Surgical Revision of Scars

Martha H. Viera, MD; Sadegh Amini, MD; Ran Huo, BS; Whitney Valins, BS; Brian Berman, MD, PhD

Scars are the consequence of alterations in the skin's healing process after trauma, surgical interventions, or skin diseases prone to leaving scars. Associated symptoms, decreased functionality, and an aesthetic appearance tend to have a severe impact on the psyche and quality of life of individuals bearing scars. Several surgical techniques have been developed to reposition scars toward relaxed skin tension lines, hide scars in nearby anatomical landmarks, or flatten or fill raised or depressed scars, respectively. Among these techniques, we reviewed scar revision methods ranging from simple excisions to W- and Z-plastys, advancement flaps, and geometrical closures, among others. We discuss the effectiveness and current application of dermabrasion, microdermabrasion, lasers, fractional photothermolysis, skin grafts, and soft tissue augmentation modalities. The role of adjuvant treatments including intralesional corticosteroids, 5-fluorouracil, interferons, radiation therapy, topical imiquimod, silicone, and compression is examined. Newer and potential therapies are also mentioned. In conclusion, the best long-lasting, recurrence-free cosmetic result is achieved by the combination of appropriate techniques applied to each scar in a personalized fashion.

Following a skin injury as a consequence of trauma or surgical intervention and disease processes including acne, alterations in the healing process may result in the development of visible scars. In some cases, scars are associated with decrease in functionality and symptoms such as pain, tenderness, and itching,[1] impacting the psychology, aesthetic appearance, and social interaction of individuals bearing dramatic scars as evidenced by a reduction in Quality of Life and Dermatology Life Quality Index scores measured in clinical trials.[2,3]

There is a wide variety of scars, ranging from well-healed, mature scars to raised scars. Widespread (stretched) scars develop when the fine lines from the original wound become stretched and widened, whereas atrophic scars are flat or depressed below the level of the surrounding skin and are generally small and often round with an inverted center (eg, acne scars). Hypertrophic scars are raised and stay within the boundaries of the original wound, usually spontaneously regressing several months after the initial injury.[4] Keloids are raised scars that spread beyond the boundaries of the original wound, frequently continuing to grow over time, invading into the surrounding skin, and usually recurring after excision.[5]

Clinical scars are visibly distinguishable from normal skin because they are raised (keloids and hypertrophic scars), depressed (atrophic scars), or flat (striae and mature scars) as compared with the surrounding, adjacent normal skin. Early lesions are often erythematous and may become brownish red and then pale as they mature, depending on the degree of neovascularization,

Dr. Viera is Senior Clinical Research Fellow; Dr. Amini is Volunteer Clinical Research Fellow; Mr. Huo and Ms. Valins are Clinical Research Fellows; and Dr. Berman is Professor of Dermatology and Internal Medicine, all at Department of Dermatology and Cutaneous Surgery, University of Miami, Miller School of Medicine, Florida.

Dr. Berman is an advisory board member and speaker for Graceway Pharmaceuticals, LLC, and sanofi-aventis US, LLC, and a researcher for Graceway Pharmaceuticals, LLC.

Correspondence: Sadegh Amini, MD, University of Miami, Miller School of Medicine, Department of Dermatology and Cutaneous Surgery, 1600 NW 10th Ave, RMSB, Room 2023A (R250), Miami, FL, 33136 (dermatology.research@med.miami.edu).

immune effects, bilirubin breakdown products following intracutaneous bleeding, and hemosiderin deposition. Scar consistency varies from soft and doughy to rubbery and hard. Lastly, most scars have regular and well-defined borders; however, some are irregular and possess a claw-like configuration as observed in some keloids.

Histopathologic evaluation reveals that scars have thinner and more condensed collagen fibers, mostly parallel to the epidermis; flattening of the rete ridges and dermal-epidermal junction; and reduced or lack of elastin fibers, blood supply, nerve supply, and skin appendages as compared with normal skin.[4-7] Additionally, collagen fibers in keloids and hypertrophic scars tend to be more eosinophilic than in normal scars with multiple areas configured in a whorl-like or nodular pattern alternating with the regular scar tissue.

Scar formation, type, and appearance vary upon the age of the individual (appearing worse in younger people),[8] skin type (keloids are more common in African Americans, Hispanics, and Asians, whereas hypertrophic scars are more common in white individuals),[9] and anatomical area (keloids are more common in high-tension areas such as the anterior chest, shoulders, and scapula).

Surgical techniques applied to camouflage or hide scars must be chosen according to multiple variables including type of scar, anatomic location, type of skin, age, ethnicity, characteristics of surrounding skin, proximity to key anatomical structures, direction, width, contracture, effacement, availability of resources, and skill of the physician performing the procedure. The goal of scar revision is to create a less perceptible or less deforming scar. In this article, we compile the different surgical techniques developed to correct and improve visible scars. Due to high recurrence rates of surgery alone, adjuvant treatment modalities are almost always necessary in order to achieve a long-lasting, recurrence-free, cosmetically acceptable result.

SURGICAL TECHNIQUES

Scar Revision

The successful revision and camouflage of scars can be considered both an art and a science. The goal of scar revision is to improve the scar using one method or a combination of methods to try to make it as subtle as possible.[10]

Patients should be counseled and educated in relation with the real possibilities that any given scar has for a substantial improvement. Generally, a waiting period from 6 to 12 months should be negotiated with the patient before using any technique prematurely in order to let the scar complete its maturation.[11-13]

Any scar wider than 2 mm, longer than 20 mm, and positioned perpendicular to the relaxed skin tension lines (RSTLs) is a candidate for scar revision surgery sooner rather than later. Benefits from scar revision under specific circumstances can be obtained when performing the procedure as early as 6 weeks after the original wound.[14]

Multiple techniques have been used including scar repositioning; Z-plasty; W-plasty; geometric broken line closure; mechanical abrasive techniques (eg, scalpel abrasion, dermabrasion, microdermabrasion, dermasanding); extramarginal scar excision; intramarginal scar excision; and flap repair including V-Y advancement flap, dermal fillers, and laser resurfacing.

Before any surgical option is considered, the surgeon must know that a clean wound with fresh, healthy edges will have a better chance at obtaining a favorable cosmetic result. In addition, there should be minimal or no tension when the borders are approximated, the correct orientation must be selected, and the proper materials, including low-reactive sutures, must be chosen.[10]

Scar Repositioning

This is a good method for small scars located near an RSTL, or any other anatomical site where they can be surgically repositioned. There is always some extra tissue that will consequently be lost between the scar and the new location. Postsurgical deformities should be avoided when planning the surgery.[11,15]

Extramarginal Scar Excision

Extramarginal scar excision is surgical removal of a scar using an elliptical or fusiform excision, independently of the width and depth. Small (<2 cm), straight scars that are wide, depressed, or raised may benefit from this technique.[10-12] When this method is considered for keloids, only pedunculated keloids with narrow bases of 1 cm or less benefit from it.[16] In addition, this method leads to fast synthesis of new collagen, high recurrence rates between 45% and 100%, and larger keloids.[17-21]

Intramarginal Scar Excision

With the intramarginal scar excision technique, the scar is also excised within its borders, leaving a 2- to 3-mm margin of surrounding scar tissue. It is a good method for excising keloids and hypertrophic scars, particularly those generated by burns. The objective is to decrease the volume and flatten the scar.[10,22,23]

Punch Excision

The punch excision is a technique reserved for very deep scars with atrophic bases or sharply punched out ice-pick scars, particularly less than 3.5 mm. The scar is removed and the defect heals by second intention, sutures, or grafts. Punch elevation is an alternative for depressed

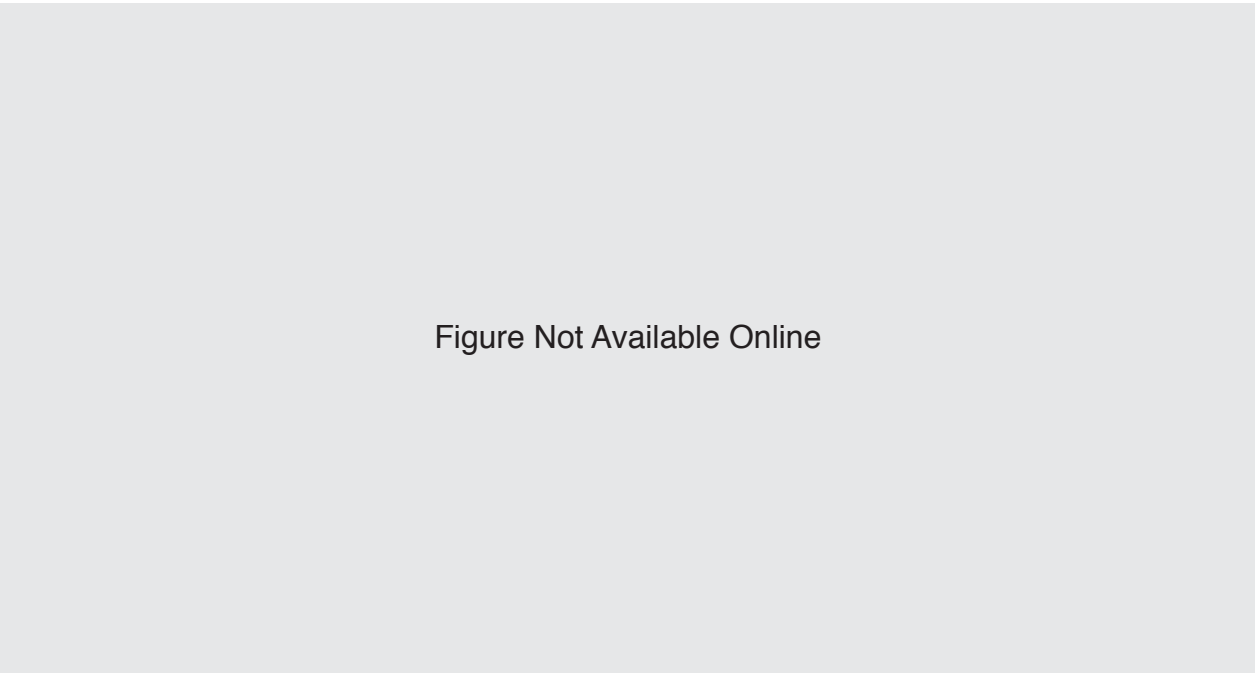

Figure 1. Z-plasty design (60°) with the lateral limbs equal in length to the central limb (A), with scar length represented by line C to D. Elliptical excision of central limb (B), with incision of lateral limbs and undermining of flaps A and B (C). Direction of transposition of A and B flaps (D), and insetting of transposed flaps should approximate without tension (E). Flaps sutured in place (F). Note corner half-hitch horizontal sutures to avoid injury to the most random portion of the flaps. Dotted line C to D illustrates theoretical gain in scar length. Reprinted from *Dermatologic Clinics,* Vol 16 No 1, McGillis ST, Lucas AR, Scar Revision, Pages 165-180, Copyright (1998), with permission from Elsevier.[10]

boxcar scars where the surgical instrument matches the inner diameter of the scar. The base is not removed but elevated to the level of the surrounding skin and then sutured.[24,25] These techniques are usually combined with other resurfacing modalities for better cosmetic results.

W-plasty Scar Revision

W-plasty is used to disrupt long scars (>2 cm) usually located perpendicular to the RSTLs, reorienting them within those lines. The technique also helps to flatten the scar and reduce contraction. The scar tissue is completely excised using a series of 60°-tipped triangles forming Ws in an interlocking fashion. It is a good method for the forehead, cheeks, chin, and nose. However, there is no gain on the length of the scar.[10-12,25]

Z-plasty Scar Revision

Z-plasty is very useful for making scars less visible and changing their direction toward a location parallel to the RSTLs, particularly for scar elongation and release of scar contracture. This is very important mainly with visible deformation of free margins, including the eyelids, nasal alar rim, and lips. In addition, this procedure helps reposition displaced anatomical landmarks such as an elevated or depressed oral commissure or eyebrow. After excising the central portion of the scar, 2 incisions equal in length to the central portion are made along the scar generating two 60° triangles, which are lifted simultaneously and transposed, producing a 90° change in the scar direction and a 75% gain in tissue length (Figure 1).[10-12,15]

V-Y and Y-V Advancement Flaps

These advancement flap techniques are used for lengthening and relaxing small (1–2 cm) scars, and also for elevating or depressing free margins such as the eyelids and the mouth. A V-shaped incision along the length of the scar is made. The V-shaped flap is advanced, leaving a new defect which is closed side to side, forming a straight line. The 2 arms of the V are then sutured side to side, giving the general appearance of a Y shape. The V-Y flap raises anatomic points, whereas the Y-V flap can lower them. An incision in the shape of a Y is made and the flap is pushed down and contracted into a V (Figure 2).[10-12,15]

Geometric Broken Line Closure

A geometric broken line closure is a refined, complicated version of the W-plasty. It is used to provide irregularity in a random fashion to long, unbroken scars that cross RSTLs. On one side, multiple geometrical shapes are outlined including triangles, semicircles, squares, and rectangles no longer that 6 mm in any dimension to obtain the best camouflage. On the opposite side of the scar,

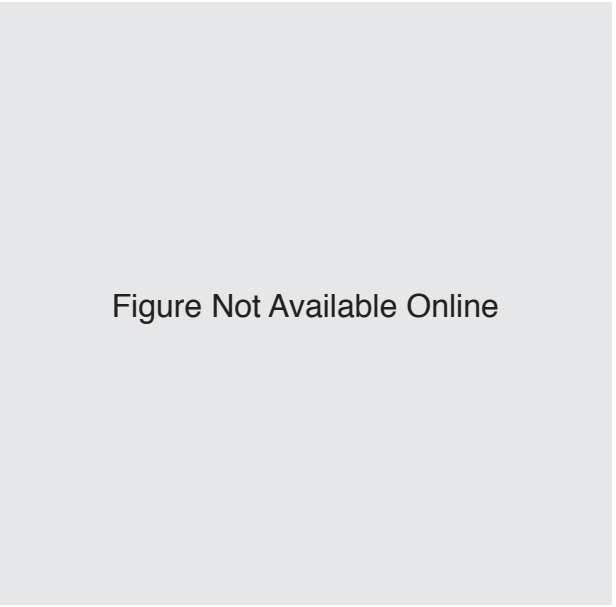

Figure 2. For a V-Y advancement flap, a V-shaped incision is made and, after wide undermining, is pulled in the direction of the open end of the V, releasing tension/contracture in this area. The remaining defect is closed side to side to form a Y (A). For a Y-V advancement flap, a Y-shaped incision is made and, after wide undermining, is pushed and contracted into a V (B). Reprinted from *Dermatologic Clinics,* Vol 23 No 1, Lee KK, Mehrany K, Swanson NA, Surgical Revision, Pages 141-150, Copyright (2005), with permission from Elsevier. [12]

mirror images are outlined. The scar is excised following the outline. Suturing each corresponding shape is done side to side. Disadvantages include time consumption and a worsened appearance when improperly designed (Figure 3).[11,12,15]

Scalpel Abrasion

Scalpel abrasions are superficial cross-hatchings of scars, particularly on the face, where microscopic Z-plasties are generated to reduce wound contraction.[11,15]

Shave Excision

Besides its role in the prevention of scar development, shave excisions achieve rapid reepithelialization and effacement in the epithelial portion of the scar, preserving the healed dermis. This method is very useful for epithelial elevations and step deformities due to lacerations or incisions made in locations such as the nose. A classic example constitutes shave excision of a rhinophyma.[11,15]

Dermabrasion

Dermabrasion is a technique used to improve textural abnormalities associated with scars. A superficial injury to the skin is created, usually to the level of the papillary dermis, resulting in a smoother, blended, effaced, and camouflaged scar. Dermabrasion is also used in combination with other scar revision techniques in a sequential fashion in order to obtain optimal cosmetic results. In this case, the dermabrasion should be performed early (6–12 weeks postoperatively). Several sessions are usually necessary, including multistage and minor touch-up procedures. Although dermabrasion can be used for any scar, the main indications include deep acne scars, traumatic and surgical scars, and rhinophyma. Keloids and hypertrophic scars should be treated first with other techniques.

Figure 3. For a geometric broken line closure, a random geometric pattern is applied to a linear scar, making it appear less predictable than a zigzag line. Reprinted from *Dermatologic Clinics, V*ol 23 No 1, Lee KK, Mehrany K, Swanson NA, Surgical Revision, Pages 141-150, Copyright (2005), with permission from Elsevier. [12]

With dermabrasion, the scar is not removed but the superficial abrasion takes place particularly in the edges and the surrounding skin, which are both brought down to the level of the depressed region, allowing the lesion to blend into the surrounding skin.

For larger areas, a powered dermabrader with a diamond fraise tip is used for a more controlled depth of ablation, whereas sterile sandpaper is recommended for smaller scars.

Postoperative time to reepithelialization is usually 7 to 10 days with the appropriate wound care. Patients should expect erythema, edema, and crusting during the postoperative period.

A thorough preoperative evaluation should include history of bleeding disorders, herpes infections, impetigo, keloid or hypertrophic scarring, koebnerizing conditions, previous isotretinoin therapy, and immunosupression. Laboratory studies such as hepatitis panel and screening for human immunodeficiency virus antibodies should be obtained.

Dermabrasion is ideal for Fitzpatrick skin types I and II because of the risk for pigmentation caused by the procedure in darker skin types. A skilled operator is required to obtain the desired cosmetic results.[10-13,15,26,27]

Dermasanding

Dermasanding is manual dermabrasion using a sterile 300- to 400-grit sandpaper, achieving a similar effect than dermabrasion with the advantages of being a simpler, cost effective, and readily accessible technique. In addition, it is also safer due to the absence of blood splatter or aerosolized infectious particles. Randomized, controlled studies have demonstrated the efficacy of the technique in the treatment of surgical scars.[28,29]

Microdermabrasion

Since its development in Italy in 1985 and approval by the US Food and Drug Administration in 1994, microdermabrasion has become a popular method among dermatologists, plastic surgeons, and aestheticians for the improvement of several conditions including acne scars and scars due to other causes. With this technique, the ablation obtained is superficial, reaching only to the level of the epidermis. Deeper scars are usually unaffected by the procedure. Advantages are that the technique does not require anesthesia; it is painless; multistage and minor touch-up procedures can be repeated at short intervals (every 10–15 days); it is simple to operate and quick to perform; it does not alter the patient's life (no downtime); it has a high degree of satisfaction; improves skin tone, texture, and pigmentation; and the concomitant use of other exfoliation therapies (eg, retinoids) can be continued without significant irritation.

Microdermabrasion units are closed-loop negative pressure systems that blow aluminum oxide microcrystals into the treated area. Other systems also use sodium chloride, magnesium oxide, and sodium bicarbonate microcrystals. The depth in the skin is determined by the strength of the flow, the speed at which the handpiece is moved, and the quantity of passes in a particular area.[30,31]

Laser Scar Revision

Keloids and hypertrophic scars have been treated with Nd:YAG and CO_2 lasers, which vaporize scars with a high incidence of recurrence and adverse events such as skin atrophy, pigmentary alterations, and pain. Newer units including the flashlamp-pumped pulsed dye laser (PDL), and particularly the 585-nm PDL, which targets blood vessels within the scar tissue, limiting further collagen production, have obtained favorable results in scar texture and pliability.

The clinical effect of lasers is obtained due to the capability of different components of the skin (melanin, hemoglobin, and water) to absorb the light and raise their temperature as a consequence, resulting in tissue damage. When a laser emits light in a pulsed fashion, the amount of thermal diffusion is limited, allowing the tissue to relax from the thermal injury, thus limiting tissue damage.

Lasers such as the Nd:YAG, erbium:YAG, and CO_2 cause deepithelialization using water as the target chromophore. They are indicated in the treatment of atrophic scars, causing similar effects to dermabrasion. Using hemoglobin as the target chromophore, PDLs penetrate the epidermis without casing deepithelialization and are more suitable for keloids and hypertrophic scars.

Equipment is expensive and a skilled operator is required. Individuals with Fitzpatrick skin types III or higher are at greater risk for developing chronic postinflammatory hyperpigmentation, which can last several months after the procedure. Other complications include erythema and edema, which persist for an average of 2 to 4 weeks with the erbium laser as compared with 3 to 6 months with the CO_2 laser. Crusting; spot bleeding; contact allergies (mostly caused by concomitant topical medications including antibiotics, sunscreens, topical steroids, and their vehicles); reactivation of herpes virus infection; bacterial and yeast infections; hypertrophic scars and keloid formation (particularly in patients taking isotretinoin within the previous 6 to 12 months); and ectropion formation are other complications that can occur.[10,12,13,27,32-34]

Cryosurgery

The application of low temperatures to keloids and hypertrophic scars with liquid nitrogen and nitrous oxide causes damage to the cells and microvasculature, leading to necrosis within the scar tissue.[35,36] Several reports have obtained partial flattening in up to 85% of cases,[37-40] and recurrence rates with single sessions up to 33%.[41] As monotherapy, the recommendation is to treat in multiple sessions, lowering recurrence rates to 2%.[35,36,41] Early scars tend to respond better than mature ones.[40] Common adverse events related to cryotherapy include pain, necrosis, edema, atrophy, infections, hyperpigmentation, and hypopigmentation, with the latter being typically significant in darker skinned patients.[35,36,40,42]

Fractional Photothermolysis

Introduced in 2004, fractional photothermolysis has been an important technology that has demonstrated effectiveness in the treatment of several skin conditions including acne and hypopigmented scars. Skin ablation is produced in a columnar fashion, sparing the surrounding epidermis and underlying dermis from treatment, leaving it intact. There is stimulation of collagen remodeling and neocollagenesis, with the improvement of texture of the treated area. Adverse events are similar to the ones observed with all ablative techniques but tend to be less frequent. They include transient

erythema, edema, dryness, scabbing, milia or acne, pigmentary alterations, prolonged healing, or infections.[24,43]

Skin Grafts

Split-thickness or full-thickness skin grafts can be applied to the scar's bed to repair or camouflage the scar. Skin grafts have been used for acne scars, keloid and hypertrophic scars, and burn scars and have a recurrence rate of 59%. Studies have shown that half of patients with skin grafts develop keloids at the donor site.[44,45]

Early excision and grafting of pediatric facial burns demonstrated to be safe and free of complications such as acute airway decompensation or regrafting.[46] Early excision and delayed skin grafting of facial burns also decrease facial scar hypertrophy and deformity.[47]

New grafting techniques, such as cultured autodermal grafts, offer improved initial management of burns that may subsequently optimize scar revision in these patients.

Table 1

Surgical Modalities for the Revision of Scars by Type of Scar

Type of Scar	Treatment
Burn scars	Skin grafts
Deep acne scars (including ice-pick scars and pitted scars)	Punch excision Dermabrasion Fractional photothermolysis Skin grafts
Depressed boxcar scars	Punch excision Punch elevation Lasers
Depressed scars	Extramarginal excision Soft tissue augmentation Lasers
Hypopigmented scars	Fractional photothermolysis
Keloids and hypertrophic scars	Extramarginal excision Intramarginal excision Shave excision Lasers Skin grafts
Long scars perpendicular to RSTLs	W-plasty Geometric broken line closure Dermabrasion
Rhinophyma and step deformities	Shave excision Dermabrasion
Slightly elevated scars, flat scars	Microdermabrasion Dermasanding Fractional photothermolysis Lasers
Small scars near RSTLs	Scar repositioning V-Y plasty and Y-V plasty
Small, straight, wide scars	Extramarginal excision Lasers
Superficial acne scars	Microdermabrasion Fractional photothermolysis

Abbreviation: RSTL, relaxed skin tension line.

Soft Tissue Augmentation

With soft tissue augmentation, a substance is injected into the dermis underlying depressed or concave scars with the objective of elevating that area to the level of the surrounding, normal skin. Soft tissue augmentation provides long-lasting, appearance-enhancing results and can be combined with other modalities for more effective results. The substances used have all been successful including collagen, gelatin matrix implants, fat injections, silicone, calcium hydroxylapatite, and hyaluronic acid, among others. The advantages of these substances include being injected in an office setting, easy procedure for a skilled operator, cost effectiveness, and minimal discomfort.[10,48-50]

Specific indications for surgical treatment modalities according to the type of scar are summarized in Table 1. A list of surgical treatment modalities for special conditions where scars cause deformities of free margins and displacement of anatomical landmarks are summarized in Table 2.

ADJUVANT TREATMENT MODALITIES

Intralesional Therapies

Corticosteroids—Injecting corticosteroids is a technique reserved for keloids and hypertrophic scars. The most widely used steroid for intralesional injection is triamcinolone acetonide, which can be injected directly into the scar tissue to reduce size, volume, and symptoms, or applied in combination with other techniques such as surgical modalities, compression, or both to prevent their recurrence. During the surgical removal of the scar, steroids can be injected directly into the wound edges before suture closing and repeated as needed postoperatively.

Steroids have a more favorable cosmetic impact when treating recent (less mature) scars than older, more mature keloids and hypertrophic scars. When treating the scar itself, the injection should be placed into the bulkiest area of the scar in the dermis or subcutaneous tissue.

TABLE 2

Special Conditions Where Scars Cause Deformities of Free Margins and Displacement of Anatomical Landmarks

Condition	Treatment
Deformation of free margins (eg, eyelids, nasal rims, lips)	Z-plasty
Reposition of displaced anatomical landmarks	Z-plasty
To elevate or depress free margins	V-Y plasty and Y-V plasty

Favorable results have also been obtained with topical applications of corticosteroid creams, ointments, or tapes. Long-term complications, particularly at higher concentrations, frequency, or quantity include subcutaneous tissue atrophy, pigmentary alterations, and thinning of the skin.[10-12,15]

5-Fluorouracil—5-Fluorouracil (5-FU) is a pyrimidine analog that inhibits fribroblast proliferation through its antimetabolite activity. Although in vitro it has demonstrated to be effective as a monotherapy for keloids and hypertrophic scars,[51] clinical studies have reported high recurrence rates.[52] Favorable outcomes with 5-FU have been obtained after the combination with corticosteroids and laser treatments.[51,53-55] Long-term follow-up evaluation in a prospective, placebo-controlled study resulted in the improvement of clinical and immunohistochemical parameters after a single postsurgical application of 5-FU.[54] The most common adverse event reported was mild pain at the injection site.

Interferons—Interferons (IFNs) are cytokines secreted mainly by T helper lymphocytes and are known to down regulate the excessive production of glycosaminoglycans and collagens I, II, and III, to interfere with collagen cross-linking, and to enhance the activity and levels of collagenase.[42,56-58] In addition, IFN-γ induces apoptosis in myofibroblasts and inhibits transforming growth factor β gene regulation.[59-61] Berman and Flores[62] reported an 18% recurrence rate with the application of IFN-α2b postsurgery at the excised keloid site as compared with a 51% recurrence rate with surgery alone and a 58% recurrence rate with postsurgical application of triamcinolone acetonide. Conejo-Mir et al[63] obtained a 66% cure rate and a 33% recurrence rate 3 years following the application of IFN-α2b postexcision with a CO_2 laser. The most common adverse events reported with IFN use are pain at the injection site and flulike symptoms for up to 72 hours postinjection.[64-66]

EXTERNAL RADIATION

Radiation Therapy

Radiation therapy increases the rate of apoptosis in normal and keloidal fibroblasts, causes alterations in the gene expression of the extracellular matrix, and damages the connective tissue stem cells.[67-70] As monotherapy, cure rates range from 10% to 94%, with recurrence rates of 50% to 100%.[17,19] Postsurgical radiation therapy either immediately after or within 2 weeks after keloid excision, increases response rates from 65% to 99% as compared with radiation therapy alone.[70-74] Recurrences tend to occur within 1 to 3 years.[65] More favorable results have been reported with fractionated doses for 7 to 10 days.[75] Radiation therapy also reduces common symptoms associated with keloids such as pruritus, pain, and tenderness.[35] Common adverse events related to radiation therapy include hyperpigmentation, ulceration, pruritus, and erythema.[20,36,65,71,72]

TOPICAL TREATMENTS

Imiquimod 5%

Imiquimod 5% is a topical immunomodulator that binds to intracellular toll-like receptors 7 and 8, inducing local production of cytokines such as IFN-α, IFN-γ, tumor necrosis factor α, interleukin (IL)-1α, IL-6, IL-8, IL-12, resulting in a Th_1-dominant response.[76-82] Imiquimod 5% has also been shown to stimulate the production of IL-6, IL-8, and IFN-α in keratinocytes.[76-82] In a dose-dependent fashion, IFN-α and IFN-γ inhibit human fibroblast collagen production.[83] Berman and Kaufman[84] reported a recurrence rate of 0% after a postsurgical follow-up period of 24 weeks. Two patients did not complete the study and were considered treatment failures, which could have increased the recurrence rate to 15.4%, still much lower than prior reports of surgical treatment alone.[17,67,71] Adverse events reported were mild hyperpigmentation in 63.6% of the patients and mild to moderate irritation. Additional clinical trials have reported high efficacy and low recurrence rates with the postsurgical application of imiquimod 5% cream with follow-up periods up to 96 weeks.[85-87]

Silicone

Silicone is a cross-linked polymer of dimethylsiloxane that can be used as a topical gel or as an impregnated elastic sheet. It is usually applied under daily occlusion for intermittent periods of at least 12 hours for 4 to 6 months.[64] Its mechanism of action is still poorly

understood. It causes a reduction in scar volume and an increase in scar elasticity in 60% to 100% of the cases.[88] Reports have indicated that postsurgical application of silicone may prevent up to 75% to 85% of hypertrophic scar and keloids.[89-91] Several randomized clinical trials have obtained statistical differences in scar parameters, controlling symptoms with the use of silicone after surgical excision of hypertrophic scars as compared with controls,[92] preventing the development of abnormal scars in high-risk patients.[93]

Compression

Compression is thought to cause ischemia, increase collagenase activity, and decrease tissue metabolism.[35,88] The induction of local hypoxia leads to fibroblast and collagen degeneration, decrease in cohesion between collagen fibers, and lower amounts of chondroitin-4-sulphate.[94,95] In addition, compression decreases hydration of the scar, reducing neovascularization and extracellular matrix production.[35,67] Compression has been used for the reduction of size and induration of keloids and hypertrophic scars,[67,88,96,97] with some reports ranging its efficacy as monotherapy between 60% to 85%.[17,98,99] Pressure should be maintained daily for 18 to 24 hours for a minimum of 4 to 6 months and up to 2 years, with recurrence rates of 0% after treating hypertrophic scars.[88,100] Postsurgical application of compression has obtained efficacy rates of 90% to 100%, particularly after the excision of earlobe keloids.[17,18,101,102]

A list of adjuvant therapeutic modalities can be found in Table 3.

TABLE 3
Adjuvant Treatment Modalities

Route of Application	Agents
Intralesional	Corticosteroids 5-FU Interferons
External radiation	Radiation therapy
Topical	Imiquimod Silicone Compression

Abbreviation: 5-FU, 5-fluorouracil.

OTHER TREATMENTS

Additional treatment modalities have been reported in the literature, obtaining mixed results including retinoic acid, tacrolimus, cyclosporine A, quercetin (onion extract), pentoxifylline, methotrexate, tamoxifen, doxorubicin, verapamil, and prostaglandin E_2.

Newer treatments, agents in development, and promising compounds include botulinum toxin; transforming growth factor β3; IL-10; vascular endothelial growth factor inhibitors; etanercept; manose-6-phosphate inhibitors; the combination of hydrocortisone, silicon, and vitamin E; gentian violet; photodynamic therapy; intense pulsed light; UVA1; and narrowband and broadband UVB.[103] Further studies are required to determine their role in the treatment of scars.

CONCLUSION

Surgical modalities for scar revision have been developed to improve and ultimately correct the cosmetic appearance of visible scars. These techniques reposition, hide, and camouflage scars, placing them parallel to RSTLs, or within anatomical landmarks very effectively. However, additional techniques are required when scars are difficult to hide or tend to recur due to their location in high-risk anatomical areas or for constitutional (genetic) factors. The goal in these cases is to improve the scar's features, such as elevation, color, texture, and length, in order to make them appear as similar as possible to the surrounding normal skin. Scars that are difficult to treat and scars with high recurrence rates when excised, such as keloids, usually require adjuvant therapies including the application of topical or intralesional agents intrasurgery, postsurgery, or both. Lastly, improving scar cosmesis correlates with the improvement in the quality of life of individuals bearing scars, which may had been impacted physically, psychologically, and socially by a disfiguring, visible scar.

REFERENCES

1. Mustoe TA, Cooter RD, Gold MH, et al. International clinical recommendations on scar management. *Plast Reconstr Surg*. 2002;110:560-571.
2. Holme SA, Beattie PE, Fleming CJ. Cosmetic camouflage advice improves quality of life. *Br J Dermatol*. 2002;147:946-949.
3. Graham JA, Jouhar AJ. The importance of cosmetics in the psychology of appearance. *Int J Dermatol*. 1983;22:153-156.
4. Fette A. Influence of silicone on abnormal scarring. *Plast Surg Nurs*. 2006;26:87-92.
5. Blackburn WR, Cosman B. Histologic basis of keloid and hypertrophic scar differentiation. clinicopathologic correlation. *Arch Pathol*. 1966;82:65-71.
6. Anderson RR, Parrish JA. The optics of human skin. *J Invest Dermatol*. 1981;77:13-19.
7. Mannor GE, Phelps RG, Friedman AH, et al. Eyelid healing after carbon dioxide laser skin resurfacing: histological analysis. *Arch Ophthalmol*. 1999;117:913-916.
8. American Academy of Dermatology. What is a scar. http://www.aad.org/public/Publications/pamphlets/WhatisaScar.htm. Accessed March 31, 2009.
9. Robles DT, Moore E, Draznin M, et al. Keloids: pathophysiology and management. *Dermatol Online J*. 2007;13:9.

10. McGillis ST, Lucas AR. Scar revision. *Dermatol Clin*. 1998;16:165-180.
11. Thomas JR, Ehlert TK. Scar revision and camouflage. *Otolaryngol Clin North Am*. 1990;23:963-973.
12. Lee KK, Mehrany K, Swanson NA. Surgical revision. *Dermatol Clin*. 2005;23:141-150, vii.
13. Bradley DT, Park SS. Scar revision via resurfacing. *Facial Plast Surg*. 2001;17:253-262.
14. Decherd ME, Calhoun KH. Scar Revision and Camouflage. Paper presented at Grand Rounds Presentation, UTMB, Department of Otolaryngology; April 24, 2002. Available at: http://www.utmb.edu/otoref/Grnds/Scar-Revision-2002-04/Scar-Revision-2002-04.pdf. Accessed March 31, 2009.
15. Tardy ME Jr, Thomas JR, Pashcow MS. The camouflage of cutaneous scars. *Ear Nose Throat J*. 1981;60:61-70.
16. Berman B, Viera MH, Amini S, et al. Prevention and management of hypertrophic scars and keloids after burns in children. *J Craniofac Surg*. 2008;19:989-1006.
17. Berman B, Bieley HC. Keloids. *J Am Acad Dermatol*. 1995;33:117-123.
18. Berman B, Bieley HC. Adjunct therapies to surgical management of keloids. *Dermatol Surg*. 1996;22:126-130.
19. Lawrence WT. In search of the optimal treatment of keloids: report of a series and a review of the literature. *Ann Plast Surg*. 1991;27:164-178.
20. Cosman B, Wolff M. Correlation of keloid recurrence with completeness of local excision. a negative report. *Plast Reconstr Surg*. 1972;50:163-166.
21. Salasche SJ, Grabski WJ. Keloids of the earlobes: a surgical technique. *J Dermatol Surg Oncol*. 1983;9:552-556.
22. Engrav LH, Gottlieb JR, Millard SP, et al. A comparison of intramarginal and extramarginal excision of hypertrophic burn scars. *Plast Reconstr Surg*. 1988;81:40-45.
23. Yang JY. Intrascar excision for persistent perioral hypertrophic scar. *Plast Reconstr Surg*. 1996;98:1200-1205.
24. Rivera AE. Acne scarring: a review and current treatment modalities. *J Am Acad Dermatol*. 2008;59:659-676.
25. Frith M, Harmon CB. Acne scarring: current treatment options. *Dermatol Nurs*. 2006;18:139-142.
26. Harmon CB. Dermabrasion. *Dermatol Clin*. 2001;19:439-442, viii.
27. Roy D. Ablative facial resurfacing. *Dermatol Clin*. 2005;23:549-559, viii.
28. Poulos E, Taylor C, Solish N. Effectiveness of dermasanding (manual dermabrasion) on the appearance of surgical scars: a prospective, randomized, blinded study. *J Am Acad Dermatol*. 2003;48:897-900.
29. Emsen IM. Effect of dermasanding (manual dermabrasion) with sandpaper on the appearance of both postsurgical and burn scars. *Aesthetic Plast Surg*. 2007;31:608-611.
30. Grimes PE. Microdermabrasion. *Dermatol Surg*. 2005;31(9 pt 2):1160-1165.
31. Savardekar P. Microdermabrasion. *Indian J Dermatol Venereol Leprol*. 2007;73:277-279.
32. Lupton JR, Alster TS. Laser scar revision. *Dermatol Clin*. 2002;20:55-65.
33. Fitzpatrick RE. CO_2 laser resurfacing. *Dermatol Clin*. 2001;19:443-451, viii.
34. Alster TS, Lupton JR. Erbium:YAG cutaneous laser resurfacing. *Dermatol Clin*. 2001;19:453-466.
35. Kelly AP. Medical and surgical therapies for keloids. *Dermatol Ther*. 2004;17:212-218.
36. English RS, Shenefelt PD. Keloids and hypertrophic scars. *Dermatol Surg*. 1999;25:631-638.
37. Layton AM, Yip J, Cunliffe WJ. A comparison of intralesional triamcinolone and cryosurgery in the treatment of acne keloids. *Br J Dermatol*. 1994;130:498-501.
38. Shepherd JP, Dawber RP. The response of keloid scars to cryosurgery. *Plast Reconstr Surg*. 1982;70:677-682.
39. Zouboulis CC, Blume U, Büttner P, et al. Outcomes of cryosurgery in keloids and hypertrophic scars. a prospective consecutive trial of case series. *Arch Dermatol*. 1993;129:1146-1151.
40. Rusciani L, Rossi G, Bono R. Use of cryotherapy in the treatment of keloids. *J Dermatol Surg Oncol*. 1993;19:529-534.
41. Har-Shai Y, Amar M, Sabo E. Intralesional cryotherapy for enhancing the involution of hypertrophic scars and keloids. *Plast Reconstr Surg*. 2003;111:1841-1852.
42. Meier K, Nanney LB. Emerging new drugs for scar reduction. *Expert Opin Emerg Drugs*. 2006;11:39-47.
43. Cho SB, Lee JH, Choi MJ, et al. Efficacy of the fractional photothermolysis system with dynamic operating mode on acne scars and enlarged facial pores. *Dermatol Surg*. 2009;35:108-114.
44. Tredget EE, Nedelec B, Scott PG, et al. Hypertrophic scars, keloids, and contractures. The cellular and molecular basis for therapy. *Surg Clin North Am*. 1997;77:701-730.
45. Apfelberg DB, Maser MR, Lash H. The use of epidermis over a keloid as an autograft after resection of the keloid. *J Dermatol Surg*. 1976;2:409-411.
46. Housinger TA, Hills J, Warden GD. Management of pediatric facial burns. *J Burn Care Rehabil*. 1994;15:408-411.
47. Zhang G, Sun Y, Yan R. Management of deep facial burn with early postburn debridement and delayed skin grafting. *Zhonghua Shao Shang Za Zhi*. 2001;17:327-329.
48. Klein AW. Techniques for soft tissue augmentation: an 'a to z'. *Am J Clin Dermatol*. 2006;7:107-120.
49. Cheng JT, Perkins SW, Hamilton MM. Collagen and injectable fillers. *Otolaryngol Clin North Am*. 2002;35:73-85, vi.
50. Kasper DA, Cohen JL, Saxena A, et al. Fillers for postsurgical depressed scars after skin cancer reconstruction. *J Drugs Dermatol*. 2008;7:486-487.
51. Apikian M, Goodman G. Intralesional 5-fluorouracil in the treatment of keloid scars. *Australas J Dermatol*. 2004;45:140-143.
52. Kontochristopoulos G, Stefanaki C, Panagiotopoulos A, et al. Intralesional 5-fluorouracil in the treatment of keloids: an open clinical and histopathologic study. *J Am Acad Dermatol*. 2005;52(3 pt 1):474-479.
53. Fitzpatrick RE. Treatment of inflamed hypertrophic scars using intralesional 5-FU. *Dermatol Surg*. 1999;25:224-232.
54. Uppal RS, Khan U, Kakar S, et al. The effects of a single dose of 5-fluorouracil on keloid scars: a clinical trial of timed wound irrigation after extralesional excision. *Plast Reconstr Surg*. 2001;108:1218-1224.
55. Manuskiatti W, Fitzpatrick RE. Treatment response of keloidal and hypertrophic sternotomy scars: comparison among intralesional corticosteroid, 5-fluorouracil, and 585-nm flashlamp-pumped pulsed-dye laser treatments. *Arch Dermatol*. 2002;138:1149-1155.
56. Berman B, Villa AM, Ramirez CC. Novel opportunities in the treatment and prevention of scarring. *J Cutan Med Surg*. 2004;8(suppl 3):32-36.
57. Larrabee WF Jr, East CA, Jaffe HS, et al. Intralesional interferon gamma treatment for keloids and hypertrophic scars. *Arch Otolaryngol Head Neck Surg*. 1990;116:1159-1162.
58. Pittet B, Rubbia-Brandt L, Desmoulière A, et al. Effect of gamma-interferon on the clinical and biologic evolution of hypertrophic scars and Dupuytren's disease: an open pilot study. *Plast Reconstr Surg*. 1994;93:1224-1235.
59. Yokozeki M, Baba Y, Shimokawa H, et al. Interferon-gamma inhibits the myofibroblastic phenotype of rat palatal fibroblasts induced by transforming growth factor-beta1 in vitro. *FEBS Lett*. 1999;442:61-64.
60. Rumalla VK, Borah GL. Cytokines, growth factors, and plastic surgery. *Plast Reconstr Surg*. 2001;108:719-733.

61. Tredget EE, Wang R, Shen Q, et al. Transforming growth factor-beta mRNA and protein in hypertrophic scar tissues and fibroblasts: antagonism by IFN-alpha and IFN-gamma in vitro and in vivo. *J Interferon Cytokine Res*. 2000;20:143-151.
62. Berman B, Flores F. Recurrence rates of excised keloids treated with postoperative triamcinolone acetonide injections or interferon alfa-2b injections. *J Am Acad Dermatol*. 1997;37(5 pt 1):755-757.
63. Conejo-Mir JS, Corbi R, Linares M. Carbon dioxide laser ablation associated with interferon alfa-2b injections reduces the recurrence of keloids. *J Am Acad Dermatol*. 1998;39:1039-1040.
64. Al-Attar A, Mess S, Thomassen JM, et al. Keloid pathogenesis and treatment. *Plast Reconstr Surg*. 2006;117:286-300.
65. Brissett AE, Sherris DA. Scar contractures, hypertrophic scars, and keloids. *Facial Plast Surg*. 2001;17:263-272.
66. Poochareon VN, Berman B. New therapies for the management of keloids. *J Craniofac Surg*. 2003;14:654-657.
67. Rockwell WB, Cohen IK, Ehrlich HP. Keloids and hypertrophic scars: a comprehensive review. *Plast Reconstr Surg*. 1989;84:827-837.
68. Luo S, Benathan M, Raffoul W, et al. Abnormal balance between proliferation and apoptotic cell death in fibroblasts derived from keloid lesions. *Plast Reconstr Surg*. 2001;107:87-96.
69. Lee KS, Jung JB, Ro YJ, et al. Effects of x-irradiation on survival and extracellular matrix gene expression of cultured keloid fibroblasts. *J Dermatol Sci*. 1994;8:33-37.
70. Borok TL, Bray M, Sinclair I, et al. Role of ionizing irradiation for 393 keloids. *Int J Radiat Oncol Biol Phys*. 1988;15:865-870.
71. Darzi MA, Chowdri NA, Kaul SK, et al. Evaluation of various methods of treating keloids and hypertrophic scars: a 10-year follow-up study. *Br J Plast Surg*. 1992;45:374-379.
72. Levy DS, Salter MM, Roth RE. Postoperative irradiation in the prevention of keloids. *AJR Am J Roentgenol*. 1976;127:509-510.
73. Klumpar DI, Murray JC, Anscher M. Keloids treated with excision followed by radiation therapy. *J Am Acad Dermatol*. 1994;31 (2 pt 1):225-231.
74. Ragoowansi R, Cornes PG, Moss AL, et al. Treatment of keloids by surgical excision and immediate postoperative single-fraction radiotherapy. *Plast Reconstr Surg*. 2003;111:1853-1859.
75. Brown LA Jr, Pierce HE. Keloids: scar revision. *J Dermatol Surg Oncol*. 1986;12:51-56.
76. Edwards AD, Diebold SS, Slack EM, et al. Toll-like receptor expression in murine DC subsets: lack of TLR7 expression by CD8 alpha+ DC correlates with unresponsiveness to imidazoquinolines. *Eur J Immunol*. 2003;33:827-833.
77. Imbertson LM, Beaurline JM, Couture AM, et al. Cytokine induction in hairless mouse and rat skin after topical application of the immune response modifiers imiquimod and S-28463. *J Invest Dermatol*. 1998;110:734-739.
78. Wagner TL, Ahonen CL, Couture AM, et al. Modulation of TH1 and TH2 cytokine production with the immune response modifiers, R-848 and imiquimod. *Cell Immunol*. 1999;191:10-19.
79. Hemmi H, Kaisho T, Takeuchi O, et al. Small anti-viral compounds activate immune cells via the TLR7 MyD88-dependent signaling pathway. *Nat Immunol*. 2002;3:196-200.
80. Hengge UR, Benninghoff B, Ruzicka T, et al. Topical immunomodulators—progress towards treating inflammation, infection, and cancer. *Lancet Infect Dis*. 2001;1:189-198.
81. Kono T, Kondo S, Pastore S, et al. Effects of a novel topical immunomodulator, imiquimod, on keratinocyte cytokine gene expression. *Lymphokine Cytokine Res*. 1994;13:71-76.
82. Arany I, Tyring SK, Stanley MA, et al. Enhancement of the innate and cellular immune response in patients with genital warts treated with topical imiquimod cream 5%. *Antiviral Res*. 1999;43:55-63.
83. Jimenez SA, Freundlich B, Rosenbloom J. Selective inhibition of human diploid fibroblast collagen synthesis by interferons. *J Clin Invest*. 1984;74:1112-1116.
84. Berman B, Kaufman J. Pilot study of the effect of postoperative imiquimod 5% cream on the recurrence rate of excised keloids. *J Am Acad Dermatol*. 2002;47(suppl 4):S209-S211.
85. Clark J. Imiquimod vs. interferon: cream matches injection in terms of eliminating keloid recurrence while showing no adverse events. *Dermatology Times*. December 1, 2002.
86. Martin-Garcia RF, Busquets AC. Postsurgical use of imiquimod 5% cream in the prevention of earlobe keloid recurrences: results of an open-label, pilot study. *Dermatol Surg*. 2005;31(11 pt 1):1394-1398.
87. Stashower ME. Successful treatment of earlobe keloids with imiquimod after tangential shave excision. *Dermatol Surg*. 2006;32:380-386.
88. Niessen FB, Spauwen PH, Schalkwijk J, et al. On the nature of hypertrophic scars and keloids: a review. *Plast Reconstr Surg*. 1999;104:1435-1458.
89. Ahn ST, Monafo WW, Mustoe TA. Topical silicone gel for the prevention and treatment of hypertrophic scar. *Arch Surg*. 1991;126:499-504.
90. Fulton JE Jr. Silicone gel sheeting for the prevention and management of evolving hypertrophic and keloid scars. *Dermatol Surg*. 1995;21:947-951.
91. Cruz-Korchin NI. Effectiveness of silicone sheets in the prevention of hypertrophic breast scars. *Ann Plast Surg*. 1996;37:345-348.
92. Li-Tsang CW, Lau JC, Choi J, et al. A prospective randomized clinical trial to investigate the effect of silicone gel sheeting (Cica-Care) on post-traumatic hypertrophic scar among the Chinese population. *Burns*. 2006;32:678-683.
93. Gold MH, Foster TD, Adair MA, et al. Prevention of hypertrophic scars and keloids by the prophylactic use of topical silicone gel sheets following a surgical procedure in an office setting. *Dermatol Surg*. 2001;27:641-644.
94. Sherris DA, Larrabee WF Jr, Murakami CS. Management of scar contractures, hypertrophic scars, and keloids. *Otolaryngol Clin North Am*. 1995;28:1057-1068.
95. Kischer CW, Shetlar MR, Shetlar CL. Alteration of hypertrophic scars induced by mechanical pressure. *Arch Dermatol*. 1975;111:60-64.
96. Linares HA, Larson DL, Willis-Galstaun BA. Historical notes on the use of pressure in the treatment of hypertrophic scars or keloids. *Burns*. 1993;19:17-21.
97. Ward RS. Pressure therapy for the control of hypertrophic scar formation after burn injury. A history and review. *J Burn Care Rehabil*. 1991;12:257-262.
98. Haq MA, Haq A. Pressure therapy in treatment of hypertrophic scar, burn contracture and keloid: the Kenyan experience. *East Afr Med J*. 1990;67:785-793.
99. Rose MP, Deitch EA. The clinical use of a tubular compression bandage, Tubigrip, for burn-scar therapy: a critical analysis. *Burns Incl Therm Inj*. 1985;12:58-64.
100. Clark JA, Cheng JC, Leung KS, et al. Mechanical characterisation of human postburn hypertrophic skin during pressure therapy. *J Biomech*. 1987;20:397-406.
101. Brent B. The role of pressure therapy in management of earlobe keloids: preliminary report of a controlled study. *Ann Plast Surg*. 1978;1:579-581.
102. Pierce HE. Postsurgical acrylic ear splints for keloids. *J Dermatol Surg Oncol*. 1986;12:583-585.
103. WebMD. Keloid and hypertrophic scar. http://emedicine.medscape.com. Accessed on March 23, 2009. ■