Pediatric Facial Plastic and Reconstructive Surgery

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Chapter 20: Wound Healing and Scar Revisions in the Pediatric Patient

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This chapter discusses the basis of wound healing in respect to not only the phases of response to injury but also the factors affecting the healing of wounds, including the effect of vitamins, age, foreign bodies such as sutures, anemia, and drugs. The chapter then describes abnormal wound healing including hypertrophic scars and keloids. Special problems such as electrical and thermal burns to the lip and neck, and principles of scar revision will complete the chapter. It is the author's strong feeling that meticulous attention paid to the management of the physiologic principles of wound healing is essential for optimal cosmetic and functional results.

Phases of Wound Healing

There are three (some authors say four) phases of wound healing: the exudative or inflammatory phase (sometimes subdivided into hemostatic and inflammatory phases); the proliferative phase; and wound contraction or the remodeling phase.

Inflammatory or Exudative Phase

The inflammatory or exudative phase begins immediately and can extend for several days. In the early phase, the disruption of blood vessels causes the wound to fill with blood, serum proteins, clotting factors, and platelets. Collagen is involved in the reparative process from the initiation of the wound throughout the end stages of remodeling. It is in the initial phase of the inflammatory (or hemostatic) portion of wound healing that there is interaction between collagen and platelets. The activated plateles not only initiate coagulation but will also release biologically active substances such as growth factrs, fibrinogen, von Willebrand's factor, fibronectin, and other substances that promote cell migration into the wound. The fibrin clot itself not only assists in hemostasis but also acts as a scaffold for later fibroblast migration.

Within the first 5 to 6 hr postinjury, neutrophils enter the wound as a result of the release of chemotactic factors. This is considered by some to be the latter portion of the inflammatory exudative phase or, by others, the beginning of the second phase (inflammatory) of wound healing. Monocytes will begin to accumulate within 24 to 48 hr. While the neutrophils are present to fight contamination from bacteria, the monocytes (macrophages) not only phagocytose bacteria, but will digest the neutrophils and bacteria and release biologically reactive substances that will assist in the tissue debridement and release of growth factors (platelet-derived growth factor (PDGF)) and transforming growth factors, type beta (TGF-beta), which is necessary for the initiation of the granulation tissue.

The biological consequences of the phagocytic portion of this phase will result in the increased oxygen consumption, phospholipid metabolism, glucose utilization, and production

of hydrogen peroxide. Polymorphonuclear (PMN) dysfunction can occur in patients with thermal injuries. The PMN will serve the role of generating electrically excitable oxygen molecules that will assist in proline hydroxylation. This helps to explain that one way to accelerate wound healing is by producing an inflammatory response.

Macrophages produce a growth factor that stimulates fibroblasts, fibronectin, and angiogenesis or neovascularization. Fibronectin will become an insoluble matrix that will cross-link with collagen and other cells or cell surfaces (platelet aggregation to injured vessel walls). Later this same fibronectin will cause clot retraction. Thus, although PMNs are present for a relatively short time, the monocytes remain longer and play a major role in the transaction between the inflammatory and granulation tissue phases.

Proliferative or Granulation Phase

The second phase of wound healing occurs approximately 3 to 4 days after injury. It is characterized by a rapid increase in fibroblast numbers and in epithelial cell mitoses, as well as an increase in the synthesis of extracellular collagen and proteoglycans. Reepithelialization of the wound will begin within hours of the injury, but it increases during this rapid mitosis as cells migrate along the new fibrin bridge. The epithelial migration will continue until cells touch one another, causing contact inhibition and signaling the end of epithelial expansion and the beginning of keratinization. Most sutured wounds will have epithelial coverage within 4 days after the insult.

Following the fibroblasts is a proliferation and ingrowth of capillaries, Angiogenesis is very complex, and involves chemotactic-stimulated migration, which causes the wound to become filled with granulation tissue (fibroblasts, macrophages, and new capillaries). The angioneogenesis will begin approximately 48 hr after injury and the ingrowing fibroblasts will produce collagen, elastin, and proteglycans within 3 to 4 days. The collagen synthesis itself is extremely active 5 to 7 days after injury. The interdigitation of collagen in the midportion of the wound with collagen along the wound edges forms a source of intrinsic wound strength.

Wound Contraction (Matrix Formation) and Remodeling Phase

The third phase of wound healing (wound contraction or matrix formation and remodeling) is best characterized by a reduction in the numbers of fibroblasts, macrophages, and wound vascularity. Fibronectin is eliminated from the wound matrix, while at the same time type I collagen will accumulate. Later, type III collagen will form with fibronectin. The contraction component in this phase is due to myofibroblasts, causing the wound to contract by 0.6 to 0.75 mm per day. This occurs when random collagen fibers are replaced by parallel cross-link fibers, yielding most commonly a flatter, softer wound with increased tensile strength. The capillary reduction occurs within 6 to 18 weeks and the remodeling process is completed between 6 to 18 months.

During the remodeling phase, the scar size is a function of wound tension, pressure, age of the patient, and oxygen supply to the area. Mechanical stress will promote collagen synthesis and deposition, often yielding a hypertrophic scar. Hypoxia will also stimulate collagen formation and deposition. As the scar matures, it will usually become more dense

as a result of loss of fluid and volume (corresponding to wound contraction). For a summary of cellular activity in wound healing, see Table 1.

Table 1. *Summary of cellular activity in wound healing*

- Platelet
  - Hemostasis
    - Aggregation
    - Coagulation
  - Release of active substances
    - Chemotactic factors
    - Vasoactive factors
    - Growth factors
    - Enzymes (proteases)
- Macrophages
  - Scavenger functions
    - Bacteria and other pathogens
    - Tissue debris
    - Neutrophils
  - Release of active substances
    - Vasoactive mediators leading to angioneogenesis
    - Chemotactic factors
    - Growth factors
    - Enzymes (proteases)
- Fibroblasts
  - Wound contraction
    - Migration into wound
    - Deposition of fibronectin and collagen
    - Contraction
  - Formation of wound matrix
    - Fibronectin
    - Proteoglycans
    - Collagen types 3/1
    - Enzymes (proteases)
    - Hyaluronic acid
- Epidermal Cells
  - Formation of new epidermis
    - Migration over wound surface
    - Formation of new tissue
  - Release of active substances
    - Vasoactive factors necessary
    - Enzyme (proteases)
    - Growth factors.

Factors Affecting Wound Healing

Local Factors

There are multiple local factors that have a profound influence on the quality of wound healing. One of the first and most important factors is the manner in which the wound was inflicted. Adverse wound healing can be expected to be seen in contused wounds, severely infected wounds, and traumatic tattooing in and about the wound. Perhaps the most important factor is the orientation of the wound in relationship to normal skin tension lines (the "relaxed skin tension lines" (RSTL) of Borges). The RSTLs are generally at right angles to the direction of underlying muscle pull, and are most easily seen in the aging wrinkle lines in the geriatric patient. Wounds crossing the RSTL are difficult to close accurately (without tension) and have a higher tendency to become widened and hypertrophic. Tension along the suture line, especially when associated with motion, tends to lead to widened, hypertrophic scars. Immobilization helps facilitate initial healing. The mechanical stress associated with movement leads to a decrease in the mitotic activity and an impairment in the ingrowth of vessels.

The nature of the injured skin is of extreme importance also. The tight, wrinkeless skin of the pediatric patient will do well in hiding scars from simple surgical incisions or lacerations. However, when the wound crosses the natural tension lines, or is closed under tension, the pediatric patient will be more likely to show scar hypertrophy than the elderly patient with loose, wrinkled skin. Also, the location of the wound is important since wounds of the scalp will contract very little, whereas a wound near the commissure of the mouth will have a tendency to cause lip deformity because scar tissue contracture is a virtually unopposed force.

The tissue type and complexion of the patient is also extremely important since an individual with a darker, olive complexion will be more likely to form pigmented, hypertrophic scars. Surprisingly, the converse can also be true in black patients where deep abrasions and burns may cause scars that will remain unpigmented for very long periods of time. A fair-complexed patient may be more predisposed to widened scars that remain reddened for longer periods of time.

Blood supply and hematoma formation are also extremely important. Ischemia will cause failure of the wound to heal. Fortunately, in the pediatric patient who has scalp, facial, or neck injuries or incisions, the blood supply is usually excellent and is rarely a problem. Hematoma formation, however, is one of the most common wound-related complications, not only causing problems with basic wound healing but also predisposing the patient to a wound infection. Thus, prevention of hematoma formation should be the goal. Where possible the surgeon should attempt to assure adequate coagulation by having the patient avoid medication that tends to interfere with clotting (anti-inflammatory drugs, salicylates, etc), and to correct bleeding disorders (in hemophiliac patients or patients with decreased platelet or fibrinogen deficiencies). If there is significant oozing at the time of surgery, and ligation and/or electrocautery is inadequagte, the use of suction drains (either the standard Jackson-Pratt or, in smaller wounds, the adaptation of a scalp vein IV infusion set to make a small suction drain) is advised. One can further assist coagulation by the use of compression dressings if the wound's supply will tolerate the external pressure. If these methods fail, the surgeon may

be well advised to pack the wound until hemostasis is secure and return after 48 to 72 hr to perform a delayed primary suturing.

Wound infection is one of the most common problems associated with suboptimal wound healing. The best treatment for infection is prevention. Factors influencing the incidence of infection include impaired local blood supply, hematoma formation, foreign bodies, patient status (nutrition, electrolytes, metabolism (diabetes), anemia, and drug ingestion - steroids or antimetabolyte chemotherapeutic agents), and surgical technique. It is interesting to note that in the early stages both infection and wound healing are very similar in their cellular and vascular responses to insult. It is well known that a delay in wound healing occurs when wounds are inoculated with bacteria. Also, it has been shown that there is a decreased tensile strength in wounds contaminated with *Staphylococcus.* The principal biochemjical abnormalities in infected wounds appear to be a disturbance in collagen metabolism, probably via lysosomal enzymes present in PMN cells, and a depressed effect on fibroblasts leading to a disturbance in collagen synthesis. In a wound that is significantly contaminated, the most efficient method for reducing the risk of infection is to dissect and remove devitalized tissue without attempting to remove an entire wound surface. When this is impractical, one can attempt to dilute the bacteria by irrigation with a physiologic saline solution, employing the physiologic solution in combination with some type of antibiotic (bacitracin is most commonly used), or using a surface antibacterial agent (povidone-iodine (Betadine) is most commonly used in this fashion). Once a wound becomes infected, closure is not advised since that may cause the formation of an abscess. Instead, it is best to leave the wound open until signs of inflammation are gone, and then consider secondary wound closure.

Surgical Technique

Surgical technique is very important in the creation of an adequate wound. Borges and Alexander recommend that the orientation of the wound in relation to normal skin lines of tension (RTSL) is possibly the single most important factor in scar prognosis. The lines of tension should be at right angles to the direction of the underlying muscle pull. Any wound that crosses the lines of skin tension will have a tendency to widen and hypertrophy. The width of most scars is proportional to the magnitude of skin tensions. Thus, not only the planning of incisions is essential, but also the conversion of suboptimal wounds using rotational flaps or Z- or W-plasties is important. Also, the reduction of skin tension by the use of undermining and approximation with multiple layers of wound closure is essential. The concept of having wound edges in apposition prior to the final subcuticular or skin suture layer is very important. In the face, the dynamic skin tensions are perpendicular to natural skin wrinkles and parallel to the direction of contraction of the underlying mimetic muscles. Thus, a linear scar intersecting the wrinkle lines can result in severe scar tissue contracture. When the directional orientation of a wound tends to predispose to a widened, unattractive, or hypertrophic scar, it is very important that the physician warn the patient and the family of the probability of unacceptable scar formation and the likely need for scar modification later. At a later date (usually 12 months or later), the scar could be revised and placed in more optimal skin tension geometric patterns with either a W-plasty or a Z-plasty, making the orientation of a portion of the wound perpendicular to dynamic skin tensions.

Wound closure techniques depend on the type of wound. Basically, two types of wound exist: (a) one characterized by loss of tissue; and (b) one with no evidence of loss of tissue. Usually primary closure can be accomplished in the wounds without loss of tissue, whereas wounds with significant tissue loss require flaps or grafts to close the defect.

Timing of wound closure is also important. The main decision is whether to close the wound primarily, or whether to delay closure. In the head and neck, time is not the major criterion. If the wound appears to be in an area with minimal devitalized tissue, and without evidence of significant inflammation, usually closure can be accomplished even several hours after the initial injury. If, however, there is severe devitalization of tissue, or if there is evidence of wound infection, open wound management prior to delayed primary closure is reasonable. This can be accomplished by the use of fine mesh gauze covered by a sterile dressing.

The method of wound closure is important. Most commonly, sutures are inserted to close both deep and superficial layers. Dermal sutures are utilized to reduce skin tension. These dermal sutures serve as a precaution against wound dehiscence. Most commonly, minimally reactive absorbable sutures such as polyglycolic acid or polyglactin sutures are used. Not only will these sutures reduce skin tension, but also they will tend to obliterate potential dead space and reduce hematoma formation. The skin is then closed with 5-0 or 6-0 suture using the technique of approximating the midportion of the wound and then, with further sutures, bisecting the residual portions of the unclosed wound. For skin sutures, one might utilize a 5-0 or 6-0 nylon-propylene suture on a swage cutting needle. In wounds that are stellate or jagged-edged, percutaneous suture closure of the skin edges gives much better results than taping.

General Factors

Age

Wounds in children tend to heal more rapidly and actively than those in the older patients. This is frequently due to increased blood supply, as well as less systemic disease. However, there does appear to be a slight increase in the tendency for reddened or hypertrophic scars in children. Aging affects all stages of wound healing. In open wounds in the elderly patient, the time before contraction begins is lengthened, the healing rate is slower, and the final degrewe of epithelialization is late. In the elderly, the breaking strength of incisional wounds is lower and the bursting force is less. The rate of wound healing varies inversely with age. Collagen and elastin are markedly altered in the elderly with soluble collagen decreasing, whereas insoluble collagen increases with age. Elastin also increases in the elderly, but the quantity of elastin fibers decreases, causing atherosclerosis and poor wound vascularization.

Nutrition

Malnutrition plays a major role in wound healing complications. Proteins are necessary for collagen synthesis, fibroblast proliferation, revascularization, and the formation of lymph channels. Severe protein deficiency will inhibit all phases of wound healing, including humoral and cell-mediated antibody responses, and phagocytic action, and it will delay fibrous

tissue matrix formation. Hypoalbuminemia will cause edema and interfere with microvascularization. Vitamin deficiencies will also affect wound healing. Specifically, vitamin A enhances epithelial rate of collagen synthesis, collagen cross-linking, and synthesis of glycoprotein and proteoglycans, as well as collagenase production. Viral and bacterial infections are enhanced by vitamin A deficiency, and vitamin A supplements reverse the adverse effects of glucocorticoids.

Vitamin B serves as a cofactor for enzyme reactions, and is required for a white cell function and antibody formation. Clinical vitamin B deficiency states do not appear to delay wound healing. Vitamin C is required for hydroxylation of lysine and proline and collagen synthesis, capillary formation and strength, macrophage migration, neutrophil function, and inflamatory response, as well as immune reactions. Since humans do not store vitamin C, deficiency may occur rapidly in chronically ill patients. Vitamin D is essential for new bone formation and for transport and metabolism of calcium, but there is no evidence that a deficiency state reduces bone healing in human surgical subjects. Vitamin E has anti-inflammatory properties because it reduces functioning free radicals. It also may improve cell-mediated immunity and inhibit platelet aggregation. Vitamin K is essential for the synthesis of clotting factors VII, IX, X, and prothrombin. Deficiencies of vitamin K lead to increased risk in bleeding diatheses and hematoma formation. Finally, zinc deficiency can result in retardation and delay in the repair process as a result of impairment of synthesis of nucleic acid and protein (especially collagen). It is also essential in cell membrane function and for vitamin A transport. although there is no therapeutic value in the administration of zinc in the nondepleted state, zinc-deficient patients may be aided by an intake of 220 mg of zinc sulfate three times a day. Zinc exscess (greater than 40 mg/dL) can impair wound healing.

Anemia

Anemia, especially in hypovolemic states, will cause tissue hypoxia and result in poor wound healing. Anemia is also associated with an increased infection rate, although it is usually difficult to separate anemia from problems leading to poor nutritional states.

Diabetes Mellitus

Patients with diabetes mellitus have wound complication rates of up to 10% incidence. This is usually related to infection, although additional factors include neuropathy and atherosclerosis. Improved management of diabetes mellitus has reduced the risk to the patient. Specifically, preoperative modification of insulin requirements is important. Diabetics also appear to have white cell defects, which contribute to their propensity for postoperative wound infections. However, the greatest improvement in diabetic wound management is related to proper control of blood sugar in the perioperative period. Thus, it is important to make certain that the diabetes is well controlled with the appropriate endocrinology consultation.

Medications

Glucocorticoids (corticosteroids) exhibit significant inhibitory effects on wound healing. Low doses (less than 10 mg prednisone per day) have minimal, if any, effect in adult patients, but repeated high dosages (40 mg or more prednisone per day) will adversely affect

wound healing, including (a) reduction of fibroblastic proliferation and granulation tissue; (b) polymorphonuclear leukocytes with reduced motility, phagocytosis, and adhesiveness; (c) reduction of lymphocytes in tissues and blood; (d) macrophages with reduced mitosis and phagocytosis; (e) vasoconstriction resulting in reduction of blood and nutritional supply and nutrient supply; and (f) reduction of synthesis of collagen, proteoglycan, and glycosaminoglycan.

Anticoagulants such as sodium warfarin (Coumadin) and heparin increase the risk of hematoma formation, bacterial infection, and wound dehiscence. Nonsteroidal anti-inflammatory agents (aspirin, phenylbutazone) will decrease tensile strength of wounds, and increase the risk of bacterial infection and hematoma formation.

Although antineoplastic agents have been implicated in delayed wound healing, the results are conflicting. It has been suggested that actinomycin D or bleomycin in the perisurgical period may cause some impairment of wound closure. However, it has also been surmised that although limited clinical data do not confirm significantly diminished wound healing when these agents are used, most surgeons prefer withholding chemotherapeutic agents in the first 7 to 10 postoperative days.

Colchicine, an anti-inflammatory agent, interferes with fibroblasts and their secretion of collagen precursors, as well as stimulates collagenase and reduces the secretion of histamine granules from mass cells. It was used by Peacock in combination with penicillamine or beta-aminopropionitrile in the treatment of keloids and hypertrophic scars. Penicillamine can reduce the tensile strength of healing wounds by affecting collagen cross-linking, and can also be used to modify hypertrophic scars. Large doses of penicillin would theoretically yield enough penicillamine to cause wound healing problems, but in fact, this has not been shown to be clinically significant. Beta-aminopropionitrile is a lathyrogenic agent that yields reduced tensile strength adn reduces collagen fiber formation. It has also been utilized to modify adhesions and keloid formation.

Jaundice, Uremia

Theoretically, jaundice can affect wound healing in the realm of a poor absorption of vitamin K, and thus decreased clotting factors yielding increased wound hematoma. Hepatic dysfunction will also cause problems with protein metabolism. However, the role of jaundice in predisposing to wound healing complications is multifactorial in that these patients are also usually malnourished and have protein abnormalities. Thus, one must consider not only the effect of the hepatic dysfunction on clotting mechanisms but also the patient's dietary intake. Uremia will also show evidence of reduction in wound strength with a higher incidence of wound dehiscence. Uremia will cause inhibition of fibroblast ingrowth. Again, the poor nutritional status of the uremic patient may be of primary importance.

Thus, as this portion of the chapter illustrates, optimal wound healing is dependent upon multiple factors. The surgeon must be moderately cognizant of the effect of the above elements so that he can optimally prepare his patient for surgery.

Hypertrophic Scars and Keloids

When one is dealing with either hypertrophic scars or keloid formation, one must realize that there may be some intrinsic abnormality in the patient's physiologic reaction to tissue insult that will cause difficulties in attaining optimal satisfactory results. In reviewing the literature in preparation for this chapter, the author finds the words "hypertrophic scar" mentioned in conjunction with keloid formation, but rarely does he find a definition of the term. Most commonly, it is utilized by various authors to mean noticeable scar tissue that may be raised, reddened, or widened, but it is not the exuberant deposition of huge, raised amounts of collagen in healed skin wounds. Peacock et al have clinically differentiated between the two entities by defining a keloid as an excessive collection of scar tissue that has no resemblance to the shape of the original wound and extends beyonds the confines of the wound, whereas a hypertrophic scar is excessively large but retains the original shape and remains within the confines of the original wound.

In both conditions, there appear to be abnormalities in both the synthesis and degradation of collagen. Specifically, Cohen and Diegelmann have shown that collagen synthesis in both conditions is greater than in normal skin, and that in the keloid collagen, synthesis is greater than that in hypertrophic scars. They hypothesized that hypertrophic scars are merely raised because the rate of collagen synthesis was greater than the usual scar tissue. It is not clear whether the increased collagen synthesis in keloids is due to an increased number of collagen-synthesizing fibroblasts, a normal number of fibroblasts stimulated by some factor to make more collagen, or fibroblasts growing at a rapid rate.

The tendency to form hypertrophic scars is related to several significant factors. One factor is the tension placed upon the scar tissue. Wounds that tend to have more tension will be at a higher risk to form excess scar tissue. This tension may be in the form of crossing the RSTL of Borges in regions of tension near joints or in circumferential scars. At times it may be necessary to splint the area or immobilize the region (if possible).

The age of the patient is also important since fibroblastic activity in patients under age 30 is much greater than in patients 60 years of age and over. Thus, the chance for a bulkier scar is greater in the younger patient, although the remodeling process may cause the scar to flatten nicely, and in the early stages it may show evidence of hypertrophy.

Crushed tissue, large areas of dead space, and infection are also stimuli for scar hypertrophy. Finally, noticeable skin suture marks may occur when the sutures are left in place for excessive periods of time. Specifically, in the presence of percutaneous sutures, epithelial cells will tend to migrate downward, following the suture. If the sutures are removed before the 8th postoperative day, this epithelial invasion will regress, leaving minimal deformity. However, after 8th day, the suture track reaction to the foreign body becomes extremely intense and a permanent deformity (permanent needle scars) may result.

Race may also play an important role in healing, especially in patients with keloid formation. Large hypertrophic scars can occur in fair-skinned individuals, but they are more commonly seen in darker-skinned persons. Keloids are most commonly seen in black-skinned individuals.

The treatment of hypertrophic scars and keloid formation is obviously one of prophylaxis. In patients with a history of increased scar formation, one should make every attempt to reduce the tension on the wound, place incisions within the appropriate skin creases or areas of reduced tension (or reduce the tension by primary W- or Z-plasties or rotation flaps), and remove skin sutures before the 8th postoperative day. However, in spite of the above treatments, one still may encounter hypertrophic scars or keloid formation. Intralesional injections of triamcinolone are useful in modifying the wound healing. One can inject triamcinolone (10 mg per cc) utilizing a 25- to 27-gauge needle. If hypertrophic scar formation (or keloid) is suspected, the injections can begin within the first 3 postoperative weeks. Further 3-week courses may be given if there is itching in the scar, or if the scar hypertrophy appears to be unresolved. In patients who do not have a history of hypertrophic scar formation, one can initiate injection at the first sign of the problem. If the result of injections is unsatisfactory, then one could proceed with scar revision and immediate injection of steroids, or if the lesion is in a readily accessible and optimal site, pressure dressings can be applied. The pressure appears to reorient collagen bundles parallel to the surface of the skin and will cause a reduction in the level of chondroitin sulfate associated with scar hypertrophy. The disadvantage of this treatment is that it must be maintained continuously for at least 9 to 12 months, since premature release of the pressure will cause recurrence of the lesion. Also pressure dressings are not effective in lesions that have been present for 6 to 12 months or longer.

Radiation therapy has been a controversial modality utilized for the treatment or prevention of keloids and hypertrophic scars. It has no effect on established lesions, but it may be of benefit in the prevention of lesions in the immediate postinjury or revision case. Doses of 1.000 rad, given singly or in divided doses can suppress proliferation of fibroblasts. One recommendation is giving three to four daily treatments of 300 to 400 rad to each surface with a 100-kV machine. This would be a very unusual course of therapy in the pediatric patient, especially over active bone growth sites or in the region of endocrine glands. For the keloid or hypertrophic scar that has been present for 6 months or longer, the treatment will usually depend upon the size and location. As we have mentioned, radiation therapy and pressure dressings will not be successful in the management of these lesions. Intralesional steroid injections may be of some benefit in the long-standing case. Again, the injection can be performed using 10 mg per cc triamcinolone injections at 3- to 4-week intervals. If this is unsuccessful, then the keloid could be excised and the area closed primarily, if possible. If primary closure is possible, it would be recommended that one perform a subtotal excision. Specifically, the bulk of the central keloid would be excised with a very minimal keloid frame left on the sides of the wound to act as a splint to prevent skin retraction. The central portion of the keloid should be removed as deeply as possible. Then the wound could be either closed primarily or, if this is impossible, then a skin graft placed to cover the bed. In the postauricular area, one should excise the keloid completely and cover the defect with a full-thickness graft from the groin. If the patient has extensive resurfacing needs, it is recommended that one test a small area to see if the patient will heal appropriately, rather than try to graft the entire area at once. All grafts should be extremely thin (10/1.000 inch) split-thickness skin grafts to avoid hypertrophy at the donor site.

The complications of the therapy for hypertrophic scars and keloid formation can include local effects of the steroid injections, such as atrophy of the skin with telangiectasia. Also, complications include recurrence of the hypertrophic scar and keloid.

Burns - Lip, Oral Cavity, and Neck

Burns involving the head and neck region in children involve specific special problems. Often these burns involve the ignition of clothing. The mortality rate for victims whose clothing is ignited is at least four times higher than those whose clothing is not ignited; the severity and extent of injuries is almost 100% greater in the former than latter group; the percentage of full-thickness injury is six times greater, and the number of days of hospitalization is approximately 60% greater. The injuries are most likely due to open space heaters, kitchen ranges and matches, flammable liquids such as gasoline or pain thinners, or hot water.

Electric burns in infants and young children will most commonly occur when the child puts an electric plug or cord in the mouth. Thus, the lips, oral commissure, and tongue are the most common areas involved in head and neck electric burns. Electrical burns can be of two natures: arc and contact. The arc type of burns will involve temperatures of 2.500°C to 3.000°C, causing charring of soft tissue and bone. These are the types most comonly seen when there is contact with live electrical sockets. Physical examination will most commonly reveal a third-degree burn with a centrally depressed crater, and a slight, pale elevation of the surrounding tissue. The surrounding blood vessels are markedly injured, as are the sensory nerves. Immediately after the accident, one finds that the area of necrosis is not usually apparent, and since it may take several days to demarcate, immediate surgical debridement or reconstruction is usually contraindicated.

For electrical burns, special acrylic or Silastic appliances are usually not immediately placed in the wound. Early management includes the use of antibiotic ointments in an attempt to prevent severe infection. After the extent of tissue destruction can be determined, reconstructive procedures will involve excision of the involved tissue. If the burn is in an anatomically favorable position, immediate reconstruction after debridement is possible. However, when the burn involves a corner of the mouth, it is usually preferable to await spontaneous separation of the eschar and to allow some softening of the wound before definitive repair. Early excision can also risk the sacrifice of uninvolved tissue. In these instances, special acrylic or Silastic appliances placed at the commissure to prevent severe adhesions may be useful. Final reconstruction can be achieved after a significant period of time has elapsed and the scars are allowed to soften (most frequently 1 year is a reasonable time frame).

In the reconstruction of the patient with a thermal burn to the face, the surgeon must be cognizant of certain wound healing problems following the injury. Specifically, wound contraction will frequently be present in both second- and third-degree burns. Four areas in the face are markedly susceptible to contracture due to mobility and flexibility. These are the eyelids, the cheeks, the lips and oral tissues, and the cervical region (neck). The upper lip is pulled superiorly toward the columella via intrinsic contracture forces, whereas the lateral portions of the upper lip are pulled towards the cheeks. The lower lip and chin are pulled downward by both intrinsic scar tissue contracture and also the muscle contractions and scar contracture seen in the cervical region. The oral cavity may have circumferential contraction (purse-string manner) limiting the patient's ability to open the mouth. Similar circumferential contracture can occur in the nasal vestibule region. An interesting observation is that the

position of comfort in the burn wound is one of contracture. Also, the burn wound will contract until an opposing force is met. This opposing force can be splinting and/or exercise. This therapy may be very effective in reducing of contracture in the lip region and the neck.

The timing of repair in the facial burn patient is extremely important. One must keep in mind that allowing the wound to demarcate and show the areas of most contracture is not unreasonable, and therefore a concept of "slow is always bad and fast is not always best" is reasonable. A conservative approach including topical antibiotic therapy, debridement of scar and eschar, and eventually skin grafting is recommended in burns of the face. This conservative approach is advocated because the diagnosis of depth in most facial burns in the early posinjury wound is often misleading. An exception would be partial-thickness burns thatcan be converted to full-thickness loss from infection during the healing phase. In these patients, excision by shaving the involved tissue, and immediate grafting is warranted, oftenbetween the 3rd and 5th postburn days. Usually it is safe to perform the definitive correction of facial burn deformities at approximately 1 year or longer after the injury, when scars and initial grafts have had a chance to mature. In the intervening time between the injury and definitive reconstruction, splints and pressure masks, as well as local steroid injection, may yield improved results with hypertrophic scars. Earlier intervention is indicated in regions around the eyelid where damage to the cornea may be possible, or around the mouth where lower lip eversion causes drooling or severe neck scarring.

In reconstructing the face, one should divide the face in a map as shown. Any flap or graft applied to the face should, if possible, cover the entire aesthetic unit in order to avoid a patchwork appearance. The stages of treatment ni these patients include the acute period, which has a pregrafting and skin grafting phase, and a chronic period, which requires a waiting period (1 year or longer, as mentioned above), as well as an early and final reconstructive phase. Since the early or acute periods have been discussed above, we will concentrate here on the oral (lip) and upper neck regions. In the waiting period, one can apply stents, either intranasally or in the oral cavity, or pressure dressings over the entire face or neck. Also during this phase, the patient is encouraged to exercise the periorbital regions and the mouth and neck. The early reconstructive stage mainly involves temporizing measures such as Z-plasties or skin grafts in a nondefinitive fashion.

The final recontructive phase is not begun until hypertrophic scars have shown signs of becoming less active and scar tissue contracture appears to be stable. The relief of contractures with local flaps and Z-plasties is the first objective of therapy. Secondly, skin grafts are usually applied where possible in preference to skin flaps. Thick split-thickness grafts are much better than thin grafts, since they will be less likely to contract. Burn deformities of the perioral area and chin can be divided into upper lip, lower lip, and neck. In this region, one must again divide the area into aesthetic units as shown. The incisions for this area are shown.

When one encounters ectropion of the upper lip, this is best corrected by full-thickness grafts from the supraclavicular or postauricular regions, or by very thick split-thickness skin grafts if one cannot obtain full-thickness tissue. One should attempt to graft the entire aesthetic unit between the nasolabial folds. When the scar is excised from the upper lip, it is very important to leave some residual scar on the philtrum to avoid a flat upper lip. The contracture in the upper lip should be released by making an incision near the base of the

nares to allow the lip to move inferiorly. Scars in the area of the philtrum are left in place in an attempt to leave sufficient underlying tissue to give good philtral projection.

Reconstruction of the lower lip and chin again follows lines of aesthetic components. When scar tissue is excised from the lower lip and chin, one must remember that a circle of scar should be left on the apex of the chin, since this gives a slightly better profile. Microstomia is a very difficult problem, and may initially be treated by stenting and dental appliances. Deifinitive surgical repair would require one of a multitude of possible flaps, which is beyond the discussion of this chapter. Scar tissue contracture of the neck is usually corrected with multiple Z-plasties to lengthen the web-like vertical bands, and by excising the majoruity of the scar and replacing it with thick split-thickness grafts. Postoperative splints or neck braces are also helpful. When the scar extends over the chin or lower border of mandible, one attempts to excise the area so that a splint can be made with an extension to press against the grafts. In the cervical region, one can attempt to have an interval of 4 to 5 days between scar excision and skin grafting to allow a decent layer of granulation tissue to begin to form. Postoperatively, a splint is applied that will keep the neck extended, mold the chin-neck angle, and apply even pressure over the grafted area. Splint application is usually completed during the 2nd week postgrafting. The use of regional transposition flaps, distal flaps, or microvascular flaps is also occasionally helpful but again is beyond the realm of this chapter.

At this time the author would like to mention that when one finds hypertrophic scarring in burn patients, the surgeon is faced with a decision of an intramarginal or extramarginal excision of the hypertrophic scar. In an evaluation by Engrav et al, it was found that intramarginal excision of lesions seemed to be more efficacious in the younger patient. There are two theoretical reasons for this. First, one would feel that a scar base is an ideal area for wound healing. Second, the perimeter of the scar tissue acts as a physical restraining splint, thus reducing the effect of tension on the margins of the incision. Undermining can still be utilized even in intramarginal excisions unless the surrounding tissue has altered vascularity. In some cases, it is even reasonable to perform an overgrafting technique in which all that is removed from the hypertrophic scar is the epithelial portion, leaving the underneath thick, fibrous tissue in place as a bed for a thick split-thickness or full-thickness graft. This reduces the amount of healing necessary, and also reduces wound tension.

Scar Revision

Scar Analysis

A discussion of scar revision is, in and of itself, a topic that could be a complete chapter, or in some cases, a small monograph or book. Thus, this discussion will be limited to some basic principles and concepts, rather than a multitude of specific cases.

The surgeon should be cognizant of evaluating multiple factors involving the patient and the scar areas before proceeding with the therapeutic recommendation. One of the first concepts is to evaluate the individual host in regard to tendency to form keloid or hypertrophic scars in other areas of the body, or with a family history of similar problems. One should also evaluate the person's psychological makeup in order that one is not trapped into dealing with a patient who has unreasonable expectations. This is especially true if

previous scar revisions have been unsuccessful. One may find that the patient may indeed have a lesion that cannot be improved. Younger patients will have greater skin tension, and thus will have a tendency to cause scar spreading and hypertrophy. This must be pointed out to the parents of pediatric patients, especially in areas where there is constant motion. The scars in these pagtients will usually become redder and remain erythematous for much longer periods of time.

One must also evaluate the time lapse between the injury and the patient's request for revision. Some surgeons feel that scars can be revised 2 to 4 months after injury in adult patients and older children, whereas in children less than 7 years of age, one should wait at least 6 months before revision. The rationale behind this concept is that the scar is most noticeable between 2 weeks and 4 months, and thus if revision is done, there will be a more obvious improvement with greater satisfaction from the child and parents. However, this author feels that since scar tissue continues to remodel and improve up through at least 12 months postinjury, one is justified in waiting until between 6 and 12 months postinjury before attempting scar revision. However, if there is functional deformity such as ectropion or difficulty in neck extension or opening the mouth, early revision is definitely warranted.

The surgeon must also pay special attention to various characteristics of the scars, which include pain, step-off deformities, pigmentation, length, shape, depression, width, hypertrophy, and direction. The treatment of painful scars is difficult and most likely will not lead to great success in the alleviation of pain. Distortion of anatomic landmarks is significant and is a definite indication for early repair, especially with ectropion or difficulty in extending the neck or opening the mouth. Step-off deformities along the vermilion-cutaneous border of the mouth or along the nostrils will require opening the wound and usually a multiple-layer closure. Stitch marks may indicate a tendency toward hypertrophic scar formation unless the previous surgeon's notes show that the sutures were left in for a lengthy period of time. If stitch marks are present, this may require dermabrasion, planing with the scalpel, or, if extremely pronounced, excision or incorporation in a running W or geometric pattern revision. Pigmented scars may be revised with dermabrasion or surgical planing if the lesions are superficial, but if they are deep-seated, complete correction may require full-thickness skin excision. The patient and family must be prepared for full-thickness excison if the latter is the case.

When planning a scar revision, the surgeon must remember that the scar has three components: the line, the contour, and the color. It is the integration of these three components into one final entity, the scar, that makes a patient either satisfied or dissatisfied with the result.

The line of the scar may be extremely minute, but still be visible if it passes across or near normal structures that are misapproximated, or it may be noticeable because it is widened (even though it is flat).

The contour of the scar involves a three-dimensional depression or elevation. Both depressed and elevated scars contribute to many patients' dissatisfaction. Scar contracture may not only be of significance in itself (this leads to scar hypertrophy or keloid formation), but also may cause displacement of normal structures such as the eyebrow, lower eyelid, lip, or nasal ala or columella. The contour may be altered also in the trapdoor type of scar.

Finally, the color of the scar is significant because any deviation from the color match with the surrounding tissue will usually make the scar noticeable. Examples of this are deep pigmented scar tissues in black individuals, darkly pigmented scar tissue in Caucasians, traumatic tattoos, or the markedly reddened scars seen frequently in the pediatric population. Thus, in selecting individuals for scar revision, each of the above factors must be taken into consideration when counseling the patient. The patient and his family must be reminded that a scar line is going to be inevitable when any scar is excised. Thus, one can only attemtp to improve the result, but one cannot guarantee that an improvement will come to pass.

When discussing timing with a patient, one again must take into consideration such problems as malalignment, a spreading scar, a cnotracted scar, or trapdoor scar. Whereas color changes may improve with time, and thus repair could be delayed in these cases, the trapdoor defect or functional deformity caused by severe contraction should have early intervention.

Methods of Scar Revision

There are two factors in wound healing that are beyond the control of the surgeon when dealing with scar tissue and scar revisions. These are the area where the wound has occurred, and the amount of normal tissue that is lost. The surgeon should attempt, when at all possible, to place incisions in relaxed skin tension lines (RSTL). Unfortunately, there are occasions where the skin tension is considerable in any direction, and this will widen even the most meticulously revised scar. Scars overlying joints, or where there is marked skin motion, will also tend to be difficult to revise satisfactorily. The choice of excision and revision techniques will be discussed later, but during scar excision the scalpel should be held at right angles to the skin to prevent beveling. One can either utilize interrupted skin sutures with subcutaneous closure of an inverted absorbable suture, a single layer running closure (this is extremely unusual), or a running subcuticular suture to oppose skin edges after the subcutaneous tissue is closed with an absorbable inverted interrupted suture. The running subcuticular suture should be a 4-0 nonabsorbable material (5-0 nonabsorbable would be adequate if the wound is very short). When using the subcuticular suture, the wound edges may be taped together with Steri-Strips.

Simple Excision - Primary Closure (Fusiform Excision)

A simple excision is the most basic type of scar revision. This can be utilized in scars that parallel the RSTL and are reasonably short. The long axis of the incision must follow the RSTL even if it is curvilinear. The excision should not cross anatomic landmarks such as vermilion of the lip. Occasionally an M-plasty is needed for shorter scars. A variation of this technique is serial excision in which the scar may be extremely wide and thus must be excised in multiple stages. One disadvantage of this is the multiple-stage technique and thus the use of skin expanders has reduced the need for serial excision.

Occasionally the excision of a lesion and/or scar may be such that a single advancement flap is optimal (often in the forehead or facial groove regions). In order to reduce tension on the flap as it is advanced, triangles at each end of the flap (called Burrow's triangles) are removed, and the skin undermined in advance. At times, a bilateral advancement flap is necessary, as is often seen near the philtrum and columella of the upper lip and nose. Here, one performs a V-to-Y advancement.

Rotation Flaps

Rotation flaps are extremely useful in not only excision of small lesions, but also large lesions, especially in the nasolabial crease, and thus near a natural skin line. Also, large areas of cervical or buccal scars may be treated with large rotation advancement flaps. The general principle of this is shown, where a scar is excised and the flap is undermined. A triangle is removed at the distal end of the flap to reduce a dog-ear deformity.

Z-Plasty

Z-plasties are one of the most widely utilized scar revision techniques in reconstructive surgery. The principle of this technique is threefold: (a) the lengthening of the linear scar contracture, (b) realigning the scar within lines of minimal tension, (c) a breaking up of a straight line scar into a multiple Z configuration. A Z-plasty results from a transposition of two triangular flaps. The central line of the Z-plasty is called the central limb or incision. An attempt is made to construct the limbs of two equilateral triangles so that the angles are 60° with limbs of equal length. After excision of an unsightly scar, the flaps may be undermined and then transposed in a more satisfactory configuration in regard to the RSTL. Thus, the rotation allows these scars lie in a more satisfactory alignment but the price paid is that of two additional scars. Usually this trade-off is beneficial. Although the above discussion talked about equilateral triangles with 60° angles, one can adjust the lengthening by altering the angles at the apices of the transposition flaps. The greater the angle, the greater the lengthening. However, the majority of Z-plasties will have angles from 30° to 60°, with a 30° Z-plasty increasing the length approximately 25%, a 45° Z-plasty increasing the length 50%, and a 60° Z-plasty increasing the length by almost 75%. This technique can be utilized in a serial fashion and thus one could have multiple Z-plasties running along a very long line in an attempt to break up a prominent scar. Usually, certain basic principles can be followed to simplify Z-plasty utilization: (a) In the majority of cases, a simple Z-plasthy with an angle from 45° to 60° is optimal. (b) The minimum angle utilized should be at least 30°, since an angle less than this would risk avascular necrosis of the tip. (c) Long scars are best treated with a multiple Z-plasty technique. (d) The central limb is placed along the scar line or line of contractuer, with the lateral limbs drawn in one of either two directions, but utilizing the principle that the lateral limbs, which are parallel to each other, should also parallel the RSTL. If drawn incorrectly, they may lie perpendicular to the RSTL, and thus negate the entire concept of the scar revision.

W-Plasty

The concept of the W-plasty, or zigzag-plasty, is that of a series of interposed triangular advancement flaps that break up a straight line scar. Once the eye perceives any part of a scar, it will immediately follow it and see the entire scar. Multiple tiny Z-plasties are more likely to be visible to the eye than are running W-plasties, because the Z-plasties are more predictable and will lengthen the scar line. To perform a W-plasty, the scar is excised along with some normal tissue in a zigzag fashion. The completed W-plasty causes the scar to be irregular without adding to its length. It also breaks up the forces of contracture, and thus lessens scar tension. Since the W-plasties are irregular, this makes the scar less visible without causing any increase in the longitudinal aspect. The angles between

the interposition flaps are usually between 45° and 75°. The main drawback of the running W-plasty is a sacrifice of some healthy skin plus the difficulty in making the interdigitating parts conform to one another.

A Z-plasty has an advantage in that it elongates the contractile scar while changing the scar's direction, and thus will more likely place the resultant wound more nearly parallel to the RSTL lines. The Z-plasty will also utilize all the available skin without excising normal tissue. Finally, the Z-plasty will allow one to adjust the location of adjacent or displaced tissue. The W-plasty is an excellent procedure in the forehead, temporal area, chin, and cheeks, as well as the lower lip. It has an advantage of being less likely to displace normal anatomic landmarks since it does not involve the transposition of tissue. The W-plasty is indicated where the scars are perpendicular or nearly perpendicular to the lines of minimal tension. Unfortunately, a W-plasty will increase the tension in the scar because it does sacrifice some normal tissue and thus should be utilized only where there is adequate surrounding tissue. When the tissue surrounding the scar is somewhat deficient, or when the scar should be elongated, a Z-plasty is preferable.

A variation of the running W-plasty is a geometric broken-line closure, which is a design of irregular, unpredictable geometric figures as shown. The geometric broken-line closure came into use because the W-plasty was predictable. Therefore, one can excise a scar and make irregular, unpredictable geometric patterns to attempt to correct this deficiency of the W-plasty. These geometric closures can be triangles or rectangles of varying sizes, configurations, and patterns. Again, all incisions should run as close as possible to parallel the RSTL. This type of closure will not lengthen the scar but does cause the sacrifice of some normal tissue. It is usually wise to design the flaps to have a progression of heights so that the height is greater in the middle and less at either end.

Dermabrasion

Dermabrasion is a method of modification of facial scars by mechanically removing the epidermis and some, but not all, the dermis. This can be used as an adjunct to scar camouflage and occasionally can be used on the acutely injured patient to feather or touch up the lateral margins of the laceration or traumatic area. Dermabrasion can also combine this with a running Z-plasty, W-plasty, or geometric broken-line revision, but one must be careful to preserve hair follicles and sebaceous glands.

Dermabrasion is performed using any one of a number of devices designed for this procedure. These devices are lightweight and should have a very rapid RPM (ideallly, 20.000 or greater). Although one could use wire brush abraders, these can be dangerous in the hands of the novice, and therefore diamond fraises of varying shapes (tapered, wheels, and cylinders) are available. The surgeon should be careful to protect both the patient and the operative team with the use of safety glasses, and also by keeping surgical sponges out of the operative field. This technique is best utilized for smoothing out depressions in the skin, including depressed scars, mild acne pitting, and fine wrinkles. It should not be utilized for keloids or hypertrophic scars. Postoperatively, one can utilize multiple types of wound care, including Telfa or Adaptic dressings, Debrisan ointment, or any of multiple antibiotic ointments.

The patient must be well informed about postoperative care and potential complications with dermabrasion. Specifically, erythema will occur and may last for 3 months or longer. Areas of telangiectasia may occur temporarily or less commonly be permanent. Small inclusion cysts (milia) are also a possibility. Hyperpigmentation has occurred, especially in dark-skinned individuals, and thus this technique should be used very sparingly in these patients. Hyperpigmentation can be brought about by exposure to sunlight, which patients are advised to avoid during the summer months. If sun exposure must occur, then the patient must utilizde sun-screen of a high sun protective factor. Hypopigmentation can also occur postoperatively. The surgeon has minimal control over this hypopigmentation. Individuals who tend to form keloids or hypertrophic scars are not good candidates for this procedures. Finally, the patient must be warned that it may be necessary to perform more than one dermabrasion procedure.

The "Trapdoor" Scar Deformity

The trapdoor deformity is a description of a healing problem where one has the effect of retraction of curved scars, giving rise to an outward bulging projection over the surrounding skin. These scars are frequently seen in lacerations that are deep and have beveled edges. Multiple theories have been put forth concerning the trapdoor deformity, including lymphatic and venous obstruction, the piling up of fatty tissue, a bevel-shaped flap, scar hypertrophy, and mechanical factors. Indeed, the trapdoor deformity probably comes about because of a combination of factors. Specifically, when one has a horse-shoe-shaped, bevel-edged laceration, there may be three very important factors playing significant roles: (a) retraction of the curved scars with a depression in one area and a thickening of the skin over the beveled edge, with a bulging of the curved flap; (b) an interruption of tension lines by the curved scar; and (c) local movements of muscles in varying areas of the body (especially around the nasal and oral cavities), which leads to unequal retraction of muscle segments caused by differential divisions at the time of injury. The combination of these factors frequently will lead to the trapdoor deformity.

The trapdoor treatment depends upon the size of the scar. If the scar is small, then one can excise the area in a fusiform fashion and place the final scar parallel to the RSTLs. However, one must follow certain specific rules in utilizing this technique: (a) the skin incision must be perpendicular to the surface, (b) there must be slight undermining of the border, and (c) the deep layers must be reconstructed and closely approximated with subcuticular stitches. If the wounds are large, and thus preclude a fusiform excision along the long axis of the RSTL, then one should perform serial excisions in approximation to the RSTL, utilize Z-plasties or V-to-Y plasties in scars that are in an alignment that is not situated in the direction of the RSTL, and utilize a running Z-plasty or W-plasty on long scars that are perpendicular to the RSTL. The W-plasty is extremely useful in trapdoors along the mentum and chin regions.

Complicatinos of Scar Revision

The cosmetic surgeon is faced with similar complications to those found in any other part of the body. These include hemorrhage, hematoma formation, infection, and wound dehiscence. However, certain other difficulties tend to surprise and frustrate both the surgeon and patient. These include reactions to the suture, which are usually heralded by the use of

subcutaneous or subcuticular catgut or chromic catgut. In these cases, the wound will become erythematous and there will be spontaneous drainage as the patient rejects the foreign body. One can only suggest to the patient that they treat the areas topically with antibiotic ointment, and the surgeon should remove any sutures that extrude toward the surface. Sebaceous hypertrophy also can be seen in the revision of facial scars, especially in patients who have seborrhea. This occurs with stimulation of the sebaceous apparatus in the areas of the incision margin. Thus, rather than attempting to excise these wounds (where the scar appears to be depressed), it would be better to either shave the edges with a scalpel, or to utilize dermabrasion. If excision and reapproximation are necessary, the wound should be beveled slightly in an attempt to alleviate the situation. If skin sutures are left longer than 8 days, one may find epithelial tunnels, which will then show suture marks. Thus, early removal of the skin sutures is necessary. Also, one may find milia (firm, pearly white cysts), which result from the trapping of hair follicles in the scar tissue beneath the epithelium. These are best treated by incising the area overlying the cyst and allowing the cyst and its lining to extrude. Finally, scar hypertrophy and keloid formation remain a distinct possibility and must be treated as mentioned in the earlier portions of this chapter.

In conclusion, the author would like to encourage the reader to continue to review the literature for new and better techniques in the evaluation of, and nonsurgical and surgical management of, patients with facial lacerations and wounds. The subject of wound healing is a rapidly changing area of otolaryngology, and it will require constant vigilance on the surgeon's part to keep abreast of new findings.