Superficial skin resurfacing

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Anatomy of the skin

The approach to superficial resurfacing of the skin necessitates a thorough understanding of the skin’s distinct anatomy and histology. The epidermis is the most superficial layer of the skin and provides a critical barrier of protection. The principal building block of the epidermis is a keratinocyte. The epidermis is composed of four distinct layers: the cornified, granular, spinous, and basal layers. The cornified layer is the most superficial and provides significant protection to the skin. It is formed from flattened, enucleate keratinocytes and compacted keratin granules. It varies in thickness, with the eyelid being the thinnest and the palms and soles the thickest. The granular cell layer contains keratinocytes and lamellar bodies, which contribute to the cornification of the skin. The spinous layer contains keratinocytes as well as a variety of immunologic cells. The basal layer, the deepest layer of the epidermis, contains basal cells whose duplicative effort replaces the cells of the superficial layers every 2 weeks. The basal layer also contains melanin producing melanocytes, which provide pigmentation in the skin.

The dermis has the important function of thermoregulation and supports the vascular network to supply the avascular epidermis with nutrients. The dermis is subdivided into two zones, a papillary dermis and a reticular layer. The dermis contains mostly fibroblasts, which are responsible for secreting collagen, elastin, and the viscous ground substance that gives support and elasticity of the skin. Also present are immune cells that are involved in defense against foreign invaders passing through the epidermis.

There are two main types of collagen in the dermis. Type I collagen, constituting 80% of dermal collagen, imparts tensile strength to the dermis. Type III collagen, constituting 15% of dermal collagen, anchors the epidermis to the dermis. Papillary dermis is made primarily of Type III collagen with a small amount of Type I collagen and fibronectin. Reticular dermis is primarily made of Type I collagen with Type III collagen and fibronectin serving adjunct roles [1]. Elastic fibers constitute approximately 3% of the dermis and provide the skin elasticity and resilience.

Histopathology

After the age of 28, the first signs of skin aging begin to appear [2]. Intrinsically aged skin results in atrophy of skin components. Although aged skin is thin with reduced elastic capacity, if there has been minimal sun exposure clinically, aged skin is smooth and unblemished. Collagen production and cross-linking of the distinct collagen bundles is decreased. Dermal elastic fibers are fewer, thicker, and less functional. Decreasing production of sebum that accompanies maturation of the skin explains the frequent finding of xerosis in mature patients.

Environmental factors such as sun exposure and smoking greatly accelerate these changes [3]. Dermatoeheliosis, also known as actinically damaged or photoaged skin, is morphologically and histologically

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distinct from intrinsically, nonsolar exposed aged skin. Photoaged skin clinically presents as deep rhytides, uneven pigmentation and mottling, and in its most severe form, a thickened texture reminiscent of leather. Most cutaneous damage secondary to that caused by ultraviolet radiation is to the dermal connective tissues, which is largely caused by increasing important matrix metalloproteinases responsible for the orderly destruction of collagen fibers, which in turn keeps in check total production [4]. Characteristic dermal findings of actinically damaged skin include haphazard arrangement of collagen and elastin fibers, degradation of collagen, breakdown in elastic fibers known as elastosis, and excess dermal melanosomes, telangiectasias, and precancerous lesions known as actinic keratoses [5].

**Cutaneous resurfacing**

Skin resurfacing by the cosmetic surgeon is a process that causes a controlled injury to skin and then stimulates a wound healing response. In response to injury, fibroblasts in the papillary dermis increase production of type I and type III procollagen in addition to transforming growth factor beta-1. The collagen increase in turns thickens the dermis, which enhances the tensile strength of the skin and yields the clinical appearance of rejuvenation.

Skin resurfacing is most commonly classified on the basis of injury depth. Superficial wounding extends to the stratum granulosum or papillary dermis. Medium-depth wounding results from extension into the upper reticular dermis. Deep wounding extends into the midreticular dermis. If the resurfacing technique is limited to superficial injury to the epidermis and superficial papillary dermis, healing generally will occur without scarring. If the injury extends deep into reticular dermis beyond adnexal structures, scarring is the likely result [6]. Invasive treatments for the treatment of solar damaged skin and facial rhytids include ablative laser resurfacing with carbon dioxide and erbium laser wavelengths, dermabrasion, and deep chemical peeling agents. Ablative resurfacing achieves the outcome of rejuvenation by the destruction of the outermost and thus most photodamaged layers of the skin. The subsequent laying down of newly formed collagen and a tightened skin appearance follows this removal [7]. Recently, multiple studies have been reported the use of nonablative techniques for skin rejuvenation. In this article, we review options we can offer patients for skin rejuvenation.

The term *resurfacing* encompasses many arenas. This spectrum includes retinoids and other topical preparations, varied depths of chemical peeling, dermabrasion and microdermabrasion, ablative and nonablative resurfacing, and one of the newest technologies, radiofrequency coablation. Retinoids are known to reverse photodamage and increase epidermal turnover and the production of dermal collagen and elastin. Importantly, they have been shown to stimulate neoangiogenesis, an effect that often can be seen in the early period of titration. For those patients who cannot tolerate retinoids, retinols are an option. Retinols are vitamin A aldehydes, and they penetrate the skin well. They are converted into small amounts of retinoic acid but are tolerated with significantly less irritation.

**Chemical peeling**

Chemical peels are the application of chemical agents that damage skin in a controlled manner. These agents can be subdivided into superficial peels, which remove the stratum corneum, and superficial peels, which remove the entire epidermis. Superficial peels require repetitive peeling sessions to obtain optimal results. The limiting therapeutic factor of superficial peels is its depth and dermal pigmentation, particularly evident in cases of severe dermatoheliosis and melasma, which will not be improved despite repeated application.

Since the days of ancient Egypt when it was rumored that Cleopatra used the debris of the bottom of wine barrels for facial rejuvenation, people have been using chemoexfoliation methods to rejuvenate skin. The original chemoexfoliant was lactic acid, an active ingredient of sour milk that was used topically by the nobles as part of an ancient skin rejuvenation regimen. In the Middle Ages, old wine with tartaric acid as its active ingredient was used for the same purpose. Today, these historical chemoexfoliants are known to contain alpha hydroxy acids, which are the active ingredients responsible for the skin exfoliation.

Modern day chemical peeling originally was promoted by dermatologists, such as P.G. Unna, who first described the properties of salicylic acid, resorcinol, phenol, and trichloroacetic acid (TCA). Slowly, the early practitioners of chemical peels began to develop other peeling agents for varying depths of penetration. In the 1960s, Baker and Gordon developed a deep peeling agent, which was able to smooth deeper furrows, especially around the mouth. From the 1980s to the present, an explosion has occurred in the mass of research on this subject, with the eluci-
mentation of many different types of peels, each for a specific range of problems [8].

The chemical peel produces a controlled partial thickness injury to the skin. After the insult to the skin, a wound healing process ensues that can regenerate epidermis from surrounding epithelium and adnexal structures, which leads to the development of new dermal connective tissue. The result is an improved clinical appearance of the skin, with fewer rhytids and decreased pigmentary dyschromia.

To understand the peeling process, there are a few key concepts. The concentration and pH of a peeling agent determine its effectiveness. Generally the greater the concentration, the more potent the chemical agent; however, concentration may vary based on the method of dilution. The strongest method is a dilution of a saturated solution. The weakest method is grams of acid crystal mixed to 100 mL of water. (The weight to weight method and the weight to volume method are intermediate in determining the concentration of the peeling agent.) The pH is also important in determining the effectiveness of the peeling agent. This occurs when the free acid component is biologically active, that is, when the pH is close to the pKA.

Chemical peeling is classified by the depth of penetration (Table 1) [9]. The process of healing involves coagulation and inflammation and is followed by reepithelialization, granulation tissue formation, angiogenesis, and a prolonged period of collagen remodeling. It is this remodeling that accounts for the continuing appearance of clinical improvement in the months following the peel procedure. A useful grading system for pretreatment classification is the Glogau system (Table 2) [10]. Peel depth can be modulated in response to the individual patient’s classification.

Application of most peeling agents is similar. Before application, the skin to be treated is first defatted with acetone or rubbing alcohol. The clinical endpoint is a brisk erythema. Because most chemical peels are lipophobic, this facilitates greater depth and promotes even distribution of the peel. The peel is applied to subunits of the face with gauze or a cotton-tipped applicator. To provide a consistent effect it is important to apply the chemical peel evenly and prevent pooling of the agent on the face. The peel is blended with untreated skin by feathering it at the edges.

Alpha hydroxy acids

Alpha hydroxy acids (AHAs) have been used for thousands of years to improve the appearance of the face. Chemically, AHAs are organic carboxylic acids with a hydroxyl group at the alpha position. AHAs are known to normalize keratinization by diminishing corneocyte adhesion in the granular cell layer; this in turn promotes improved cell turnover. Commonly used AHAs derive from fruit and dairy products, such as glycolic acid from sugar cane, lactic acid from fermented milk, citric acid from fruits, tartaric acid from grapes, and malic acid from apples. With its particular affinity for the skin, glycolic acid is the most commonly used.

The efficacy and penetrating depths of assorted AHA preparations are greatly dependant on their concentration, the vehicle, and the pH. Brands available for over the counter use are set at 3% to 10% concentrations and induce a slow exfoliation over several weeks. This strength can also be used as a

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**Table 1**

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<tr>
<th>Peel depth</th>
<th>Depth of penetration</th>
<th>Clinical depth</th>
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<tbody>
<tr>
<td>Superficial</td>
<td>0.06 mm</td>
<td>Granular layer</td>
</tr>
<tr>
<td>Medium</td>
<td>0.45 mm</td>
<td>Papillary to upper reticular dermis</td>
</tr>
<tr>
<td>Deep</td>
<td>0.6 mm</td>
<td>Midreticular dermis</td>
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**Table 2**

<table>
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<tr>
<th>Glogau classification of photoaging</th>
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<tr>
<td>Group I—Mild</td>
</tr>
<tr>
<td>Age 28–35</td>
</tr>
<tr>
<td>No keratoses</td>
</tr>
<tr>
<td>Little wrinkling</td>
</tr>
<tr>
<td>No scarring</td>
</tr>
<tr>
<td>Little makeup</td>
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pretreatment for a higher concentration peel or another resurfacing modality. Higher strengths may be used by supervised estheticians (8%–30%) for light peels. The highest strengths (40%–70%) are available for dermatologists and cosmetic surgeons for in-office peels.

The cautions are intuitive. The surgeon must take care to note the expiration date as the peel will lose effective potency with time. Solutions with a pH below 2 that contain only free glycolic acid have the potential to induce crusting and necrosis. These products can be buffered with an addition of sodium bicarbonate or sodium hydroxide, resulting in a higher pH and a weaker acid. This is independent of the concentration used. The desquamative and proliferation-stimulating effects of lactic acid are pH and concentration dependent, suggesting the “free acid” concentration is the active moiety [11].

Standard strength of professional grade AHA peels is 50% or higher. Alpha hydroxy peels differ from other peels in that they are time dependent. The time to peel is dependent on both the concentration and the pH of the peel. Higher concentrations and lower pH require shorter peeling periods. After placement of an AHA preparation, the skin becomes erythematous. A frost is not desirable in an alpha hydroxy peel because it denotes penetration depth into the dermis [12].] The AHA peel requires neutralization with cold water or a basic solution. Mild stinging and erythema typically disappears after 1 hour. The subsequent exfoliation takes place over a few days with reepithelialization in about 1 week. Multiple treatments may be required for the desired result and should be spaced at least 3 weeks to allow the epidermis to recover [13].

Complications of AHA peels are mild and temporary when the peel is performed by trained professionals using standard techniques. Care is taken to be certain the eyes are protected and that the patient has no known allergies to any of the peel’s ingredients. Standard of care is for prescription retinoid users to cease use of the agent for 2 to 3 days before a superficial peel to avoid excess irritation. Patients are susceptible to postinflammatory skin pigmentation and require UV-A and UV-B protection both before and after the peel [14].

Deeper than intended peeling may occur if neutralization is not performed within the correct window. In our experience, we also have seen increased erythema and inflammation surrounding peeled areas over sites recently augmented with bovine collagen implants. We either avoid these areas with peeling or defer placement of filler agents for a 2-week period before peeling.

**Jessner’s peel**

A Jessner’s peel is a combination of salicylic acid, 14%, lactic acid, 14%, and resorcinol, 14%, in alcohol. It is considered a mild peeling agent. Its ability to disrupt the barrier function of the epidermis is used as an ideal primer for trichloracetic acid (TCA) peels. This allows TCA peels to penetrate safely and evenly. Jessner’s solution peeling action is through intense keratolysis. Alone, Jessner’s is an easy to use peeling agent without timing restriction. Skin sloughing occurs within 2 to 4 days with subsequent epidermal regrowth [15].

**Tricholoracetic acid**

TCA typically is used as a superficial-intermediate-to-deep peeling agent in concentrations ranging from 20% to 50%. TCA (10%–35%) has been used for many years and is safe to use at lower concentrations. At higher concentrations, such as 50% and above, TCA has a tendency to cause scarring and is less manageable than other agents used for superficial peels [16].

TCA is a keratocoagulant that produces a frost or whitening of the skin, which is dependent on the concentration used. Level I frosting, defined as erythema with streaky whitening of the face, is the endpoint for superficial resurfacing. Level II frosting is defined as white coated frosting with patches of erythema showing through. Level III frosting, which is associated with penetration through the papillary dermis, is a solid white enamel frost with minimal visible erythema. Level III frosting must be reserved for areas of severe actinic damage [17].

TCA’s peeling mechanism of action at lower concentrations is through protein precipitation. Vigorous rubbing of the agent, as compared with blotting, yields a deeper penetration. This technique is not time dependent, and the agent does not require neutralization. During the procedure, if frosting is not uniform, reapplication may be performed until frosting of a desired plateau is reached. Once completed, skin sloughing proceeds for several days, and reepithelialization is complete within 10 to 14 days. Patient discomfort is controlled with oral pain medications.

The results of TCA peels of superficial depths are mild reversal of some fine wrinkles and improvement in dyspigmentation. The results may not be that which is achieved by TCA 35% Jessner’s combination but the recovery period and risk are also less [18].
Salicylic acid

Beta hydroxy acids (BHA), also known as salicylic acids, are not AHAs but are chemically defined as having an organic carboxylic acid with a hydroxyl group at the beta position. BHAs have more of a predilection for sebum-containing cells and are lipid soluble; therefore, they are an excellent peeling agent for comedonal acne. Another benefit of salicylic acid is that it does not need to be neutralized. After applying BHA to the skin, salt formation on the skin is seen [19].

Salicylic acid appears to provide a safe, mild rejuvenation to skin. A study by Grimes et al [20] looked at concentrations of salicylic acid between 20% and 30% in Fitzpatrick skin types V and VI. Eighty-eight percent of patients described a moderate to significant improvement in acne, oily skin, textural changes, melasma, and postinflammatory pigmentation. Minimal side effects occurred in 16% of patients. Klingman et al [21] also demonstrated that BHAs at 30% concentration were efficacious in photoaged skin. They found reduction in fine lines, surface roughness, and pigment spots.

Adverse effects, usually only found with high-dose oral ingestion, include headache, nausea, and ringing of the ears, each of which may be resolved with a few glasses of water and rest. These have never been reported with a peel procedure. Cutaneous side effects are minimal and can include erythema and mild irritation to the salt formation on the skin [22].

Dermabrasion

Dermabrasion is an older technique that removes the epidermis and upper dermis by use of an abrasive wheel driven by a high-speed engine. This mechanical removal yields a middermal wound. Healing is achieved via reepithelialization and repigmentation from the residual adnexae. Areas with diminished numbers of adnexal structures, such as keloids and hypertrophic scars, respond poorly to mechanical dermabrasion, as these adnexae are not present [23]. Dermabrasion has been used less frequently since the advent of laser resurfacing but still offers an excellent option in the treatment of deep acne scars.

Microdermabrasion

Microdermabrasion originated in Italy in 1985 by Marini and Lo Brutto [24], who reported both gross and histologic improvement in treated skin. Its ease of performance and lack of downtime has made it a patient favorite in the United States. Clinically, it is mechanical debridement of the most superficial layers of epidermis. It commonly involves the use of a closed-loop, negative pressure system with debrouding aluminum oxide crystals to ablate the superficial layers of the epidermis. Some systems use sodium chloride, which is a positive pressure system [25].

Quality and purity can vary substantially among the crystals. The substance most often used is aluminum oxide. It is usually preferred because of its hardness, inertness, and superior abrasion qualities. Alternative crystal mediums also are available and range from bicarbonate sodium to salt. Advantages of bicarbonate sodium is its purity and its ability to dissolve in water during clean up, leaving no gritty residue. A disadvantage is that it is softer than aluminum oxide and requires more passes.

Performance of a microdermabrasion treatment is technically simple. No preoperative anesthesia or antibiotics are necessary. The handpiece has a vacuum that draws in the skin as the handpiece is passed over the area to be resurfaced. Depth of abrasion is controlled by pressure of the crystals being propelled, pressure of the handpiece, and speed of the pass. The face typically requires two passes, with the second pass perpendicular to the first one. Vertical passes only are advisable for the neck area.

Historically, with microdermabrasion the assumption was that repetitive intraepidermal injury allowed for gradual improvement of photodamaged skin by stimulating fibroblast activity and new collagen deposition in the dermis [26]. A recent study from Freedman et al [27] suggests that microdermabrasion may indeed yield greater histologic improvement than previously realized, with treated patients showing histologic evidence of thickening of the epidermis and dermis, flattening of the rete pegs, vascular ectasia, and perivascular inflammation, and newly deposited collagen and elastic fibers as compared with control subjects. A study by Shim et al [28] demonstrated a statistically significant improvement in roughness, mottled pigmentation, and overall improvement in skin appearance in a small number of patients.

Laser resurfacing

Ablative

When performed by an experienced practitioner, ablative laser skin resurfacing yields often dramatic and reproducible improvement in the appearance of
photoaged skin. It does so by reducing solar-induced dyspigmentation and rhytids and improving allover skin tone [29]. Histologically these changes correlate with architectural normalization of the epidermis and the formation of a dermal repair zone comprised of parallel collagen arrays [30]. The two principal lasers used are the carbon dioxide (CO2) and erbium yttrium-aluminum-garnet (Er:YAG) systems with similar mechanisms: ablation of photodamaged skin, thermal collagen contraction, and stimulation of immediate and delayed collagen remodeling.

Resurfacing requires an understanding of laser biophysics and its interaction with skin. Laser is an acronym for light amplification by stimulated emission of radiation. The therapeutic action of light energy is the product of characteristics distinct to laser light and resultant laser-tissue interactions. Laser light is monochromatic, that is, the emitted light is of a single wavelength. At specific wavelengths, specific absorption of light energy by a pigmented target can occur. Another property is coherence, both in time and space, analogous to a marching band in step. A third property is collimation, which is the emission of a powerful beam of light in a parallel manner, which permits its focus into very small spot sizes, which permits precise tissue destruction [31].

Lasers have several vital characteristics that influence their ultimate effects in tissue. The fluency, power density, and frequency each function to adapt the effect of laser light on tissue. Tissue ablation occurs when the tissue is heated to its boiling point. Depending on the absorbance of the host target tissue, the laser may cause unintended damage to the surrounding tissue. This can be measured with the tissue relaxation time. When the time of laser delivery is greater than the tissue relaxation time, unintended damage to the surrounding tissues results. When the energy level is greater than the critical value for the particular tissue and delivered in less than the thermal relaxation time for that particular tissue, tissue is ablated with minimal heat conduction to the surrounding tissues [32]. The understanding of this principle of selective photothermolysis changed the face of laser resurfacing and issued in a new era in the treatment of photoaging.

The CO2 laser emits infrared light with a 10,600-nm wavelength absorbed by intracellular and extracellular water. After this energy is absorbed, water is immediately converted into steam with charring of tissue and minimization of collateral tissue damage [33]. Pulsed systems provide the further advantage of minimizing damage to adjacent tissue [34]. In trained hands, impressive results are achieved safely in the treatment of severe photoaging. The major limiting factor for this procedure is the extended duration of recovery, with pronounced erythema for 3 to 6 months. Additionally, significant postoperative complications such as oozing, bleeding, and infection can occur [35]. Also of great concern is the risk of delayed permanent hypopigmentation seen in up to 20% of patients when multiple-pass CO2 resurfacing is performed [36,37].

The demand for less aggressive resurfacing modalities led to the development of the short-pulsed Er:YAG laser. The Er:YAG laser, with a wavelength of 2940 nm, produces laser irradiation in the near infrared portion of the electromagnetic spectrum. This wavelength corresponds to a main peak of water absorption, one that is much more (10–15 times) efficiently absorbed by superficial (densely water containing) tissues. The Er:YAG arrived on the scene to great enthusiasm as practitioners hoped for a comparable clinical result to CO2 with more rapid wound healing; however, Er:YAG resurfacing has since been shown to yield less apparent clinical improvement for rhytids than CO2 at equivalent depths of treatment [38]. This is because the Er:YAG system produces only about 5 to 20 µm of thermal damage per pass, as opposed to the 50 to 125 µm of thermal damage seen with each CO2 pass. Short pulsed erbium treatment can be adjusted to go superficially, mimicking the benefit of a microdermabrasion, or it can go deeper with multiple stacked passes, mimicking the benefits of CO2. Short pulsed erbium is best used as a superficial treatment at the limits of the dermis.

CO2 systems produce a significant amount of thermal effect, which acts as a “heat sink” for the next laser pass, thus yielding increased damaged collagen and leading to an increase in new collagen production [39]. Additionally, the Er:YAG system offers the laser surgeon poor intraoperative hemostasis, unlike the excellent hemostasis provided by CO2. Recently, modulated (short and long pulsed) Er:YAG systems have been introduced to facilitate deeper ablation of tissue and improve intraoperative hemostasis [40,41]. Indications favoring use of a short pulsed Er:YAG system are mild to moderate photodamage and superficial dyspigmentation as well as patients with darker skin phototypes [42]. The modulated systems offer results between that of CO2 and short-pulsed Er:YAG systems.

Er:YAG systems yield photomechanical benefits whereas the CO2 laser yields a photothermal effect. The lack of a photothermal effect for the Er:YAG laser means that heat does not dissipate deeper into the surrounding tissues. There are fewer complications of severe thermal damage with the Er:YAG laser; how-
ever, this also translates to less collagen shrinkage with the Er:YAG laser versus the CO2 laser [43]. Er:YAG resurfacing may soon reemerge as a middle ground option, providing patients with an moderate improvement in skin texture and tone beyond that achieved with microdermabrasion and superficial treatments, yet without the associated downtime of the deeper reaching modalities (Fig. 1).

Nonablative

The devices available for nonablative photorejuvenation can be split into two primary categories: visible light devices 532 nm (green) and 585 nm (yellow), light best suited to treat pigmentary and vascular lesions. These wavelengths are strongly absorbed by oxyhemoglobin and melanin in the epidermis and superficial dermis. Mid-infrared wavelength devices at 980 nm, 1320 nm, 1450 nm, and 1540 nm are coupled with cooling mechanisms that serve to protect the epidermis while simultaneously stimulating direct collagen remodeling in the dermis. These mid-infrared wavelengths are absorbed primarily in water (intracellular and extracellular) and can uniformly heat tissue independent of skin type [44].

While using a pulsed-dye laser for treatment of vascular periocular lesions, it was noted that there was a decrease in the clinical appearance of rhytids [45]. Histologic improvement of dermal collagen also was noted after treatment with the pulsed-dye laser. Using these findings, Zelickson et al [46] evaluated the use of a 585-nm pulsed-dye laser in the treatment of facial rhytids and reported improvement. The Zelickson report suggests that there also is potential efficacy with lower fluences than used in the trial and less associated purpura, the endpoint that patients find least cosmetically acceptable.

The first system specifically designed for the purpose of nonablative resurfacing was a 1320-nm neodymium:yttrium-aluminum-garnet (Nd:YAG) laser (Cool Touch, Roseville, CA). The goal of this system, similar to that of the previously described systems, is improvement of rhytids without the creation of an open wound. The 1320-nm wavelength is advantageous in its high scattering coefficient. Thus, the laser irradiation scatters throughout the treated dermis after nonspecific absorption by dermal water. Studies have reported that this system is able to produce thermal stimulation of dermal fibroblasts within the papillary and midreticular dermis while concomi-

Fig. 1. Patient with mild rejuvenation seen after use of Er:YAG laser treatment. (A) Pretreatment. (B) Post treatment.
stantly cooling the epidermis to protect it from undesired thermal injury [47].

Histologically, there is replacement of the irregular collagen bands with organized new collagen fibrils [48]. In a 1999 study, Kelly et al [49] reported the use of the 1320-nm wavelength Nd:YAG laser for the treatment of facial rhytids. They reported statistically significant findings at 12 weeks. In another study of similar technology, Goldberg et al [50] reported clinical improvement in 8 of the 10 patients in the study. The Cooltouch was modified and reintroduced as the Cooltouch II, which delivers energy densities up to 24 J/cm² with a 10-mm spot handpiece. The laser handpiece has three roles: delivery of the laser pulse, application of a thermal spray for cryogen cooling, and a sensor for the assessment of skin temperature at the surface.

Recently, Ross et al [51] described the Smoothbeam 1450-nm diode laser system that was also shown to be modestly effective for the nonablative treatment of photoaged skin. The 1450-nm wavelength is extremely well absorbed by water. Sixteen patients (14 periorbital, 2 perioral) with rhytids were treated with split-face treatments: half had four visits 3 weeks apart with the 1450-nm laser device and the contralateral side with cryogen cooling alone. The laser uses spray cryogen cooling to protect the epidermis and permit selective dermal heating. The authors reported mild to moderate improvement in 12 to 16 patients [51].

The long pulse 1064-nm laser with its low scattering coefficient and weak absorption by water and melanin also has been shown to improve the appearance of coarse wrinkles and fine lines and to reduce skin laxity. After seven treatment sessions spaced over 14 weeks, the patients’ subjectively graded improvements in their skin’s appearance mirrored masked physician observers recognized improvements in the same categories. This data reached statistical significance [52].

Intense pulsed light, a nonlaser light source, can be delivered at a variety of wavelengths (590 to 1200 nm.) Filters permit the inclusion and exclusion of given wavelengths [53]. With blockage of shorter wavelengths, deeper wavelengths can be absorbed in the dermis and yield nonablative dermal remodeling [54]. Goldberg reported on five patients who underwent four sessions of intense pulsed light source therapy and from whom pretreatment and 6-month posttreatment biopsies were obtained. Their results indicated histologic evidence of new upper papillary dermal collagen formation [55]. The intense pulsed light has particular utility for the treatment of dyschromias and mottling [56].

**Thermal resurfacing**

Thermal resurfacing, also referred to as cold ablation (coblation) or radiofrequency ablation is essentially removal of the outer layer of skin via bipolar electrical current. It is a descendent of the bipolar systems originally used in orthopedics to resurface joint cartilage. Whereas lasers rely on heat to remove tissue, coblation disrupts molecular bonds at the cellular level by the movement of ions and free electrons, which strike the bonds and disrupt them [57].

The coblation procedure begins with application of povidone iodine to the skin. A local anesthetic may then be applied. Lastly, saline gel is placed over all of the areas to be treated. This gel is essential because the coblation device energizes particles in the saline gel, which will subsequently strike tissues and disrupt tissues via their movement [58].

Coblation also can use a saline solution. Isotonic saline is then passed over the stylet while the stylet remains in constant contact with the skin. Three bipolar strips separated by 1 mm are situated at the end of a stylet. Ions within the isotonic saline are energized by the bipolar frequency and form a “plasma” of charged ions able to break down molecular bonds within the tissue and cause separation of the epidermal–dermal junction. The plasma is theorized to form a plasma shield, decreasing both the energy reaching the target tissue and reducing the collateral tissue damage [59]. Skin is aggressively precooled and cooled during electrical current production to protect the epidermis and dermis [60].

Topical and oral pain relief is given before the procedure. For deeper treatments a local anesthetic field block is performed. Antiviral and antibiotic prophylaxis are given to all patients because the layer of necrotic cobalted debris that will slough off after the treatment is a potential bacterial culture medium [61].

Coblation injury is reported to be in between that of CO₂ laser resurfacing and Er:YAG laser resurfacing. If the voltage is reduced so that the saline is not transformed into plasma, small vessels can be coagulated, creating a bloodless field; however, some authors have reported some bleeding with over-aggressive wiping of skin, especially in those patients with prominent extant telangiectasias at the time of treatment. Ablation may be effective in some scar revision. It is probably a less effective method to reduce rhytids than CO₂ or Er:YAG lasers [62]. Coblation also appears to have less erythema than laser resurfacing. Grekin [63] reported less pronounced erythema and resolution within 2 months. Coblation appears to offer potential as a superficial
resurfacing device. Because of the limited studies on thermal resurfacing, the ideal parameters of the device are not yet known.

**Contraindications to resurfacing**

Given the significant potential morbidity after cutaneous resurfacing procedures, all candidates must receive an extensive preoperative treatment evaluation. Patients must be screened for their ability to tolerate the necessary recuperation and unpleasant cosmetic period immediately following the procedure. The preoperative medical evaluation should include a complete medical history and any medication allergies. Patients with active cutaneous infections must be excluded from therapy.

Patients should be asked about any history of poor scarring or keloid development, allergic tendencies, connective tissue disorders, or a history of oral herpes simplex virus. Recent literature suggests that patients with a recent history of isotretinoin (Accutane) ingestion are at an increased risk for keloid development; consequently, these patients should not be treated for 12 months after completion of the course of therapy [64].

In expert hands, complications are minimal. The exception is poor technique [65]. Most complications occur during the postoperative reepithelialization process. There are differing opinions on the prophylaxis of herpes infections [66,67]. We routinely prescribe prophylaxis for all Er:Yag and CO₂ laser procedures as well as TCA chemical peels regardless of history. Seven percent of patients developing a herpes infection after laser resurfacing do not report any history of herpes labialis Fig. 2) By contrast, superficial chemical peels limited to the epidermis and superficial dermis that do not result in deep epitheliazation are not routinely prophylaxed [68].

A history of resurfacing or surgical procedures is another important possible contraindication. Some authors list prior blepharoplasty or face lift within the last 2 months as a relative contraindication to resurfacing because of the increased risk of scarring and temporary altered blood supply postoperatively; however, there is documented evidence of successful outcomes in patients who have had resurfacing done simultaneously with facelifts. Nonetheless, caution is warranted when resurfacing an area with vascular compromise secondary to a recent procedure. Additionally, patients who have had prior skin muscle flap lower eyelid blepharoplasty through a subciliary approach may have reduced lower lid laxity secondary to weakened orbicularis muscular support. Skin contraction and further compromise to lower lid support following medium depth resurfacing of thin lower eyelid skin can occur placing the patient at particular risk for ectropion.

Other pertinent historical facts include allergic or hypersensitivity reactions to topical anesthetics, petrolatum, or lanolin. Although rare, allergies to these agents can be problematic because they are used routinely in superficial and deeper resurfacing protocols and can lead to extension of the dermal injury. Also, patients with recurrent facial candidal or adenexal infections require precautionary measures before a superficial resurfacing procedure, and they may be relatively contraindicated for a deeper treatment.

**Summary**

Best results for all rejuvenating modalities lie in the joint hands of the cosmetic surgeon and the patient. The absolute need for pretreatment education and posttreatment care cannot be overemphasized. Additionally, other adjunctive efforts such as judicious use of botulinum toxin and the use of filler substances can optimize results. Ultimately it is a combination of the surgeon’s skill and the patient’s
compliance with instruction that determines the overall result. Setting realistic expectations for patients is an absolute imperative and will serve to maximize patient satisfaction.

References


