	Low risk of infection Minimal line scar
	(ii) Closed late (>48 h)	Increased risk infection Increased risk chronicity More significant scar
	(iii) Unclosed	Healing by granulation (similar to type Ibii), but in absence of tissue loss) High risk of chronicity
'Normal'/secondary excisional – tangential tissue loss, no replacement	(i) Above mid-dermis	Rapid re-epithelialization (<10 days) Minimal clinical scar
	(ii) Below mid-dermis	Slow/absent re-epithelialization High risk of infection and chronicity Closure by scar
Type II: Neoclassical		
(a) 'Supranormal' by skin replacement	(i) Early split- or full-thickness skin grafting of type Ibii)	Present gold standards Cosmetic and functional problems remain Donor defect
	(ii) Biotechnological skin replacements (Cuono technique, *in vitro* composite grafts)	Ideally autologous and with viable cellular elements Opportunity to improve on types Ibii and IIai
(b) 'Supranormal' with apparent acceptance of tissue loss	(i) Type Ibi treated with cultured keratinocyte allografts or vapour-permeable membrane (ii) Chronic full-thickness wound treated with cultured keratinocyte allografts or vapour-permeable membrane	Follicle recruitment by activated extended follicle units Simulation of an acute wound environment

to healing will be slow, because closure will rely on wound base contraction and edge re-epithelialization. As a consequence, the quality of the scar will tend to 'pathological'.

'Normal'/secondary excisional wound healing (type Ib)

Where surgery requires skin excision, or cutaneous trauma is tangential (e.g. burn injury or friction loss) and the tissue loss is not replaced, then the time to healing and the scar quality will depend on the depth of the loss. Re-epithelialization and restoration of barrier function from adnexal remnants will be slower, the deeper the injury.

Above mid-dermis (type Ibi)

Tangential tissue loss above the mid-dermis leaves a partial-thickness wound that will heal in around one week under ideal circumstances, and result in a 'controllable' scar – as in the surgical split-thickness skin graft donor site. If a traumatic tangential injury clearly reaches the mid-dermis acutely (i.e. a mid-dermal burn injury), then optimal wound management is key to prevent extension of the tissue loss to type Ibii. In extreme Fitzpatrick types, even these type Ibi wounds can scar pathologically.

Below mid-dermis (type Ibii)

Tangential tissue loss below the mid-dermis leaves a partial-thickness wound that will take three weeks or more to heal, and as a consequence will be more liable to chronicity and significant scarring.[7] It is at this depth, or beyond, that intervention is considered. A full-thickness defect can only re-epithelialize from the wound edge, and will otherwise close substantially by scar contraction of the base.

Abnormal wound healing

Systemic and local factors may reduce the quality of the skin and/or affect wound healing adversely. These are important to recognize and optimize.

Systemic factors

Systemic factors may be congenital or acquired. There are a handful of congenital conditions that affect the processes of healing, and in some instances the clinical quality of the skin and healing. A range of defects in collagen synthesis is seen in Ehlers–Danlos syndrome, and healing is poor.[23] The skin is vulnerable, and healing slow in epidermolysis bullosa, where for example, in the junctional variant, laminin 5 is deficient in the epidermal basement membrane zone.[24] The autosomal recessive premature ageing condition progeria manifests many features of normal 'acquired' ageing.[25]

With age, the changes in healing processes are fairly global, resulting in a delay in wound closure and a reduction in wound strength. How much these changes are the result of increasing comorbidities associated with age, rather than age itself, is not entirely clear. Other acquired systemic factors include: nutrition, drugs, diabetes and smoking. Vitamin C deficiency is the classical example of a nutritional factor involved in wound healing. Although scurvy is not likely with Western diets, vitamin C is an essential cofactor for collagen synthesis. Vitamin A deficiency is also rare in the developed world, but vitamin A can reverse steroid-induced collagenase activity. Zinc is important to many enzyme systems, and deficiency can be seen in large burn injury. In those same injuries, albumin can plummet to around 10 g/L, and although this will delay healing, closure can be achieved.

Obesity is epidemic now in the Western world, and associated with many comorbidities and wound complications following surgery.[26] Plastic surgery reconstructions following bariatric surgery are challenging. Large blood vessels will have developed to support the tissue volume, and these may contribute to postoperative bleeding complications. Despite these hypertrophic vessels, tissue perfusion may be poor, and the tissue lymphoedematous and critically colonized. Furthermore, closure after such excisional surgery is by definition under tension, so that infection and dehiscence are more common.

Anti-inflammatory systemic glucocorticoids, non-steroidal anti-inflammatory drugs and chemotherapy drugs globally suppress the cellular responses to wounding. Chemotherapeutic angiogenesis inhibitors, such as bevacizumab, a vascular endothelial growth factor-neutralizing antibody fragment used in colonic cancer, cannot be prescribed six weeks before or after surgery to limit the wound healing risks.[27]

Diabetes mellitus may affect healing in a variety of complex ways, particularly in the lower limbs. Patients with diabetes are susceptible to atherosclerosis in larger vessels, and tissue oxygen delivery is further reduced by the stiffness of the red blood cells, and the higher oxygen affinity of glycosylated haemoglobin.[28] These effects are compounded by any neuropathy, and impaired cellular immunity, phagocytosis and bacterial killing.

Smoking may affect wound healing in both immediate and longer term ways.[29] Nicotine causes sympathetic vasoconstriction, and carbon monoxide shifts the oxygen dissociation curve to the left. Long-term smoking accelerates atherosclerotic changes. Smoking appears to be a particular problem in surgery to superficial soft tissue planes, where wide skin undermining with the sacrifice of multiple perforators, and closure under tension are combined, as in abdominoplasty surgery.

Local factors

One of the most controllable local factors for incisional wounds is surgical technique and the handling of tissue. Tissue handling within the specialty can be observed, par excellence, under the microscope during a microvascular anastomosis, where poor

handling results in anastamotic thrombosis.[30] Local factors often reflect systemic comorbidities, so that poor blood supply and oxygen delivery, and even critical bacterial colonization are more often than not a local manifestation of a systemic factor. Conversely, the radiotherapy that results in local thromboendarteritis obliterans and causes healing problems over time may also have systemic effects.[31] In terms of recurrence, radiotherapy is the most effective treatment for keloid scars, damping down the 'overhealing' provided it follows extralesional excision directly.[32] Breaches in the skin are inevitably colonized by commensals. With time and increasing bacterial number, the body mounts an inflammatory response, and the colonization is termed critical. Critical colonization is not anticipated until around 48 h as a rule of thumb. Once 10^5 organisms are present per gram of tissue, the wound may be considered infected. Chronicity and some level of colonization go hand in hand. Bacterial biofilms are prevalent in chronic wounds, including anaerobic organisms not isolated by standard culture systems, and this is an area of particular current interest in such wounds[33] – and also of course in subcutaneous/cavity wounding and scarring (e.g. breast capsular contracture).[34]

Mechanotransduction

There is a sense that the skin is constantly subclinically injured to some degree by sheer forces, and indeed even the force of gravity.[35] This may drive the baseline turnover of the skin. The effects of physico-mechanical forces on cell behavior, termed *mechanotransduction*, are becoming increasingly recognized and understood in wound healing.[36] In 1861, Karl Langer observed that the skin exhibits intrinsic tension,[37] and Langer's lines, defined by the direction in which circular excisional wounds will elongate to ellipses by anatomic site, are used today to orientate excisional surgery. *Tensegrity* describes the way in which mechanical forces regulate biological systems via perturbations in structural architecture; disruption of tensional integrity triggers cellular pathways that restore mechanical homeostasis.[38] Cells also actively generate intracellular tension, *cell traction forces*, as they interact with their environment during, for example, migration.[39] A cell-centric view of mechanotransduction is, however, inadequate. Conformational changes in the extracellular matrix by mechanical forces can reveal cryptic binding sites and expose growth factors. Non-structural, extracellular, *matricellular* proteins (e.g. connective tissue growth factor) are increasingly implicated in the regulation of healing and scar formation.[40] Mechanosensing in the skin is a feature not just of fibroblasts but also of keratinocytes and nociceptors. It is quite possible that physical cues during wound healing direct, in part, stem cell fate within that niche.

Any therapeutic effects of silicone gels, pressure garments and linear taping may work through mechanical offloading and mechanotransduction pathways. The use of Botox A to control tension across healing facial scars and improve scar quality is an interesting new approach.[41] Vacuum-assisted closure has become a common approach to complex wound management, and although poorly understood, must rely to a large extent on mechanotransduction.

TYPE II – NEOCLASSICAL CUTANEOUS WOUND HEALING

'Supranormal' healing by skin replacement (type IIa)

Classically, intervention to close a wound was sometimes considered 'tertiary' wound healing. The great variety of techniques now available suggest a more structured classification.

Early split-thickness or full-thickness skin grafting of type Ibii (type IIai)

Those tangential traumatic or excisional wounds that are unlikely to heal in a reasonable timescale and are therefore likely to scar are most commonly closed with split-thickness skin autograft. Where the wound is full thickness, the environment optimal and the defect limited in size, a full-thickness autograft will provide a superior reconstruction. Of course, any number of local and distant skin and fasciocutaneous flaps are considered for defects that have resulted in an ungraftable bed, and this category of skin replacement feeds into other classifications of flap reconstruction. Skin and fasciocutaneous free flaps are increasingly being used to close defects in hostile comorbid conditions (e.g. in 'vasculoplastics' practice).[42]

Biotechnological skin replacements (type IIaii)

In recent years, products, often xenograft in nature, have been developed to provide the quality of a full-thickness graft reconstruction from a split-thickness skin graft donor site. 'Dermal regeneration templates', such as Integra, engraft to provide a 'neodermis' to support a thin autograft in two operative stages.[43] A single-stage Integra system has been available following the success of single-stage Matriderm grafting.[44] Included here also is the system developed by Cuono and colleagues that combines allograft dermis with autologous cultured keratinocytes in the closure of full-thickness burn excision beds.[45] Cultured keratinocyte technology represents one of the most established forms of somatic stem cell therapy, and sits most coherently within plastic and reconstructive surgery.

'Supranormal' healing with apparent acceptance of tissue loss (type IIb)

When tangential tissue loss is accepted and no apparent attempt is made to replace like with like to the level of loss, then there are still interventions that seem to present some clinical advantage. George Winter presented his understanding of tangential wound healing in a porcine partial-thickness

excisional model that included both edge and base contributions to restoration of epidermal barrier function, and introduced the world to the concept of 'moist wound healing'. This spawned a massive industry in moist wound healing dressing systems, initially vapour-permeable membranes.[46] Although this transformed the 'dry' wound management of the time, it has not proven a panacea, and far more sophisticated biological systems have evolved since (see below). The clinical evidence base for these, however, has been slow to evolve on a cost basis, and so marketplaces have yet to develop around economy of scale.

Type Ibi treated with cultured keratinocyte allograft or biological dressing (type IIbi)

Cultured keratinocyte allografts have been used for decades to accelerate partial-thickness wound healing.[47] Although they do not survive transplantation long term,[48] they present a temporary and coordinated 'growth factor factory'. It may also be that they provide a juxtacrine mechanism for 'discontinuous follicle recruitment' by bridging adnexal remnants separated by tangential partial-thickness wounding.[10] Biobrane is a conforming bilayer of porcine collagen and nylon.[49] It is at least haemostatic and adheres to a clean partial-thickness wound until shed when the epidermal barrier has been restored. The evidence for its efficacy and detail of any mechanism has never been established, but a role for the limitation of colonization of adherence seems likely.

Chronic full-thickness wound treated with cultured keratinocyte allograft or biological dressing (type IIbii)

Chronic full-thickness wounds are a huge financial burden to any healthcare system, and generally associated with comorbidity.[50] Any comorbid condition should be optimized in the first instance. There then may be some further benefit from cell-based therapy or biological dressings. Both cultured keratinocyte allografts and autografts have been used to treat such wounds.[51] It is suggested that even with poor clinical autograft take, a more acute healing picture is seen, at least clinically – healing is 'kick-started'. The wound bed can be modulated with, for example, hyaluronic acid, an important component of the embryonic extracellular matrix, to improve clinical take, perhaps by supporting a niche-like stem cell environment.[52]

Fat

There has been a huge recent interest in fat grafting, not only for augmentation including following subcision of indented scars and scar-related fat atrophy, but also to improve healing and the quality of the overlying scar.[53] It is fascinating to reflect that Sir Harold Gillies may have used whole-fat grafts in acute closure of craniofacial traumatic wounds with the same intent many decades ago.[54] It has been suggested that any effect on healing and scarring results not from the grafted fat directly, much of which is lost, but from stimulation of a local mesenchymal stem cell response. The current controversy is around the method of enrichment of autologous fat to provide safe augmentation long term, and the resolution of this will run parallel with resolution of controversy around any effect on healing and scarring. A recent randomized controlled study in normal human volunteers demonstrated that enrichment of autologous fat with cultured autologous adipose-derived stem cells was significantly more effective, in graft survival terms, than a more standard approach.[55]

Lymphoedema

Lower limb lymphovenous disease is a recognized cause of recurrent cellulitis and chronic wound healing. There is a newly recognized patient base in the morbid obese and postbariatric population, and with the evolution of supra-microsurgical techniques, the promise of new therapies (e.g. lymphovenous anastomosis and lymph node transplantation).[56]

Future

Modern genomic and proteomic techniques allow us to define the processes that control tissue volume and its nature in healing at a molecular level – broadly: migration, proliferation, differentiation and apoptosis. Those techniques require very little material from biopsy, and we should expect to see more evidence from controlled longitudinal human studies available to support our understanding of the different types of healing. The sheer complexity of the pathways and interactions within countless networks will require a systiomics, or systems biology, approach, calling on applied mathematics and computer modelling. Resources to support controlled human studies of wound healing and interventions are limited in part by a perception that the area is mundane. There are, however, many gaps in our basic understanding of what are quite fundamental processes. Cell-based therapies are expensive, but will continue to offer the most rationale wound management solutions until our understanding is more complete. Longitudinal cost–benefit analyses of novel therapies remain few and far between.

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CHAPTER 2

Basic skin flaps and blood supply

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Introduction

Flap surgery is the commonest procedure in plastic surgery and is the essence of the discipline. Critical to its success is an understanding of the soft tissues' blood supply and its compliance and mobility. As all flaps 'rob Peter to pay Paul' it is also about conceptualizing the secondary defect and minimizing its consequences. The art and craft of plastic surgery necessarily requires an aesthetic sense and experience.

Evolution

For almost a century Manchot[1] and Salmon's[2] detailed studies of the skin's vascular design were overlooked by clinicians. With limited understanding and the simplistic belief that the skin's blood supply was based on a random distribution in the horizontal plane, local flap surgery was unpredictable and its progress curtailed by an adherence to dogmatic rules such as length-to-width ratios and the superiority of proximal over distally based flaps. A generation of surgeons failed to appreciate the simple observation that circumferentially incising large skin lesions in the process of their elevation and removal did not compromise their circulation. The explanation, of course, was that their blood supply was derived from the depths and not horizontally. Surgeons no doubt were aware of the circulation from below but the reality was that sufficient numbers of these vessels had to be divided to permit the flaps to transpose or rotate, and ultimately it was the base fed by the horizontal input that was the critical lifeline. Not until it was shown that flaps could be completely islanded and still live was it possible to move flaps based on these deep vessels. In 1970, Milton elegantly highlighted the fallacy of the length-to-breadth ratio using pig studies to demonstrate the existence of arterialized zones of the integument that would survive over extreme lengths even if completely islanded, provided they retained their arterial source at their base.[3] These exciting findings breathed life back into the clinical study of the soft tissue's blood supply. True to the adage that history is written by the victors, the blind acceptance of random patterns proposed by Harold Gillies[4] was derived in part from the neglect of the publications of the Dutchman Johannes Esser (1917), Gillies' plastic surgery counterpart for the German army in World War I.[5] Instead of the complex tube pedicle, Esser performed one-stage 'arterialized biological island flaps' based on the palpable arteries of the head and neck region. He clearly recognized the fundamental concepts of the flap's axial blood supply.

The first clinical application of the new 'axial pattern' concept was the groin flap, based on the superficial circumflex iliac branch of the femoral artery.[6] The wide arc of rotation of this very long and narrow-based flap of like tissue expanded the single-stage reconstructive options for the regional wounds previously manageable only by multistage transfers requiring protracted immobilization and hospitalization.

What had been anecdotally reported in the early literature and had been empirically adopted in practice, the Indian forehead flap for nasal reconstruction and the epigastric flap in the lower abdomen, now made sense.[7] The former flap unwittingly captured the supratrochlear/supraorbital vessels and the latter the superficial epigastrics.

With this new awareness, omentum, although not skin, was quickly recognized for its application as an arterialized flap by virtue of it wearing its blood supply on the outside of its surface. The vasculature could be pruned to critical arterial pedicles and tunnelled from the abdominal cavity to cover far-flung defects and then skin grafted.

Other fundamental concepts of skin blood supply were soon elucidated, such as the myocutaneous flap, where the skin relied for its blood supply on vessels perforating through the underlying muscle. Providing the muscle was raised on its blood supply, the overlying skin would survive even when completely islanded. As muscles typically are vascularized often by a single source at their origin, the muscle pedicle added even further length to the arc of flap rotation. The gracilis[8] and latissimus dorsi myocutaneous flap,[9] reinventing the earlier findings of Tansini,[10] were the first to be described

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and widely adopted. Muscle flaps without skin followed and the anatomical articulation of their vascular basis further expanded the options for one-stage locoregional reconstructions (Figure 2.1).[11]

Fasciocutaneous flaps recognized for the first time that a significant contribution to skin blood supply was in the plane of the fascia and provided this fascia was included within the flap, large areas of previously unreliably vascularized skin would survive.[12] This particularly applied to the lower limb. Initially they were designed on the assumption their vascularity was in the plane of the fascia and in the limbs flaps were based proximally to capture its source. It was soon clear that much of this fasciocutaneous blood supply derived from perforating branches of named deep vessels emerging vertically between septofascial muscle planes from deep axial vessels, and furthermore these zones could be completely islanded from their proximal connections. Mathes and Nahai have classified fascia and fasciocutaneous flaps into three types: type A, direct cutaneous pedicle; type B, septocutaneous pedicle and type C, musculocutaneous pedicle.[11]

This led to the unifying concept of angiosome blood supply, where the tissue is considered as a three-dimensional territory or somite structure, akin to vertebrate embryological development, where not only skin but whole mesenchymal somites including skin, muscle and bone have a vascular zone.[13] This was supported by meticulous cadaver studies and expanded the earlier work of Manchot and Salmon.

Concurrent with this explosion in the understanding of the skin's blood supply, advances were being made in microvascular surgical techniques and instrumentation, initially by the neurosurgeons,[14] but quickly adopted for plastic surgery. These techniques had immediate applications for replantation, but the possibility of transplanting toes and territories of skin by anastomosing specific vessels was now opportune. The first such flap was an omental transfer to the scalp.[15] Skin flaps soon followed.[16–18]

Current understanding of the blood supply to skin

Flap surgery involves the transfer of tissue with its vascularity preserved. This requires an understanding of the physiology and anatomy of the integument's blood supply.

Physiology of the skin's blood supply

The blood supply (12.8 mL/100 g/min) to the skin greatly exceeds its metabolic needs because of its homeostatic role in thermoregulation. Perfusion of the skin's capillary beds is regulated by both local and systemic neurohumoral mechanisms. These act on precapillary sphincters and arteriovenous anastomoses, influencing the filling and emptying of the dermal plexuses and in turn not just the circulation of the skin and subcutaneous tissue, but also insensible heat loss and venous return to the heart.

Immediately after elevation of a skin flap, perfusion is transiently reduced by transection of blood vessels, inflammation, a hyperadrenergic state (associated with sympathectomy) and possible reperfusion injury. With no pharmacological means to reliably manipulate these effects at the capillary level, unless the design and execution of a flap includes an adequate circulation to overcome this ischaemic phase the flap may fail.

Anatomy of the skin's blood supply

The circulation of the skin and its underlying structures consists of a three-dimensional continuous vascular network. Conceptually this can be broken into *horizontal* and *vertical* components. Running parallel with the skin, and constituting

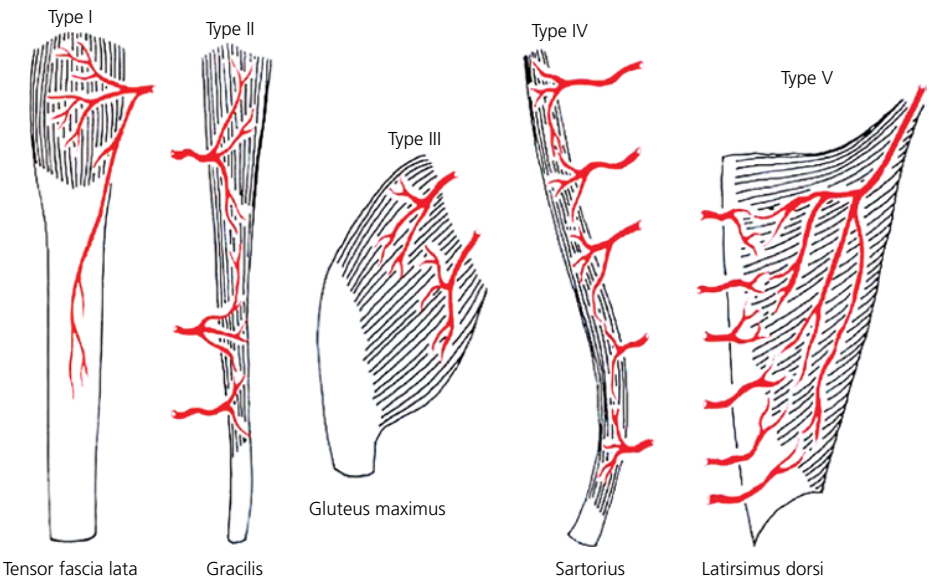

Figure 2.1 Mathes and Nahai classification. Patterns of vascular anatomy: type I, one vascular pedicle; type II, dominant pedicle(s) and minor pedicle(s); type III, two dominant pedicles; type IV, segmental vascular pedicles; type V, one dominant pedicle and secondary segmental pedicles. Source: Mathes and Nahai, 1981.[11] Reproduced with permission of Lippincott Williams & Wilkins.

the horizontal component of the skin's blood supply are the numerous vascular plexuses (Figure 2.2a). Most important of these are the subdermal plexus and the deeper suprafascial plexus. Where no deep fascia exists, an equivalent structure such as the panniculus carnosus (e.g. platysma, palmaris brevis and the dartos muscles in humans) serves a similar purpose. These are the vascular bases of (random) cutaneous and fasciocutaneous flaps (Table 2.1).

Vessels arising vertically from their source arteries either *directly* supply the skin, or *indirectly* supply the skin after nourishing deeper structures such as muscle and bone. These are known as 'perforators' and they arise from the deep named vessels, along their axial course, with highest density over the less mobile soft tissue that is adherent to underying septa. This is particularly evident in the limbs. Perforators anastomose initially with the prefascial plexus before continuing on to the subdermal plexus and are the vascular pedicles on which islands of skin and other soft tissue components may be based (Figure 2.2b). It follows that separate islands of skin and fascia, each based on a separate perforator but ultimately deriving from a common arterial axial vessel, can be raised on this common axis. This permits complex reconstructions with multiple independently oriented flaps.

The blood supply of the skin and its underlying structures can also be divided into vascular territories, or *angiosomes*.[13] Each territory is connected to its adjacent territory by bidirectional arterioles, the direction being interchangeable and determined by the relative pressure in each territory. The angiosome is the vascular basis for composite flaps.

Drainage of the skin is by a reciprocal three-dimensional venous network of avalvular bidirectional veins with a dominant subdermal component. This in turn drains into large-calibre subcutaneous veins or venae comitantes that run with perforators. Superficial lymphatics follow subcutaneous veins and deeper lymphatics follow arteries.

Venous flaps are generally transferred as free microvascular flaps, where the flap is elevated superficially with only the venous system, thereby obtaining a thin flap and reducing morbidity.[19] The flap is arterialized through its veins by anastomosing them to an artery in varying configurations, the tissue being nourished by retrograde perfusion. Understandably, their reliability is inconsistent.

Indications for flaps

Skin defects should ideally be repaired by replacement of what is missing and generally this will include fat as well as skin. In many cases the local laxity of skin will allow direct closure and conversion of the deformity to a linear scar. Sometimes, however, although the wound can be technically closed directly this may create a dish deformity with dog-ears at either end. Removal of these dog-ears only aggravates the underlying problem of missing tissue. Here, well-designed local flaps from redundant areas can redistribute the tension so as to preserve available tissue and restore normal contour.

Defects that cannot be closed directly will need grafting or replacement with flaps. Skin grafts take by engaging with the

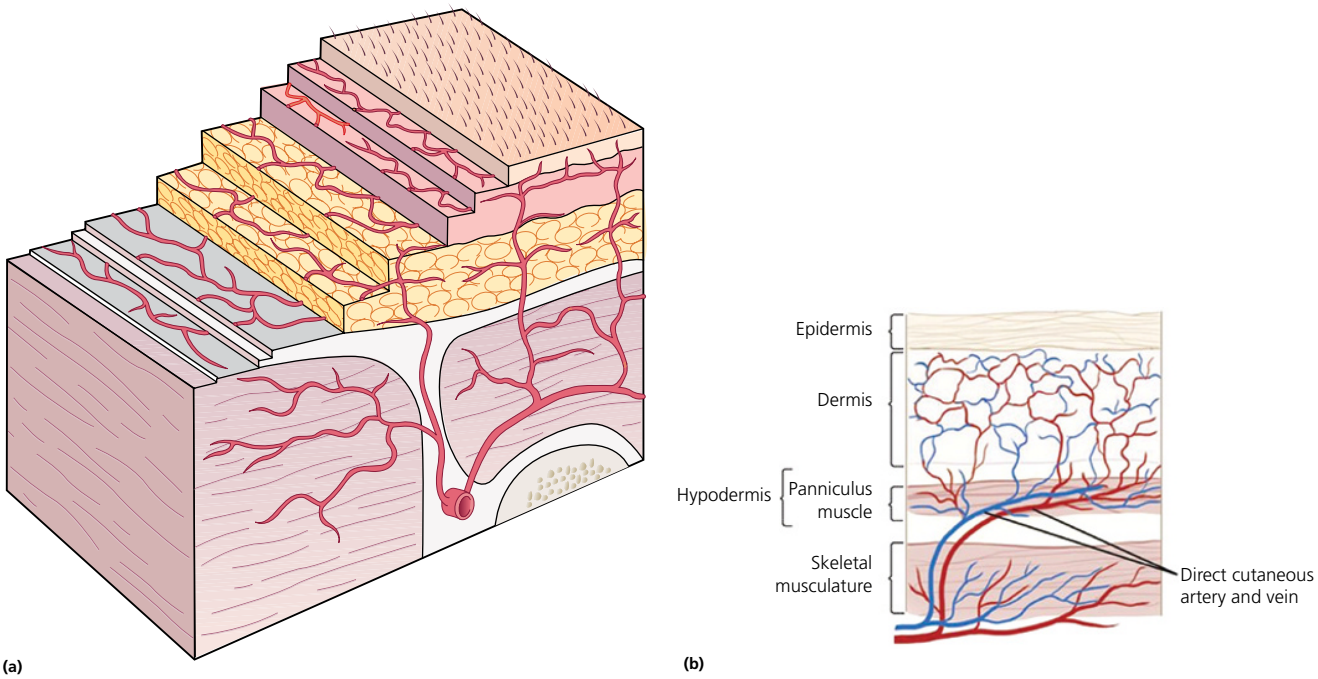

Figure 2.2 **(a, b)** Illustration showing subdermal plexus and suprafascial plexus. Also perforators.

Table 2.1 Classification of flaps based on movement and blood supply

Movement	Blood supply
(a) Local skin flaps	
Advancement	
In-continuity – rare	Random – intact skin base
Islanded (e.g. V–Y)	Random – subcutaneous base
Keystone	Perforator base
Transposition	
In-continuity (e.g. rhomboid, bilobed, Z-plasty)	Random – intact skin base
Islanded (e.g. propeller)	Perforator base
Rotation (e.g. scalp)	Random; arterialized
Combination	
(b) Regional (locoregional) flaps	
Transposition	
Cutaneous (e.g. groin flap, forehead, rhinoplasty, Abbe lip flap)	Subcutaneous arterialized
Muscle/myocutaneous	Arterial pedicle
Fasciocutaneous	Septo(myo)fascial perforators
Neurovascular island flaps (e.g. digits)	Neurovascular A–V pedicles
(c) Distant flaps	
Two-stage pedicle (e.g. cross finger, groin to hand	Random; arterialized
Multistaged 'waltzed' pedicle (e.g. tube pedicle)	Random
(d) Free flaps	
Arterial	Arterial – microvascular anastomosis
Venous	Venous blood supply
(e) Prelamination and prefabrication	Arterialized
(f) Allotransplantation – Opportunistic free flap allograft in patient simultaneously requiring organ transplant (e.g. abdominal wall with bowel transplant)	
(g) Tissue engineering – Although prefabricated and prelaminated flaps were the early prototypes of tissue engineered flaps, cell-seeded artificial scaffolds and/or adipogenic matrices are being investigated	Experimental

vasculature of the underlying bed. They are inappropriate in avascular circumstances (exposed bone, tendon, fracture sites, irradiated tissue and mobile beds) and here flaps are required as they possess an independent blood supply. Grafts often contract and may be a poor colour match; where the underlying fatty tissue is missing, they may result in contour defects and adherence to deeper tissues. Apart from burns and extensive injuries where large areas of skin are required it is generally accepted, particularly with the wide range of flap options available, that skin grafts are inferior to flaps and are rarely first choice. Other exceptions include the dorsum of the hand and foot, where thin skin is required. Few flaps meet these needs. Full-thickness skin grafts have an important place on the face, where the tissues are thin with little subcutaneous fat (eyelids, inner canthus, proximal nose and sometimes nasal tip). Flaps from regions adjacent to these sites are invariably too thick.

Elsewhere, flaps are indicated. Small defects are closed by local flaps from the immediate area and find their greatest expression in the head and neck region, particularly in association with skin cancer resection. Flaps may be in-continuity (transposition, rotation) or islanded (advancement), and their execution defines the quality of the plastic surgeon. Because of their near-perfect tissue match the well-executed local flap may be difficult to spot. Larger defects will require locoregional flaps from a distance and are most likely transposed on their narrow base to maximize their reach (arc of rotation). Because their skin texture, colour and thickness may not match that of the original defect they may require subsequent revisional surgery. Such distant flaps may be fasciocutaneous, myocutaneous or muscle flaps with skin grafts. Composites of tissue may be included – muscle or tendon, fascia or bone – allowing functional reconstruction of complex defects. Their vascular basis will be on the vascular pedicle alone or together with their associated skin, muscle or fascial carrier respectively. Usually the secondary defect will be directly closable.

For very large skin grafts or complex defects where a specialized tissue is needed, such as functional muscle or bone, innervated or hair-bearing skin, free flaps are indicated. These are based on a vascular pedicle and require microvascular anastomosis. Prefabricated (arterialized zone of specialized skin created by the implantation of a vascular pedicle so as to render it transferable as a free flap suitable to match a specific defect) and prelaminated flaps (the neovascularization of composite tissue around a vascular pedicle for subsequent transfer) are more sophisticated free flap indications. Tubed pedicle flaps are now rare but still find applications where there is an absence of recipient vessels at the defect site (Figure 2.3).

Design and application of flaps

Two concurrent considerations are critical to the successful planning of local flaps: (1) blood supply and (2) availability of adjacent mobile tissue (laxity).

Flap design and blood supply

The first consideration in designing a skin flap is to determine whether it will be viable. The vascular limitations on a flap's dimensions are not completely understood, though the principles to follow are helpful. Some flaps in some sites seem more predictable than others, despite the principles, and confidence in local flap surgery comes with trial and error, and ultimately experience with what works and what does not.

The simplest skin flaps are based on the subdermal plexus, the richness of which varies around the body (Figure 2.4). Vague length-to-breadth ratios govern the size of these so-called *random flaps*, with the only certainty being that a flap whose length equals its width will be viable anywhere on the body.