

Laser revision of scars and striae  
[The Use Of Lasers In Dermatology]

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Integumental injury sets the cascade of wound healing events into motion. In most cases, wound healing results in the restoration of skin, which is smooth and normal in appearance. Despite its normal appearance, the repaired skin will ultimately achieve only 70-80% of its original tensile strength. This is not the consequence of the healing process that is of concern. When the wound healing process deviates from its orderly pattern, scarring results. Scars are common complications of wound healing that affect millions of

individuals. Though the pigmentary and vascular alterations associated with wound healing are often transient, the textural changes caused by collagen disruption are often permanent.

The wound healing process can be divided into three stages: inflammation, granulation tissue formation, and matrix remodeling (1,2). The initial stage is defined by a structured sequence involving inflammatory cells. This cascade is orchestrated by neutrophils. Subsequently, macrophages elaborate a variety of cytokines, which create an environment amicable to granulation tissue formation. Finally, fibroblasts migrate into the area, proliferate, and recapitulate ontogeny by depositing new collagen—first type III and later type I. Simultaneously, new capillaries are produced under the influence of angiogenic factors released into the wound environment. A problem arises when this organized process takes a detour. An overzealous healing response may occur, creating a raised nodule of fibrotic tissue. Alternatively the deleted collagen is not adequately replaced, forming a pitted "golf ball" appearance. In either case, the scar is often a legacy of skewed wound healing.

Although rarely posing a health risk, patients with scars often present with complaints of associated pruritus or dysesthesia. It is imperative that the consulting physician does not overlook the patient's perception of aesthetic disfigurement, which can be detrimental to the patient's psyche. The physician must not only assess pertinent characteristics of the scar but must also take into consideration certain patient variables. The purpose of this article is to review the strengths and limitations of current laser technology used to improve the appearance and symptomatology of hypertrophic scars, keloids, striae, and atrophic scars.

Scar characteristics 

Proper classification of a scar is important. Subtle differences in clinical characteristics define the diagnosis and subsequently the treatment protocol (Table 1). Qualities such as color, texture, and morphology, as well as previous treatment attempts, affect the fluences used as well as the number of predicted treatments required for revision (3).

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Table 1. Clinical responses of scars to laser therapy

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Hypertrophic scars versus keloids 

Hypertrophic scars are pink, raised, firm, erythematous scars characterized by a decreased expression of collagenase. These scars form as the result of overzealous collagen synthesis coupled with limited collagen lysis during the remodeling phase of wound healing. The result is the formation of thick, hyalinized collagen bundles consisting of fibroblasts and fibrocytes. These collagen bundles are arranged in nodules. Despite the obvious tissue proliferation, hypertrophic scars remain within the confines of the original integumental injury. Hypertrophic scars along with their keloid counterparts usually form in body areas that exhibit slow wound healing or in pressure/movement-involved areas. The hypertrophic scar usually forms within the first month following injury. Approximately one-third of patients with hypertrophic scars complain of pruritus and dysesthesia (4,5). Unlike keloids, hypertrophic scars may regress over time.

Keloids are raised, reddish-purple, nodular scars which, upon palpation, are firmer than hypertrophic scars. Keloids exhibit a prolonged proliferative phase, which is the result of an inherited metabolic alteration in collagen. The result is thick, hyalinized collagen bundles similar to those produced by hypertrophic scars. Unlike hypertrophic scars, keloids extend beyond the margins of the inciting wound and do not regress over time (6). Keloids have also been shown to contain an increased amount of hyaluronidase (7). Their formation varies from weeks to years after the initial trauma. Although occurring in all skin types, keloids appear most frequently in patients with darker skin tones.

## Striae distensae

Striae or "stretch marks" are linear bands of atrophic or wrinkled skin. They form as the result of rapid weight loss or weight gain in areas such as the abdomen, hips, breasts, and around joints. Dermal inflammation and dilated capillaries mark the initial presentation of striae. As a result, striae initially appear erythematous with characteristic pink, lavender, and purple hues. Late in their course, striae appear hypopigmented and fibrotic. The pathogenesis of striae remains unclear, but it has been hypothesized that estrogen and mast cell degranulation with elastolysis play a role (5).

## Atrophic scars

Atrophic scars are dermal depressions most commonly caused by collagen destruction during the course of an inflammatory skin disease such as cystic acne or varicella. Surgery and trauma may also result in the formation of atrophic scars. These pitted lesions form a "golf ball"-type surface on affected skin areas. Most patients attempt to camouflage these disfiguring lesions with makeup. However, the appearance of these scars is often exacerbated by makeup due to its enhancement of the problematic textural variation.

## Patient variables

When evaluating a patient as a candidate for laser surgery, one must consider certain "patient factors" which may make the candidate less than ideal. These factors are "yellow lights" of warning, and in and of themselves are not contraindications to laser surgery. More precisely, these factors are indicators to "proceed with caution," with full knowledge that they may complicate both the surgical and postoperative management of the patient.

## Darker skin phototypes

Ethnic background is an important consideration when assessing the likelihood of a scar developing into a keloid or hypertrophic scar. Hypertrophic scars and keloids affect approximately 4.5-16% of the African American and Hispanic populations. Caucasians are less susceptible, with a white:black susceptibility ratio estimated at 1:3.5 to 1:15 (7). Likewise, ethnic background must be considered when contemplating laser outcomes. The presence of an increased amount of epidermal pigment in darker skin tones (type III or greater) interferes with the absorption of pulsed dye laser energy by hemoglobin. As a result, the amount of energy effectively delivered to dermal scar tissue is reduced. This phenomenon raises two concerns: the efficacy of laser scar treatment is reduced, and destruction of the epidermal melanin results in postoperative hypopigmentation. When considering cutaneous laser resurfacing with the CO<sub>2</sub> or erbium laser, the patient and the treating physician must be prepared for the possibility of transient posttreatment hyperpigmentation.

## Concurrent inflammation or infection

Patients with infectious or inflammatory processes must await resolution before proceeding with laser surgery. In the case of bacterial or viral infection (e.g., herpes simplex, verrucae), the possibility that the infection will koebnerize by laser irradiation must be considered. In patients with concurrent inflammatory skin disorders (e.g., cystic acne, psoriasis, dermatitis), the condition itself may worsen with laser treatment and dermal inflammation may impede postoperative healing and clinical effect.

## Medication use

Patients with atrophic acne scars who present for laser resurfacing are likely to have a history of isotretinoin

use. Isotretinoin can foster the development of hypertrophic scars after dermal resurfacing procedures due to its effect on collagen metabolism and wound repair (8). Therefore patients must have completed their last course of isotretinoin a minimum of 6 months before laser resurfacing.

## Unrealistic expectations

Currently there are no treatments that offer patients 100% improvement. Patients who would not consider laser therapy successful with less than 100% improvement of clinical results have unrealistic expectations and are not good candidates for laser surgery. Patients should realize that some degree of scarring will persist, and even multiple retreatments may not be sufficient to completely eradicate the scar(s). In addition, patients must understand their role in proper postoperative skin care. Strict patient compliance is necessary for optimal results. A noncompliant patient is not a good candidate.

## Treatment of hypertrophic scars and keloids

### Background

Traditional treatments of hypertrophic scars and keloids often involve numerous patient-"unfriendly" techniques. These techniques include topical and intralesional corticosteroids, topical retinoic acid, surgical excision and/or grafting, cryosurgery, radiotherapy, pressure therapy, intralesional interferon, occlusion, and silicone gel sheeting (6-9). These inconvenient and often painful treatments result in side effects, including atrophy and dyspigmentation (6). In addition, scar recurrence rates after traditional treatments are high.

The first laser used in the treatment of hypertrophic scars and keloids was a continuous wave argon laser. While initial reports were encouraging, subsequent studies failed to confirm the treatment's efficacy (10-13). Similarly, use of the continuous wave Nd:YAG laser (1064 nm), which selectively inhibits collagen production by a direct photobiological effect and creates tissue infarction with subsequent charring and sloughing of the treated area, also showed initial clinical improvement, but the results were transient and scar recurrences were common (14-16). Similar recurrences were observed when hypertrophic scars and keloids were excised or vaporized with a continuous wave CO<sub>2</sub> laser. When treated with the CO<sub>2</sub> laser, these scars were shown to universally recur within 1 year of treatment (17-21).

The early 1980s brought about a revolution in the use of laser technology in dermatologic treatment with Anderson and Parrish's (22) publication detailing the theory of "selective photothermolysis." Selective photothermolysis describes specific absorption of laser energy to achieve temperature-mediated localized injury in a target. Their theory led to the invention of pulsed lasers that were target-specific and highly selective. The increased selectivity decreased the amount of thermal damage to healthy tissue, thereby decreasing scarring and other unwanted side effects.

By the late 1980s the effectiveness of the vascular-specific 585 nm pulsed dye laser in treating a variety of vascular lesions (e.g., port-wine stains, telangiectasias) was widely known. Some clinicians were beginning to use it to reduce persistent erythema associated with both hypertrophic scars and keloids. However, the improvement in skin texture, bulk, and pliability of pulsed dye laser-treated scars shown by Alster et al. (23) in 1993 had not been expected. Initial testing of the pulsed dye laser system in the treatment of argon laser-induced port-wine stain scars produced improved skin texture and color without evidence of worsening or recurrence at the 6-month end-study evaluation (23) and even at remote examination 4 years later (4). Subsequent studies showed significant clinical improvement (e.g., improved texture, pliability, color, and bulk) in the treatment of surgical and traumatic hypertrophic scars within one or two treatments with the pulsed dye laser system (24-27). Hypertrophic and keloid median sternotomy as well as burn scars were also proven to be responsive to pulsed dye laser therapy without evidence of recurrence (28,29) (Figs. 1 and 2).

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Fig. 1. Hypertrophic traumatic scars on the cheek (A) before and (B) 6 weeks after the second 585 nm pulsed dye laser treatment ( $5.0 \text{ J/cm}^2$ , 10 mm spot size).

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Fig. 2. Keloid scar on the anterior chest unresponsive to intralesional corticosteroids and silicone gel application (A) before and (B) 2 months after the second 585 nm pulsed dye laser treatment ( $5.0\text{-}5.5 \text{ J/cm}^2$ , 10 mm spot size).

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There is no consensus on the mechanism by which pulsed dye lasers achieve these additional clinical effects on hypertrophic and keloid scars. Plausible explanations include laser-induced tissue hypoxia (leading to collagenesis from decreased microvascular perfusion), collagen fiber heating with dissociation of disulfide bonds and subsequent collagen realignment, selective photothermolysis of vasculature (30), and mast cell factors (including histamine, interleukins, various immunofactors) that could affect collagen metabolism (28).

Pulsed dye laser technique 

Pulsed dye laser therapy is usually performed on an outpatient basis. There is no need for general or intravenous anesthesia because the snapping sensation caused by the pulsed dye laser produces only minimal discomfort. If anesthesia is desired, topical lidocaine cream (e.g., EMLA, Elamax) with or without occlusion for 30 minutes will provide sufficient anesthesia. Any cream or makeup should be completely removed with wet gauze immediately prior to laser irradiation. Patients with scars in sensitive body locations (e.g., lips, breast, perineum, and fingers) may benefit from the use of intralesional injections or nerve blocks. Hair-bearing areas within the treatment site should be moistened with water or saline in order to reduce thermal conduction through singed surface hairs. The use of flammable substances such as alcohol or acetone should always be avoided. The patient and all operating room personnel must wear protective eyewear.

The surgical technique required for use of the pulsed dye laser calls for a series of adjacent, nonoverlapping laser pulses delivered across the entire breadth of the scar. The entire scar is treated at each session. The size, thickness, location, and color of the scar, as well as the patient's skin type, determine the energy density selected. Less fibrotic scars in sensitive skin areas (e.g., anterior chest and breast) require the use of lower energy densities, whereas thicker or darker scars can be treated with slightly higher fluences (see Table 2). In general, treatments should begin at lower fluences, allowing for the flexibility of up-ward adjustment depending on scar response. If the initial treatment session produces good results, the energy density should remain the same on subsequent treatments. If only minimal results are achieved, the treatment fluences should be increased by 10%. If the patient reports postoperative vesiculation or crusting, a lower fluence should be used with special attention placed on operative technique (avoidance of overlapping pulses).

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Table 2. Pulsed dye laser treatment considerations and protocol

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Postoperative purpura seen with the use of the vascular-specific pulsed dye laser usually resolves within 7-10 days. During the healing process the patient should be instructed to avoid extraneous manipulation of the treatment area. Showers are permitted, but care should be taken to pat lased areas dry. Gentle cleansing of the treatment area with water and a mild fragrance-free soap followed by the application of a topical antibiotic ointment is the daily postoperative care protocol. A nonstick bandage should cover the treatment area. The

lased area should be evaluated in approximately 6-8 weeks, at which time another laser treatment can be delivered, if necessary.

The most common side effect observed following the use of the pulsed dye laser is hyperpigmentation of the irradiated skin. This pigmentation will fade spontaneously with avoidance or protection from sun exposure. If hyperpigmentation is present, subsequent laser treatments should be postponed to avoid interference from a competing chromophore (or target), such as melanin. A hydroquinone-containing cream applied once or twice a day can be prescribed to speed up the fading process.

Occasionally patients may develop an allergic contact dermatitis secondary to the use of a topical antibiotic or an irritant dermatitis from an adhesive bandage. If vesiculation is present, it is imperative to determine whether it is merely the normal purpuric response or nonpurpuric and unrelated to laser irradiation. If concurrent pruritus is reported, contact dermatitis should be suspected. A mild topical corticosteroid cream should be applied until the dermatitis resolves. The offending agent, ointment, or bandage should be discontinued immediately.

Most hypertrophic scars average at least a 50-80% improvement after two laser treatments. Keloid scars or more fibrotic hypertrophic scars usually require additional laser treatments to achieve desired results.

Treatment of striae 

The 585 nm flashlamp-pumped pulsed dye laser is also used in the treatment of striae (5,31,32). Striae have been shown to respond best to lower energy densities ( $3.0 \text{ J/cm}^2$ ) (Fig. 3). Adjacent, non-overlapping laser pulses are delivered such that each individual stria is covered. Irradiated striae do not typically exhibit the characteristic purpura seen with the treatment of hypertrophic scars and keloids. Due to the lower fluences used, striae usually only appear mildly pink, representing mild postoperative tissue hyperemia and edema. Vesiculation and crusting should not be encountered when proper fluences and operative technique are used. Typically only one or two treatment sessions are necessary in order to obtain the desired results.

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Fig. 3. Striae distensae on the abdomen (A) before and (B) 6 weeks after a single 585 nm pulsed dye laser test treatment (right half of photo) at  $3.0 \text{ J/cm}^2$ , 7 mm spot

size.

The postoperative management of striae treated with the pulsed dye laser is similar to the protocol followed by patients treated for hypertrophic and keloid scars. Patients are instructed to gently cleanse the treatment areas with water and a mild fragrance-free soap. A topical antibiotic should be applied daily and the treatment area covered with a nonstick bandage. Patients should also be advised to avoid sun exposure to the treatment area during the course of treatment.

Treatment of atrophic scars 

Recontouring of atrophic facial scars with  $\text{CO}_2$  and Er:YAG laser vaporization has become popular in recent years (33-38) (see Table 3). Through their selective ablation of water-containing tissue, both laser systems offer the advantage of predictable, reproducible vaporization of tissue, yielding better control than dermabrasion (39-46). In a study comparing the histologic depths of ablation after laser resurfacing, dermabrasion, and chemical peels, Fitzpatrick et al. (47) demonstrated that skin vaporization and residual necrosis depths secondary to  $\text{CO}_2$  laser resurfacing was directly proportional to the pulse energy as well as the number of laser passes delivered. During laser resurfacing the epidermis and a variable portion of the

dermis are destroyed, with reepithelialization occurring from adjacent pilosebaceous glands. With the use of the CO<sub>2</sub> laser, in particular, the production of increased numbers of myofibroblast and matrix proteins is enhanced as a result of controlled collagen denaturation (heating) (48).

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Table 3. CO<sub>2</sub> or Erbium:YAG laser resurfacing of atrophic scars

Pulsed Er:YAG lasers are 10 times more selective for water than their CO<sub>2</sub> counterparts and thus result in enhanced tissue vaporization and reduced residual thermal damage (38). Decreased postoperative erythema is effected; however, the limited photothermal effect on tissue is countered by an overall decrease in clinical improvement. Thus short-pulsed Er:YAG laser resurfacing falls short of delivering the collagen shrinkage or "tightening effect" seen with CO<sub>2</sub> laser treatment. Overall the Er:YAG laser is effective in resurfacing skin with mild atrophic scars, yielding similar results to those of the CO<sub>2</sub> laser. In this particular setting, the Er:YAG laser may be the preferred method of treatment, offering comparable clinical effects with shorter postoperative recovery times.

Regardless of the system chosen, the goals of laser scar resurfacing are twofold: to soften the transition between the atrophic indentation and the intact (normal) skin surrounding it, and to stimulate collagen production within the atrophied area. The entire cosmetic unit must be treated in order to minimize textural or color mismatch. If treating an isolated scar, spot resurfacing may be considered. In an effort to decrease treatment time when lasing large cutaneous areas, a scanning handpiece should be used. Once de-epithelialization has been achieved (typically requiring one pass with the CO<sub>2</sub> laser at 300 mJ and two to three passes with the Er:YAG laser at 5 J/cm<sup>2</sup>), the scar edges or "shoulders" can be further sculpted with additional vaporizing laser passes. Partially desiccated tissue should be completely removed with saline- or water-soaked gauze after each laser pass in an effort to prevent charring.

Typically 300 mJ energy and 60 W power with variable-sized and shaped patterns are the laser treatment parameters used with the computer pattern generator (CPG) scanning device (Coherent Ultrapulse). Scanning devices attached to other CO<sub>2</sub> laser systems (Sharplan FeatherTouch or Luxar NovaPulse) can be used at 5-20 W/scan, depending on the system and severity of scarring. Scan sizes ranging from 4 to 10 mm in diameter are delivered to the treatment area. Treatment usually requires two to three laser passes, with care taken to remove all partially desiccated tissue between passes. Individual scar edges can be further sculpted using smaller diameter spots or scans following treatment of the entire cosmetic unit.

The Er:YAG laser is used with a 5 mm spot size at 1.0-3.0 J (5-15 J/cm<sup>2</sup>) to de-epithelialize and sculpt individual scars. A laser technique similar to that described with the CO<sub>2</sub> system is used with the Er:YAG laser; however, because Er:YAG vaporization does not typically produce a significant quantity of partially desiccated tissue, wiping of the skin between laser passes is not necessary except in hair-bearing areas (in order to reduce thermal conduction to skin through singed surface hairs). Bleeding is typically seen by the third laser pass as the result of dermal penetration and the inability of the Er:YAG laser to photocoagulate blood vessels.

Regardless of whether CO<sub>2</sub> or Er:YAG laser resurfacing is performed, treated skin will appear erythematous and edematous immediately following the procedure, with further worsening over the next 48 hours. Symptomatic palliation may be achieved with the application of topical ointments, semi-occlusive dressings, or cooling masks. The first postoperative week is a critical time period. Patients should be monitored closely for appropriate healing responses and evaluated for complications (e.g., dermatitis, infection). In the case of Er:YAG laser resurfacing, reepithelialization typically takes 4-7 days, whereas CO<sub>2</sub> laser resurfacing requires 7-10 days. Patients who have undergone full-face procedures or with large treatment areas should receive

appropriate prophylactic antimicrobials (e.g., oral antibacterial and/or antiviral medications) during the re-epithelialization process. The use of topical antibiotics on acutely irradiated skin is avoided due to the high rate of allergic or irritant contact dermatitis. Erythema is most intense and prolonged after CO<sub>2</sub> laser resurfacing (average 3-4 months). Patients on whom Er:YAG laser treatment has been performed will have minimal erythema within 1-2 weeks postoperatively.

Laser irradiation of tissue with CO<sub>2</sub> or Er:YAG laser systems may provoke a number of immediate and long-term side effects, especially when proper protocols have not been followed (49,50). A significant side effect of treatment is transient hyperpigmentation. Although observed more frequently in patients with darker skin tones, hyperpigmentation may occur in any skin type. Transient hyperpigmentation is seen early in the postoperative course, occurring approximately 1-2 months after treatment. The process is self-limiting, but resolution may be hastened with the use of bleaching creams (hydroquinone) or acid preparations (glycolic, retinoic, azelaic, and ascorbic). Hypopigmentation is a relatively late sequela of treatment-typically seen 6 months or more postoperatively-and appears to be permanent. Fortunately, true hypopigmentation (with total loss of pigment) is rare. Rather, relative hypopigmentation is most frequently observed due to the obvious color difference seen when compared to adjacent nontreated (actinically bronzed) skin. Infection is another concern postoperatively as reepithelializing skin is vulnerable to bacterial (pseudomonas, staphylococcus), viral (herpes simplex), and fungal (candida) infections. The incidence of infection is lowered with the appropriate use of prophylactic antibiotics, and more importantly, aggressive postoperative wound care (51,52). If infection is suspected, it must be diagnosed and treated early. The most severe complications of laser resurfacing include hypertrophic scarring and ectropion formation which are both due, in large part, to aggressive intraoperative laser technique. Hypertrophic burn scars can be treated effectively with 585 nm pulsed dye laser irradiation as described earlier (29), whereas ectropion typically requires surgical reconstruction.

Cutaneous laser resurfacing of moderate atrophic scars with the CO<sub>2</sub> laser yields a mean improvement of 50-80% (34-36). Collagen remodeling with further scar improvement has been reported to occur for up to 12-18 months postoperatively (37), so re-treatment of residual scars should be postponed for at least 1 year in order to accurately gauge clinical improvement (Fig. 4). The Er:YAG laser system, although effective in the treatment of atrophic scars, does not offer the same amount of collagen remodeling as does the CO<sub>2</sub> laser system and should thus be reserved for sculpting of individual scar edges and in the treatment of mild acne scarring (46).

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Fig. 4. Atrophic acne scars (A) before and (B) 6 months after full-face CO<sub>2</sub> laser resurfacing. (C) One year later, further clinical improvement is seen as a consequence of continued collagen remodeling.

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#### Summary



Current laser technology permits successful treatment of various types of scars and striae. It is not only imperative to properly classify the type of scars and striae present, but to determine which laser system will provide optimum results. The 585 nm pulsed dye laser is best used to treat hypertrophic scars, keloids, and striae. The pulsed CO<sub>2</sub> and Er:YAG laser systems effectively resurface atrophic scars. Future laser technologic advances as well as the addition of concomitant lasers and other treatments may serve to further enhance clinical results.

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