Scars are a natural part of dermal healing following lacerations, incisions, or tissue loss. They can vary in quality depending on the individual’s racial characteristics, the mechanism of the trauma, and conditions in which the wound healed—all of which are factors beyond the surgeon’s control. A scar on the face can have significant implications for the patient. What may seem like an insignificant issue to the casual observer can cause continuous frustration for the patient, affecting their daily lives. These can include psychological as well as social consequences, leading to a diminished quality of life. Factors that the surgeon can control include the favorable repositioning of the scar, proper alignment of the wound edges, and meticulous handling of the tissues.

A discussion with the patient and family is essential to be clear on their expectations for scar revision. During the consultation, it is imperative for the surgeon to clearly state that the goal is to improve the appearance of the scar and not erase it. The final result of the scar depends on a number of factors including the position and size of the scar, the location in relation to other anatomic features, and the patients’ predisposition for appropriate wound healing.

Corticosteroids

Intralesional corticosteroids are a frequently used adjunctive treatment for hypertrophic scars and keloids. Their mechanism of action involves reduction of fibroblast proliferation and collagen synthesis as well as suppressing inflammatory mediators. In addition, triamcinolone acetonide appears to cause a sizable decrease in α1-antitrypsin and α2-macroglobulin levels, both of which are increased in keloids and are inhibitors of collagenase. Triamcinolone acetonide suspension (Kenalog; Bristol-Myers Squibb Company) is used at a strength ranging from 10 to 20 mg/mL, though it can be given at a dose of 40 mg/mL for a resistant bulky lesion. It is injected into the dermal portion of the scar in the following
weeks postoperatively. If it is injected too superficially, this poses a risk of causing irreversible atrophy of the epidermis. Repeated injections are often required and are typically performed at 2- to 4-week intervals, with the total number of injections depending on the response and possible adverse effects. Ardehali et al.10 objectively monitored the response of keloids to intralesional triamcinolone acetate injections using 3-dimensional imaging. After treating the patients for a minimum of 8 weeks, the majority of patients achieved a greater than 50% response with a mean (SD) scar volume at the start of 0.73 (0.701) mL reduced to 0.14 (0.302) mL.2 Chowdri et al.11 reviewed their experience with 58 keloids and hypertrophic scars using intralesional corticosteroid injection. After doing serial injections at weekly intervals for 2 to 5 weeks followed by monthly injections for 4 to 6 months, more than 90% of the patients had no evidence of a recurrence at a mean follow-up of 30.5 months. Other potential adverse effects from intralesional corticosteroid injections are hypopigmentation and telangiectasias, which are at a greater risk of developing when higher concentrations are injected into the dermis. Overall, this is a relatively safe and effective therapy and is frequently used as first-line therapy in conjunction with surgical excision in the treatment of hypertrophic scars and keloids.

**Fluorouracil**

Fluorouracil, a pyrimidine analog with antimetabolite activity, has been used extensively in the treatment of cancer and as an adjunct to glaucoma surgery. More recently, it has been shown to have some efficacy in the treatment of hypertrophic scars and keloids. Fluorouracil has been shown to target rapidly proliferating fibroblasts in dermal wounds, thus inhibiting excessive collagen production.3 More specifically, it was demonstrated that fluorouracil blocks the transforming growth factor (TGF)-B2 gene in human fibroblasts, a known proinflammatory cytokine present in adult wounds that scar.4 A majority of studies published on intralesional fluorouracil use it in combination with other therapies such as corticosteroids and silicone sheeting. Fitzpatrick7 was the first to report improved efficacy and less painful injections with the combination of corticosteroids (triamicinolone acetonide) and fluorouracil in the treatment of hypertrophic scars. Manuskiatti et al.8 treated patients with previously untreated keloidal or hypertrophic sterna-otomy scars with intralesional injection of corticosteroid, fluorouracil, fluorouracil with corticosteroid, placebo, or 585-nm pulsed-dye laser (PDL) treatments. When comparing the primary outcomes of scar height, erythema, and pliability, fluorouracil as a single modality was shown to perform as well as the other intervention groups. When compared specifically with the intralesional corticosteroid group, fluorouracil did not exhibit the more common adverse sequela seen with corticosteroids such as hypopigmentation and skin atrophy. In addition, the fluorouracil group had a faster resolution of lesion size and scar induration compared with the PDL group.

Apikian and Goodman9 have found that the combination of fluorouracil with corticosteroids has fewer undesirable adverse effects than intralesional corticosteroid injection alone. Others have found that combined therapy provides a more rapid and acceptable response for the patient.10,11 Kontochristopoulos et al.12 treated keloids in 20 patients with intralesional fluorouracil (50 mg/mL) once weekly for 7 weeks. The results showed that 85% of the patients received more than a 50% improvement in scar cosmesis. Histologically, lesions after 6 injections showed overall reduction in the amount of hyalinized collagen fibers with less prominent vascularity and flattening of the dermal papillae without any signs of atrophy.12 Haurani et al.13 evaluated single-modality intralesional fluorouracil in the treatment of hypertrophic and keloid scars in a hypertrophic scar group (n=21) and a keloid group (n=31). Both groups were treated with the same series of injections and followed up at a 1-year interval. The only difference between the 2 groups was that the keloid group underwent excision of the lesion before treatment, whereas the hypertrophic scar group did not. At follow-up they measured scar volume and the patients’ satisfaction with overall scar cosmesis. The recurrence rate was 19% at follow-up for the keloid group and, 86% of patients believed that there was partial or complete improvement in the scars appearance at the end of treatment in the hypertrophic scar group.13 Recently, Hatamipour et al.14 used a combination of topical silicone with intralesional fluorouracil at a concentration of 50 mg/mL to treat 50 patients with keloids. The treatment group showed a statistical difference compared with the control group with respect to the number of patients who were keloid-free after a 12-month follow-up. The adverse effects associated with intralesional fluorouracil seem to be minimal, with one study reporting local erythema, swelling, pain, pigmentation, and occasional ulcers.11 Intral esional fluorouracil has been shown to be effective as a monotherapy but may have a greater impact when used in polytherapy as evidenced by the success in the aforementioned studies.

**Imiquimod**

Imiquimod cream, 5%, is a topical immune response modifier that stimulates interferon-α, a proinflammatory cytokine that increases collagen breakdown. It has also been shown to enhance the local production of tumor necrosis factors and interleukins.15 The use of imiquimod cream, 5%, has been used to prevent the recurrence of keloids after surgical excision. Berman and Kaufman16 used imiquimod cream, 5%, after surgical excision of 13 keloids from 12 adult patients.16 Imiquimod cream, 5%, was applied to the sites nightly for 8 weeks. At the 6-month follow-up visit, 1 keloids showed no recurrences. In another study, 8 earlobes were treated with imiquimod cream, 5%, after keloid removal. It was applied for 8 weeks followed by an observation period of 16 weeks. Twenty-four weeks after surgery, 6 patients remained recurrence-free.17 However, several other studies have not shown as promising of results with its use. Cacão et al.18 evaluated 9 patients after surgical excision of keloids and showed that keloids recurred in 8 patients (12 weeks after surgery in 7 patients).18 Malhotra and colleagues19 showed a complete recurrence of
presternal keloids within 4 weeks of stopping imiquimod therapy. Adverse effects among the aforementioned studies were rare, with the ones reported being mild, including irritation and hyperpigmentation. Overall, the role of imiquimod in the treatment of hypertrophic scars needs further evaluation in a large-scale setting.

Onion Extract

*Allium cepa*, or onion extract, is a topical gel (eg, Mederma; Merz Pharmaceuticals, LLC) that is an over-the-counter option for treatment of hypertrophic scars. It has exhibited anti-inflammatory, bacteriostatic, and collagen down-regulatory properties, even improving collagen organization in the rabbit ear model. However, limited clinical trials have failed to demonstrate any clinical improvement in scar height or erythema. Products containing onion extracts did not provide any additional improvement in scar cosmesis than a petrolatum-based ointment. Hosnuter et al evaluated the therapeutic activity of topical onion extract in gel form to treat hypertrophic scars and keloids. It was statistically ineffective in improving scar height and itching, though it was effective in reducing scar color.

Bleomycin

Bleomycin is a cytotoxic antibiotic isolated from a *Streptomyces verticillus* strain commonly used in the treatment of certain neoplasms as well as recalcitrant warts in dermatology. It acts by binding to both double- and single-stranded DNA, leading to breaks in the structure. Histologically, bleomycin has been shown to cause necrosis of keratinocytes and induces the expression of several adhesion molecules. Despite the histologic changes noted with bleomycin, the exact mechanism by which regression of hypertrophic scars and keloids occurs remains unclear. However, possible explanations have been hypothesized. An in vitro study was performed with human fibroblasts demonstrating that after incubation with bleomycin, it resulted in a dose-dependent inhibition of DNA synthesis in actively dividing cultures of skin fibroblasts. Bleomycin has also been shown to induce cell apoptosis in vitro and in vivo, which is resistant in keloids and hypertrophic scars. España et al used intralesional bleomycin to treat both hypertrophic scars and keloids. The standard concentration of 1.5 IU/mL was used intralesionally with a maximum dose of 2 mL/cm², maximum of 6 mL used per session. There was a total overall regression of 84%. More specifically, complete flattening (100%) was seen in 6 of 13 cases, highly significant flattening (>90%) in 6 cases, and significant flattening (75%-90%) in 1 case. Recurrences were noted in 2 patients at 10 and 12 months, respectively, in the form of a small nodule.

One study using intralesional bleomycin looked at 15 keloids that had failed a minimum of 3 intralesional injections of triamcinolone acetonide. Multiple jet injections of 0.1-mL bleomycin (1.5 IU/mL) were administered to each lesion (maximum volume per session, 3.5 mL), with injections repeated monthly. Favorable results were noted in regard to scar height reduction, erythema, and pliability, with a mean follow-up of 19 months. Of the lesions, 73% showed complete flattening. Aggarwal et al evaluated 50 patients with hypertrophic scars and keloids using bleomycin. Bleomycin was administered through multiple superficial puncture technique. Three applications were given at intervals of 15 days each, followed by a fourth and final application 2 months after the last application. Complete flattening was observed in 22 cases (44%) and significant flattening in 11 cases (22%). Interestingly, pruritus was relieved completely in 40 patients (88%). Recurrence was seen in 7 patients.

The adverse effect profile of intralesional bleomycin is far less extensive than systemic bleomycin. The main adverse effect of intralesional administration of bleomycin is hyperpigmentation and dermal atrophy. The systemic adverse effect profile, which includes hepatotoxicity and pulmonary fibrosis, is of limited concern for intralesional injections of bleomycin, since no incidents have been reported to date. Bleomycin therapy deserves further investigation with larger prospective studies.

Laser Therapy

Laser technology has evolved over the years to become the treatment of choice for certain types of scars. When considering a patient’s candidacy for laser scar revision, several factors need to be considered regarding the patient and certain characteristics of the scar. Skin phototype is of particular importance because darker skin tones require lower energy densities. The presence of increased epidermal pigment interferes with the targeted hemoglobin’s absorption of vascular-specific energy. This decreases the total amount delivered to the scar, creating a suboptimal result. In addition, the potential for increased melanin destruction can lead to an increased risk of postoperative skin dyspigmentation. As a result, more aggressive laser settings must be used with caution in patients with darker skin and for scars around thin-skinned regions such as the eyelids. The clinician should obtain a thorough history, looking specifically for inflammatory skin disorders (eg, eczema or psoriasis) or autoimmune disorders (eg, lupus), which can be exacerbated or interfere with the postoperative result. If there are any signs of infection present, the affected area should not be treated. Herpes simplex can be reactivated in the areas treated by laser, especially the perioral region. Because of this, patients need to be treated prophylactically with an antiviral medication (eg, acyclovir sodium, 400 mg by mouth, 3 times daily, or valacyclovir hydrochloride, 500 mg by mouth, twice daily) starting 0 to 2 days preoperatively and continuing for 10 days postoperatively or until reepithelialization has occurred.

A multitude of devices are present for laser scar treatment and are broken down into the following 3 categories: ablative, nonablative, and fractional technologies. The 3 types differ in their method and extent of thermal damage, length of downtime, adverse effect profiles, and degrees of efficacy.

Ablative carbon dioxide lasers initially had early promising results, since they resulted in successful vaporization of hypertrophic scars and keloids. However, it was later shown to result in scar recurrences and for this rea-
son is not advocated as a first-line treatment in laser scar therapy. However, it has been shown that the carbon and erbium:yttrium-aluminum-garnet (Er:YAG) laser are successful in recontouring atrophic scars from acne and varicella.

The vascular specific 585-nm PDL has shown impressive results in the treatment of hypertrophic scars and keloids. Subsequent studies have evaluated and substantiated the use of this laser in the prevention and treatment of hypertrophic scars. Several potential mechanisms have been described to explain the effect of PDL on scars. One study showed that PDL reduces the expression of TGF-β1, a proinflammatory cytokine, along with reduced fibroblast proliferation and collagen type III deposition. Other plausible mechanisms include selective photothermolysis of vasculature and breaking of disulfide bonds with subsequent collagen realignment. Several studies have demonstrated that higher fluences and shorter pulse durations improve scar size and pliability. Nouri et al looked at the effectiveness of the 585-nm vascular lasers, comparing different pulse durations (450 microseconds [µs] vs 1.5 milliseconds [ms]). The minimum length of the patient's scars were 2.1 cm, and they started treatment at the time the sutures were removed. The scars were divided into 3 sections: one was treated with the laser at a pulse duration of 450 µs; the midsection acted as a control; and the third was treated with a pulse duration of 1.5 ms. Both treated sections had an improvement with the shorter and longer pulse, showing an average improvement of 92% and 89%, respectively. While there was no significant difference noted between the 2 pulse durations, the difference between the treated and untreated sections was statistically significant. The most common adverse effect of PDL treatment is postoperative purpura, often persisting for 7 to 10 days. Edema may also occur in the early posttreatment period. Some clinicians use this to their advantage and perform intralesional injections of corticosteroids because it is easier to administer into the edematous laser-irradiated scar.

The 1064-nm Nd:YAG nonablative laser has been shown to selectively suppress collagen production in fibroblast cultures, and clinical studies have shown some promise in the treatment of scars. Cho et al evaluated the efficacy and safety of the 1064-nm Q-switched Nd:YAG laser with low fluence on keloids and hypertrophic scars. Their results showed a decrease in the mean score for the following lesion characteristics: pigmentation, vascularity, pliability, and height, indicating that this may be a potential treatment option for hypertrophic scars. Badawi et al used the 1064 Nd:YAG laser to treat scarring in darker skin types (Fitzpatrick skin type III-VI) and showed that it is a safe and effective treatment for scars in this patient population with reduced risk of pigment complications. Only mild transient posttreatment erythema was noted in the aforementioned studies, and no major adverse events including worsening of the lesion and posttherapy dyschromias were observed.

Fractional photothermolysis has been successfully used in the treatment of a variety of scars including surgical, acne, and traumatic scars. It is a nonablative resurfacing technique that creates microscopic noncontiguous columns of thermal injury in the dermis (commonly referred to as microscopic thermal zones) that are surrounded by zones of viable tissue. Within the microscopic thermal zones, localized epidermal necrosis occurs alongside collagen denaturation, followed by expulsion of the necrotic debris and neocollagenesis. These zones comprise approximately 15% to 25% of the skin surface area per treatment session. There is subsequent stimulation of re-epithelialization and repair that is mediated by the adjacent columns of intact tissue. Because there is a large percentage of skin that is unaffected, healing occurs faster, minimizing the total downtime for the patient. In a case report, Behroozan et al noted a 75% overall improvement in a surgical scar on the chin after a single treatment with the 1550-nm Fraxel SR (Solta Medical). A larger follow-up study consisting of 13 patients with postsurgical scars showed that after an average of 3 treatments, half of the patients had greater than 75% improvement. Glaich et al demonstrated 51% to 75% improvement in hypopigmented facial scars in 6 of the 7 treated patients with the 1550-nm Fraxel SR. In a comparison study between the 1550-nm fractionated laser and PDL, Tierney et al showed that the fractionated laser was more effective than PDL for improving the cosmesis of surgical scars. A prospective clinical study looked at 13 adults with Fitzpatrick skin types I to III and facial surgical scars with a postoperative duration longer than 6 months. Subjects were treated with the 1550-nm Fraxel SR once every 4 weeks for a total of 4 treatments. At completion of the treatments, there was a statistically significant improvement in the patient’s assessment of color, stiffness, thickness, and irregularity of the scar. In addition, observer ratings showed a statistically significant improvement in pigmentation, thickness, and pliability but not in vascularization. In general, the adverse effects of fractional lasers are minimal and transient. The complications that have been observed include erythema and posttreatment edema, and postinflammatory dyspigmentation.

**Silicone Gel**

Silicone gel is a cross-linked polymer of dimethylsiloxane used as an impregnated elastic sheet, silicone gel sheeting (SGS), silicone cream, or a topical gel. It is noninvasive and has the advantages of being inexpensive, painless, and easy to use. The exact mechanism by which SGS exerts its effects remains unclear and continues to be a subject of controversy. Some have suggested that it is the hydration of the stratum corneum rather than the inherent properties of the silicone itself that affects wound healing. In 2004, Hanasono et al performed in vitro testing on human fibroblasts from various tissues including normal, keloid, and fetal skin. Their results suggested that silicone gel is responsible for increased basic fibroblast growth factor levels in normal and fetal dermal fibroblasts and acts as a modulator in the expression of such growth factors. It has also been shown that SGS may act by down-regulating fibroblasts and decreasing fibrogenic cytokines such as TGF-β2. Gallant-Behm et al showed that SGS significantly decreased the epidermal expression of the profibrotic cytokine interleukin-1β and...
compared with other nonsurgical treatment, no treatment of hypertrophic or keloid scars was reported. Published a review in which SGS for prevention of scarring. Lastly, it appeared that the SGS reduced dermal and epidermal thickness because of a reduction in the skin and was necessary to hinder angiogenesis and cause local tissue hypoxia, leading to a reduction in the size and thickness of scars.\textsuperscript{70} A subsequent in vivo rabbit ear model explored several factors of SGS in an attempt to explain the mechanism of action. The degree of occlusion played an important role, with total occlusion having a greater decrease in scar hypertrophy than semiocclusion. They were also able to show that the oxygen permeability of SGS is not important for its mechanism of action. With the use of multiple sheets of polyurethane dressing, which made the surface impermeable to oxygen, there was no evidence to indicate that this interfered with the reduction of scarring. Lastly, it appeared that the SGS reduced dermal and epidermal thickness because of a reduction in keratinocyte stimulation.\textsuperscript{70}

The theory that oxygen permeability does not play a role is a new idea. It was previously thought that the high oxygen permeability of SGS increased oxygen tension in the skin and was necessary to hinder angiogenesis and cause local tissue hypoxia, leading to a reduction in the size and thickness of scars.\textsuperscript{71,72} In 2006, the Cochrane Collaboration published a review in which SGS for prevention and treatment of hypertrophic or keloid scars was compared with other nonsurgical treatment, no treatment, or a placebo.\textsuperscript{73} The review consisted of 15 randomized, quasirandomized, or controlled clinical trials. In the prevention studies, SGS was found to reduce the incidence of hypertrophic scars in those patients prone to scarring compared with controls. In the studies using SGS as a treatment option, statistically significant reductions were noted in scar length, width, thickness, and color amelioration. In summary, they concluded that the effects of SGS on hypertrophic and keloid scarring were unclear and warranted rigorous evaluation. O'Shaughnessy et al\textsuperscript{74} further explored the theory of occlusion reducing hypertrophic scarring by performing histomorphometric analysis of the epidermis in occluded vs tape-stripped scars. Three occlusion groups were present, one of which was a topical silicone gel. Each of the occlusive treatments was shown to decrease the transepidermal water loss, while tape stripping acted to the contrary. In additional, tape stripping significantly increased the scar elevation index, epithelial thickness, and cellularity, while the occlusion group showed reductions in all the former factors.\textsuperscript{75} In a randomized controlled trial by de Giorgi et al,\textsuperscript{76} silicone gel was applied to 110 surgical incisions for 60 days after the removal of stitches. In the treatment group, only 18 patients (27\%) had formation of a nonphysiologic scar compared with the control group, where 25 patients (55\%) had an altered scar.\textsuperscript{77} Li-Tsang et al\textsuperscript{78} evaluated the effects of silicone gel dressing and pressure therapy on posttraumatic hypertrophic scars. After 6 months of intervention, they concluded that SGS was more effective in alleviating pain and pruritus rather than scar thickness. The combined therapy group along with the pressure therapy group showed significant improvement in scar thickness.\textsuperscript{79} Dermatix (Meda Pharma), a topical silicone gel, was developed to address some of the limitations of SGS including visibility and limited adherence in some areas that may be hypermobile. Van der Wal et al\textsuperscript{80} found that the topical silicone gel was effective in promoting the maturation and improving the surface roughness of scars.\textsuperscript{77} Adverse effects with silicone polymers have been reported but most are mild. The most common are the adverse effects of skin maceration and skin eruption, which have been reported in multiple studies.\textsuperscript{73-77} All adverse effects resolved after discontinuing therapy without any adverse sequelae.

Pressure Therapy

Pressure therapy has been used as a common form of conservative management in the treatment of scars since the late 1960s.\textsuperscript{78} Several mechanisms have been proposed to explain how pressure therapy exerts its effect on hypertrophic scars. Pressure therapy is believed to accelerate wound maturation by thinning of the dermis, decreasing edema, and decreasing blood flow and oxygen to create a hypoxic environment. This hypoxic environment results in fibroblast degeneration and decreased collagen synthesis (Figure 1 and Figure 2).\textsuperscript{79} It was demonstrated in one study that pressure therapy for hypertrophic scars restored in part the extracellular matrix organization that is observed in a normal scar. In addition, it was observed histologically that pressure therapy induced the disappearance of \(\alpha\)-smooth muscle actin-
expressing myofibroblasts, likely by apoptosis.80 Myofibroblasts, the main cellular type observed in granulation tissue, are thought to be responsible for the forces that determine wound contraction81 and pathological contractures seen in hypertrophic scars.82 The role of prostaglandin E2 (PGE2) has been studied and shown to be present in increased concentrations following burn scars.83 Increased levels of PGE2 are known to increase the expression of collagenases. Reno et al84 showed that pressure therapy induced a significant increase in the release of PGE2 in hypertrophic scars suggesting an alternative mechanism for hypertrophic scar prevention. An in vitro study was performed to investigate the effects of pressure therapy on the growth of human scar-derived fibroblasts. They determined that pressure inhibits the growth and activity of human scar fibroblasts in addition to decreasing the amount of TGF-β1, a regulatory cytokine responsible for the differentiation of fibroblasts into myofibroblasts.85 Much of the research investigating pressure therapy has been focused on burn scars. Groce et al86 used pressure therapy in a randomized study to evaluate the effect on burn scars. Forty-six children were studied at 3 intervals within 6 months by a blinded observer. No significant difference was found between the 2 groups with factors such as vascularity, scar pigmentation, and pliability with the exception of scar height that did show a significant improvement.86 The standard recommended pressure is 25 mm Hg,87 but others have observed equally efficacious results using lower compression levels of 10 to 15 mg Hg (Figure 3).88,89 Overall, there is evidence that pressure therapy can play a role in the treatment of hypertrophic scars; however, more definitive research needs to be undertaken to evaluate optimum treatment parameters.

Radiotherapy

Radiotherapy has been used in the past for the treatment of keloids. Its use as a monotherapy has been shown to be inadequate for the treatment of keloids,90 which is why it is frequently combined with surgical resection. The suggested mechanism of action of radiation is the control of collagen synthesis by affecting fibroblast proliferation and inducing apoptosis.91 While various dosing regimens have been described, the best results have been achieved with 15 to 20 Gy over 5 to 6 sessions in the early postoperative period.92,93 Typically, irradiation is started 24 to 48 hours after surgery, for a total dose in the previously described range (Figure 4). A 26-year retrospective review was done to evaluate the efficacy of combination surgery and radiation in the treatment of auricular keloids. Seventy-six patients were included in the study, and the total dose of radiation administered ranged from 10 to 45 Gy, by means of weekly fractions of 5 Gy each. After a mean follow-up of 48 months, 10 cases recurred, with a 5-year relapse free rate of 80%.94 A prospective study was performed on 21 patients with 32 keloids that were treated with excision followed by radiotherapy (12 Gy in 3 or 4 fractions) with a minimum follow-up period of 12 months. The recurrence rate was 72% after a mean follow-up period of 19 months, suggesting that it might be less efficacious then suggested by other studies.95 The risk of radiation-induced malignancy seems to be exaggerated seeing that only a few cases have been described, with large treatment cohorts and extensive follow-up periods providing little evidence to substantiate this claim.91,96,97 Other potential complications reported have been hyperpigmentation and erythema. The treatment schedule and follow-up varies considerably among the studies, and most sources agree that this treatment modality should be reserved for scars that are unresponsive to other treatments.
SURGICAL TECHNIQUES

A detailed analysis of the scar is paramount in the initial evaluation of the patient. A favorable scar is one that is narrow, well positioned along aesthetic subunit borders, and in parallel with relaxed skin tension lines. Facial scars tend to mature over time and typically continue to improve for at least 1 year. Traditionally, it has been advised to allow scars to undergo that maturation period before pursuing any revision techniques. However, if the scar is not exhibiting favorable characteristics, earlier intervention after the first 60 to 90 days may be appropriate. There are a multitude of techniques to use when attempting to surgically revise a scar. These include fusiform excision with linear closure, z-plasty, w-plasty, and geometric broken line closure to name a few (Figure 5). In addition, serial excisions can also be performed for a large scar that cannot be primarily closed with a single definitive excision. This takes advantage of the skin’s ability to stretch and slowly accommodate over time.

Z-plasty is the classic technique that provides scar irregularization while changing the scar direction so that the majority of the length of the scar is aligned with the relaxed-skin-tension lines. The classic Z-plasty is a z-shaped incision using the scar as a central member and 2 peripheral members of the z configuration both equal in length, forming equal triangular flaps. In addition, it lengthens a contracted scar by adding additional intervening tissue, which makes it particularly useful in elongating a contracted scar. The amount of added length to the scar can be varied by adjusting the angles of the triangle. For example, angles of 30° will provide lengthening of the contracted area by 25%, whereas 45° angles will lengthen a wound by 50%, and 60° angles will yield a 75% lengthening. Z-plasty is useful in changing the direction of the scar, increasing scar length, elongating a contracted scar, and shifting malpositioned facial landmarks.

A particularly good use of Z-plasty is correcting trapdoor scars, also known as pin cushion scars. These scars are formed by circular or semicircular scars, which, when they contract, tend to bunch the central soft tissue, creating a trapdoor-like flap. Correction of this involves placing small Z-plasties around the perimeter of the wound, allowing for interdigititation of the flap with the surrounding skin, and in effect, lengthening the circular contracted scar (Figure 6).

When multiple Z-plasties are combined along a scar, the same benefits are maintained as a single Z-plasty. However, the resultant scar tends to be less noticeable because the various components are smaller. This is most useful in long scars, which require irregularization as well as change in scar direction, for which using a single Z-plasty would require a long incision and thus more visible scars (Figure 7).

W-plasty is a form of irregularization that tends to make a scar less noticeable and better camouflaged. Unlike Z-plasty, which is a transposed flap, W-plasty is an interposed flap that does not create lengthening of the scar. W-plasty is created by excising connected triangular units to break up the scar line in a regularly irregular fashion. A series of consecutive triangles are marked out along the wound or scar edge. The arms of the triangles should be approximately 5 to 7 mm in length, with ideally 1 arm of the triangle drawn parallel to relaxed skin tension lines.
Geometric broken-line closure is a technique that creates an irregularly irregular scar using random geometric figures as interposed flaps on each side of the excision. These geometric units are a series of squares, rectangles, and various shaped triangles placed in a random pattern. The geometry of the resultant scar is less detectable to the eye than the more predictable W-plasty. This technique is best suited for lengthier scars that traverse an aesthetic unit or broad flat surfaces such as the forehead and cheek. Like W-plasty, geometric broken-line closure is formed by interposed flaps without affecting length, as opposed to the transposed flaps and resultant increase in length with Z-plasty. Scar irregularization techniques are typically followed by a second-stage local dermabrasion. This is usually performed 6 to 8 weeks after the initial surgical procedure.

DERMABRASION

Dermabrasion is a method of controlled superficial skin ablation that is useful for smoothing out elevated scars and other skin contour irregularities. The concept of dermabrasion is that the superficial skin layers including the epidermis and part of the papillary dermis are removed, allowing the wound to re-epithelialize by the surrounding epithelium and underlining adnexal structures. It is best performed at a 6- to 8-week interval, since it has been shown that rewounding during fibrillogenesis stimulates more epidermal cells to migrate to the wound, leading to an improved appearance of the scar.98 The advantages of this modality are that it is not costly, discomfort is minimal for the patient, and there is little downtime for the patient. Also, the time required to perform the procedure is usually less than 15 minutes, making it cost efficient. Ideal candidates for dermabrasion are fair-skinned patients because there is a lower risk for dyspigmentation following the procedure. Some recommend antiviral prophylaxis (eg, acyclovir sodium, 400 mg by mouth, 3 times daily, or valacyclovir hydrochloride, 500 mg by mouth, twice daily) against herpetic infections on all patients, regardless if they have a history.99 The rationale for providing prophylaxis during the re-epithelialization process is that the herpes virus requires viable epidermal cells to establish infection, which puts the patient at greatest risk for an outbreak 7 to 10 days after surgery. Prophylaxis should also be provided 0 to 2 days preoperatively.

A diamond fraise is used instead of a wire brush to gain greater control of the instrument and to decrease the chances of abrading too deeply into the reticular dermis, creating an unnecessary scar (Figure 8).100 Preparation of the area to be dermabraded is accomplished with local anesthesia. This not only provides a nerve block but also infiltrates the area, providing distention of the skin, which assists in the procedure. The skin surrounding the scar during the procedure is stretched and tightened with 3- or 4-point tension to provide an even and firm surface for dermabrasion. The fraise should be rotating in a clockwise fashion and applied perpendicular and oblique to the axis of the scar. Feathering is appropriate to avoid any demarcation between treated and untreated regions.

During the procedure, entering the superficial papillary dermis will reveal small capillary loops easily identified by pinpoint bleeding.101 As the dermabrasion proceeds deeper, small parallel strands of white-colored collagen can be appreciated, indicating the appropriate depth. As mentioned previously, proceeding deeper into the reticular dermis will lead to damage of the underlying adnexal structures, which are critical in the proliferation of undamaged epidermal cells across the abraded surface. This can lead to unnecessary scarring.
An alternative to mechanical dermabrasion is manual dermabrasion or dermasanding. Some have advocated for this modality because it is a safer procedure in that there is no aerosolization of infectious particles or blood splatter that could infect the staff. Poulos et al. performed a prospective, randomized blinded study to assess the effectiveness of dermasanding on the appearance of surgical scars on the face. Overall, there was significant improvement in the overall appearance of the treated scars at the 3- and 6-month follow-up examinations. Gillard et al. compared the results of mechanical vs manual dermabrasion and found no differences between the 2 methods for camouflage of the scar, contour correction, hyperpigmentation, or infection during the 6 months of follow-up. They determined that both methods were equally effective at improving the cosmetic appearance of surgical scars.

Immediately after treatment, an occlusive dressing such as polyethylene oxide hydrogel (Vigilon; Bard Home Health Ltd) is applied. This is left in place for 48 hours, and after removal the patient is instructed to keep the area moist at all times with bacitracin for the next 7 to 10 days. The most common complication after dermabrasion is posttreatment pigmentary alteration. Other potential complications include milia formation and persistent erythema. The erythema is usually less of an issue for female patients, since they can use makeup to cover up the area when re-epithelialization has completed.

Overall dermabrasion is one of the oldest forms of resurfacing and is very effective at improving the cosmetic appearance of acne and surgical scars (Figure 9 and Figure 10).

**EMERGING SCAR REDUCING THERAPIES**

**ACE Inhibitors**

Angiotensin-converting enzyme (ACE) inhibitors prevent the conversion of the enzyme peptidyl dipeptidase, which hydrolyzes angiotensin I to angiotensin II and inhibits the degradation of bradykinin, a potent vasodilator. Angiotensin-converting enzyme is present in tissues composed largely of fibrillar collagen such as heart valves, and it is well documented in the cardiovascular literature that up-regulation of ACE contributes to fibrous tissue production following myocardial infarction. Stecklings et al. demonstrated the existence of a renin-angiotensin system within human skin. It has been shown that ACE is more active in higher levels in human pathologic scar tissue compared with normal and wounded skin. Recent data have indicated that angiotensin II stimulated the expression of type I collagen in human dermal fibroblasts suggesting its involvement in skin wound healing. Iannello et al. presented 2 case reports using low-dose enalapril to treat postoperative keloids. One of the patients started therapy with enalapril (10 mg, once a day) and after 4 months reported rapid improvement and eventual recovery of the keloid scar. The second case involved a postsurgical abdominal keloid scar of 2 years duration. After 6 months of low-dose enalapril therapy, there was marked improvement in the cosmetic appearance of the keloid. This is a preliminary observation that needs to be validated by a larger, randomized controlled study for the potential use as a novel therapeutic agent for the treatment of scars.

**NSAIDs and COX-2 Inhibitors**

There has been a growing interest in studying the use of nonsteroidal anti-inflammatory drugs (NSAIDs) and cyclooxygenase-2 (COX-2) inhibitors for the use in scar reduction. An early response in the inflammatory cascade is the induction of COX-2, which catalyzes the conversion of arachidonic acid to PGE2. Prostaglandin E2 is the major prostaglandin produced in the skin, derived primarily from keratinocytes within the epidermis. It
is believed that PGE2 plays a role in modulating keratinocyte proliferation and differentiation.113 The COX enzymes, which include the homeostatic COX-1 and inflammatory COX-2, are both inhibited by NSAIDs, such as aspirin and ibuprofen. Given some of the adverse effects of NSAIDs, COX-2 inhibitors were developed, such as Celecoxib (Celebrex, Searle), which specifically targets the COX-2 enzyme. The role that prostaglandins play in scar formation is not totally clear; however, it has been demonstrated in an animal model that they induce fibroblast proliferation in vitro and collagen production in wounds in vivo.114-116

Topical celecoxib was applied to incisional wounds to evaluate its effect on inhibiting various parameters of inflammation. It was shown to significantly lower levels of TGF-β1 and cause a 50% decrease in wound PGE2 levels during the inflammatory phase of wound healing. This later corresponded to decreased collagen deposition and less scar tissue in the mouse model.117 Similarly, Hoffman et al118 were able to show that COX-2 inhibitor celecoxib did not delay cutaneous wound healing in a mouse model.119 Despite these promising results, there is conflicting evidence when looking at the effect of COX-2 inhibitors on wound healing. One study used COX-2 inhibitors on full-thickness incisional wounds using a mouse model and was unable to show a significant effect on the morphologic features of the wound.119 Hardy et al120 showed that a selective COX-2 inhibitor did not alter keratinocyte proliferation or differentiation following abrasion. This result indicated that even though COX-2 expression was coincident with transient epidermal hyperplasia and keratinocyte proliferation during healing, it appears to be dispensable for proper healing because of redundant mechanisms.120

A similar topic of debate is whether inhibition of COX-1 can result in delayed wound healing. Kamper et al121 provided evidence that COX-1 played a crucial part in the regulation of the cutaneous wound healing process, suggesting that inhibition may cause delayed wound healing. A recent review article examined NSAIDs for wounds and suggested that the inhibition of PGE2 production may exacerbate excessive scar formation when used during and suggested that the inhibition of PGE2 production may exacerbate excessive scar formation when used during the later proliferative phase.122 Another study showed the contrary, suggesting that COX-1 inhibitors have no effect on tensile strength or delayed wound healing.123

Given the lack of randomized studies and the discordance among the studies assessing the efficacy of NSAIDs and COX-2 inhibitors, additional studies are needed to evaluate their potential as a scar reducing agent.

TGF-β Superfamily

Transforming growth factor-β is secreted by most cells involved in wound healing, including lymphocytes, neutrophils, macrophages, and fibroblasts. Mammals have 3 TGF-β isoforms.124 Although they share a high degree of structural similarity, they each play a separate and distinct role in the healing and scarring response. A consistent difference has been demonstrated among the different isoforms between adult and fetal wounds. It has been demonstrated that TGF-β1 and TGF-β2 are present at high levels in adult wounds that scar but at much lower levels in fetal skin wounds that do not scar.125,126 Conversely, TGF-β3 is present at high levels in fetal skin wounds and low levels in adult wounds.127,128 There is evidence that TGF-β3 counteracts the effects of TGF-β1 and TGF-β2 by reducing the influx of inflammatory cells into the wound, thereby reducing connective tissue deposition and subsequent scarring.129 Waddinton et al130 used a lentiviral vector expressing TGF-β3 in a mouse skin wounding model and showed reduced re-epithelialization density and fibroblast-myofibroblast transdifferentiation within the wound area, both of which are indicative of reduced scar formation.130

Multiple clinical trials have been performed with avotermin (Juvista; Renovo), a recombinant, active, human TGF-β3.131 A series of 3 double-blind, placebo-controlled, phase 2/3 studies using intradermal avotermin to incisions were collectively able to show that it reduced skin scarring compared with the controls. Treatment was given both before wounding and 24 hours later to the margins of 1-cm full-thickness skin incisions, which was a previously established schedule in preclinical trials. The dose ranged from 5 to 500 ng/100 µL per linear centimeter of wound margin, with all doses showing a significantly improved total scar score. One study in particular showed significant differences in collagen organization, with the avotermin-treated scars more closely resembling the basket-weave architecture of normal skin.132 A subsequent study investigated the scar-reducing effects of different dosing regimens of avotermin compared with controls 12 months after surgery.133 The patients were randomized to 2 groups. One group received avotermin at 50 ng/100 µL per linear centimeter and group 2 received avotermin at 200 ng/100 µL per linear centimeter of wound margin, with the dosing schedule at day 0 and day 1. At day 0, 1-cm full-thickness incisions were created. The primary end point was the within-subject difference in visual analog scale scores assigned by an independent assessment panel. They determined that avotermin at 200 ng/100 µL per linear centimeter of wound margin, given once or twice, produced a significantly improved scar appearance compared with controls. In addition, compared with the lower dose of 50 ng/100 µL, the 200 ng/100 µL proved to be a superior dose for improving scar appearance.

A more recent study looked more specifically at patients undergoing surgical scar revision of linear scars that were 5 cm or longer.134 An avotermin concentration of 200 ng/100 µL was used in the typical day 0 and day 1 dosing frequency. They used separate areas of the same wound as control and treatment zones. This allowed for precise histologic examination of avotermin vs placebo within the same wound, which had not been previously demonstrated. Of the avotermin-treated scar segments, 74% were histologically more similar to normal skin compared with 26% of placebo-treated scar segments. They were also able to show a greater reduction in scar surface area from baseline when comparing avotermin with placebo in addition to an overall improved scar appearance. The most recent of phase 2 clinical trials with avotermin showed that avotermin, 500 ng/100 µL, significantly improved groin scar appearance compared with placebo.135 All studies demonstrated a favorable toler-
ability profile with no serious adverse events reported. Two patients in one study developed hypersensitivity reactions locally around the injection site, which resolved without any further sequelae.133 So et al134 did not show any significant differences in the incidence and severities of itching, pain, or edema reported between the avotem- 

CONCLUSIONS

A number of therapeutic strategies have been developed in an attempt to reduce and prevent scars. Despite this, there still remains no universal consensus regarding the optimal treatment strategy. The goal of scar revision is not to erase but to make them better. Sometimes despite one's best efforts, an optimal outcome may not occur. This is why managing the patient's expectations preoperatively with realistic goals clearly stated by the surgeon is of the utmost importance. Analyzing the characteristics of the scar is one of the keys to determining how to make the scar less noticeable. Scar revision can be challenging even for the experienced surgeon. However, using the aforementioned techniques can provide the surgeon with the complete armamentarium necessary to positively influence each and every patient.

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Correspondence: J. Regan Thomas, MD, Department of Otolaryngology—Head and Neck Surgery, University of Illinois at Chicago, 1855 W Taylor St, MC 648, Chicago, IL 60612 (thomarj@uic.edu).

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REFERENCES


